UNIVERSITY OF PORTSMOUTH

FACULTY OF SCIENCE AND HEALTH

DEPARTMENT OF PSYCHOLOGY

The Impact of Impulse Control on Alcohol Use in the Context of Acute, Chronic, and Cumulative Lifetime Stress

by

James Michael Clay

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The thesis is submitted in partial fulfilment of the requirements for the award of the degree of Doctor of Philosophy of the University of Portsmouth

“Three bowls do I mix for the temperate: one to health, which they empty first; the second to love and pleasure; the third to sleep. When this bowl is drunk up, wise guests go home. The fourth bowl is ours no longer, but belongs to violence; the fifth to uproar; the sixth to drunken revel; the seventh to black eyes; the eighth is the policeman's; the ninth belongs to biliousness; and the tenth to madness and the hurling of furniture".

Eubulus (c. 375 BC)

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Alcohol consumption contributes to over 200 diseases and conditions and has severe socioeconomic consequences for both individual consumers and the society in which they live. If left unchecked, alcohol misuse can develop into an addiction. Previous research has identified stress and impulsivity (i.e., the tendency to act in haste and without foresight) as key risk factors for alcohol misuse and addiction. Furthermore, prior work has shown that impulsive behaviour strengthens acute psychosocial stress–induced alcohol craving and consumption. However, both stress and impulsivity are multifaceted and relatively little is known about how different measures of impulsivity or different stressors effect the stress–impulsivity–alcohol pathway. Therefore, the overall aim of this thesis was to better understand the impact of impulsivity on alcohol use in the context of acute, chronic, and cumulative lifetime stress. Specifically, the hypothesis that the effects of stress on alcohol use behaviour would be strengthened by impulsivity was tested across several studies ranging from experimental studies to large–scale national cohort approaches. First, drinking behaviour was assessed in response to *acute* physical, psychosocial, and mixed stressors. It was found that heightened negative urgency (i.e., the tendency to act rashly under extreme negative emotions) and negative affect were associated with increased levels of alcohol craving. Second, the COVID–19 pandemic was utilised to understand the impact of *chronic stress* on alcohol use behaviour, finding that stress and impulsivity were both independently associated with increased alcohol use behaviour. However, the direction of the negative affect and personality interactions went in the opposite direction to that which was predicted in the primary hypothesis of this thesis. Finally, the main hypothesis of this thesis was tested and extended in the context of *cumulative lifetime stress*. It was hypothesised that the association between cumulative lifetime stressor exposure and lifetime alcohol use would be mediated by emotional dysregulation, and that increased impulsivity would strengthen these relationships. The data supported these predictions and negative urgency was found to be a critical moderator, strengthening these relationships. Overall, this thesis provides a nuanced overview of stress–impulsivity interactions in terms of alcohol use and highlights the importance of negative urgency and emotional regulation in these relations.

Declaration

Whilst registered as a candidate for the above degree, I have not been registered for any other research award. The results and conclusions embodied in this thesis are the work of the named candidate and have not been submitted for any other academic award.

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I doubt anyone who know me during my early life would have expected me to go on to pursue a PhD. My time studying at university has allowed me to find something that I am passionate about. I am thankful for the opportunity to do so.

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List of Abbreviations

APQ	Alcohol Problems Questionnaire
AUC	Area Under the Curve
AUD	Alcohol Use Disorder
AUDIT	Alcohol Use Disorders Identification Test
BAC	Blood Alcohol Concentration
BART	Balloon Analogue Risk Task
B–BAES	Brief Biphasic Alcohol Effects Scale
BCS70	1970 British Cohort Study
BDMA	Brain Disease Model of Addiction
BIS–11	Barratt Impulsivity Scale – 11
BMI	Body Mass Index
BPM	Beats per Minute
CLS	Centre for Longitudinal Studies
CPT	Cold Pressor Task
DALYs	Disability–Adjusted Life–Years
DAQ	Desires for Alcohol Questionnaire
DBP	Diastolic Blood Pressure
DDT	Delay Discounting Task
DERS–SF	Difficulties in Emotional Regulation Scale Short Form
DMQ–R	Revised Drinking Motives Questionnaire
DOSPERT	Domain–Specific Risk–taking Scale
DSM	Diagnostic and Statistical Manual of Mental Disorders
EFA	Exploratory Factor Analysis
ETOH	Etiologic, Theory–based, Ontogenetic Hierarchical Framework
GDP	Gross Domestic Product
GLMM	Generalised Linear Mixed Model

GRQ	General Risk Question
HED	Heavy Episodic Drinking
HIT	Hand Immersion Trial
HPA	Hypothalamic–Pituitary–Adrenal Axis
HR	Heart Rate
HRV	Heart Rate Variability
HSE–21	Health Survey for England, 2021
ICC	Intraclass Correlation Coefficient
ICD	International Classification of Diseases
LDH–q	Lifetime Drinking History Questionnaire
LMM	Linear Mixed-Effects Model
LRDG	Low–Risk Alcohol Drinking Guidelines
MA	Mathematics Assessment
MAP	Mean Arterial Pressure
MAST	Maastricht Acute Stress Test
MCS	Millennium Cohort Study
MI	Multiple Imputation
MSBS	Multidimensional State Boredom Scale
NA	Negative Affect
NCDS	National Child Development Study
NIAAA	National Institute on Alcohol Abuse and Alcoholism
NSHD	National Study of Health and Development
NS–SEC	National Statistics Socioeconomic Class
PA	Positive Affect
PANAS	Positive Negative Affect Schedule
PFI	Personal Feedback Intervention
PHQ–4	Patient Health Questionnaire for Depression and Anxiety

PSRS	Perceived Stress Reactivity Scale
RMSSD	Root Mean Square of Successive Differences Between Normal Heartbeats
RR	Response Rate
SARS-CoV-2	Severe Acute Respiratory Syndrome Coronavirus–2
SBP	Systolic Blood Pressure
SDNN	Standard Deviation of Normal–to–Normal Intervals
SES	Socioeconomic Status
SOS–S	Short Stress Overload Scale
SSRT	Stop Signal Reaction Time
SST	Stop Signal Task
STAI	State–Trait Anxiety Inventory
STRAIN	Stress and Adversity Inventory for Adults
SUD	Substance Use Disorder
SUPPS–P	Short UPPS–P Impulsivity Scale
TADD	Titrating Alternatives Delay Discounting Task
TADD	Typical Atypical Drinking Diary
TSST	Trier Social Stress Test
VAS	Visual Analogue Scale

Dissemination

Publications included in thesis

Chapter 3

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Chapter 4

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Chapter 5

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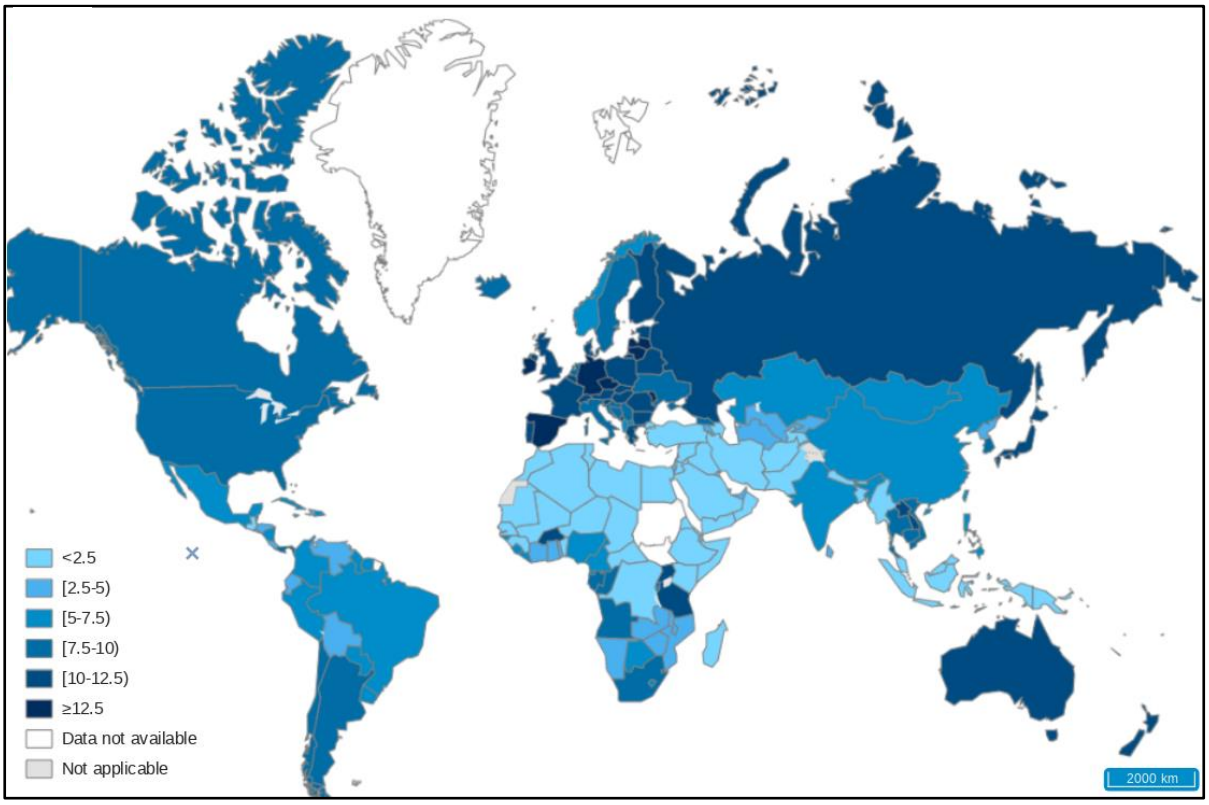
Chapter 1. Introduction

Alcohol is a psychoactive substance that can produce pleasure (i.e., hedonistic effects), intoxication (i.e., impaired physical and mental functioning), and dependence (i.e., persistent and excessive use of a substance, despite adverse consequences). Drinking alcohol is embedded in history and culture, and continues to play a key role in many social and religious events (Hanson, 2013). It is one of the most commonly used psychoactive substances in the world and current estimates suggest that approximately 43% of people (aged 15 year and over) drink alcohol globally (World Health Organization, 2018a), with the majority of this consumption occurring in the Americas, Europe and the Western Pacific (see Figure 1.1).

Figure 1.1 Estimated age 15+ alcohol consumption in litres of pure ethanol by country. Adapted from World Health Organisation (2023a) under the Creative Commons Attribution 4.0 International License.



The purchase and consumption of alcohol is *legal* for those aged 18 years and over in the UK, with children between 16 and 17 years of age permitted to consume beer, wine, or cider with a meal (*Licensing Act*, 2003). Consequently, the majority (57%) of British people consume alcohol each week (Office for National Statistics, 2018). However, despite alcohol use being legal and commonplace, it is not without risk. For instance, The World Health Organization recently stated that “no level of alcohol

consumption is safe…" (World Health Organization, 2023). This is due to the significant global public health concerns associated with alcohol–related harm (World Health Organization, 2022).

1.1. Consequences of alcohol use

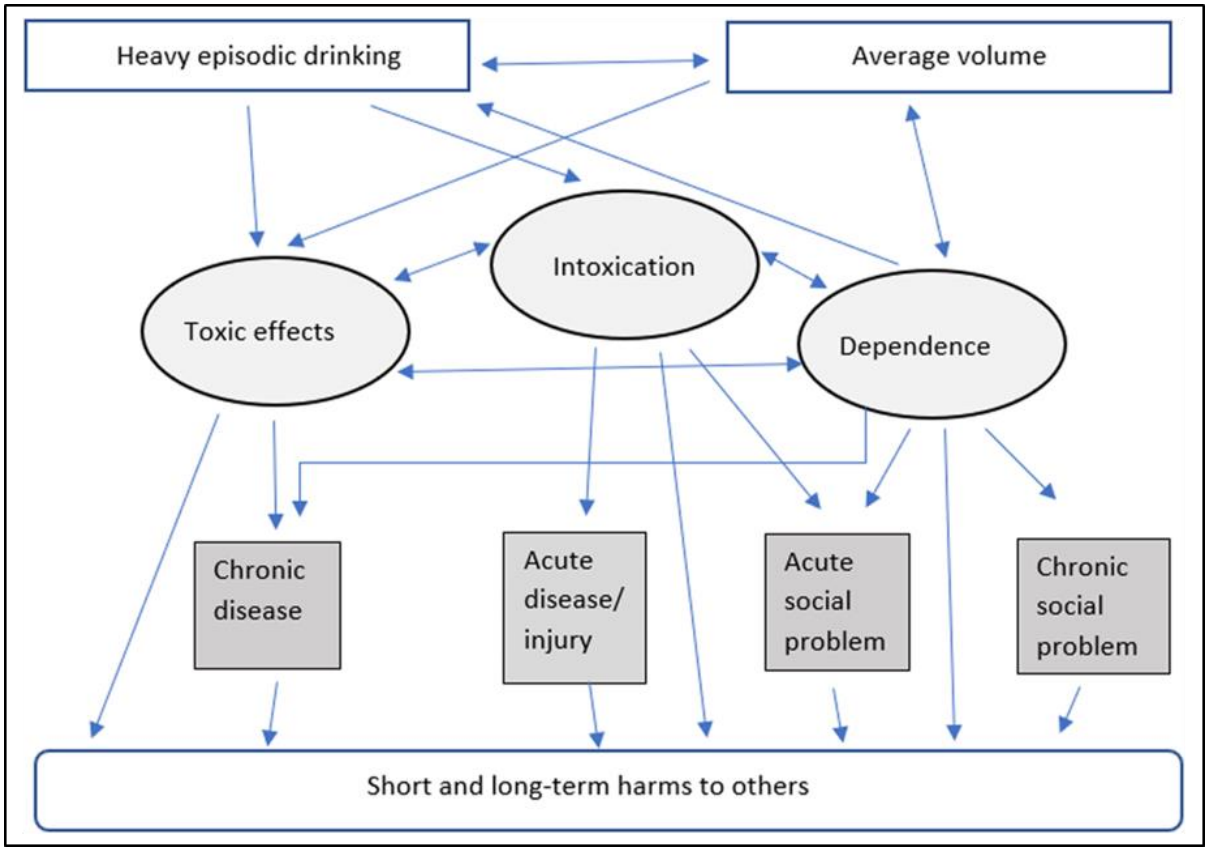
Alcohol consumption contributes to over 200 diseases and conditions and has severe socioeconomic consequences for individual users and the society in which they live (World Health Organization, 2018a). The level of harm associated with alcohol use is proportional to the volume and quality of the alcohol consumed, and the pattern of consumption (see Figure 1.2 for overview). For instance, Rehm et al. (2010) identified dose–response relationships in all alcohol–related disease categories (except depressive disorders) with risk increasing as a function of volume consumed. Moreover, managed Alcohol Programs (i.e., programs designed to manage the supply of alcohol to heavy–drinkers by providing regular measured doses) have been shown to be an effective harm reduction strategy, particularly among those who consume non–beverage alcohol (e.g., rubbing alcohol, cooking wine) (Stockwell & Pauly, 2018. Furthermore, HED is known to be more harmful that spreading the consumption of the same quantity of alcohol over several drinking sessions (Naimi et al., 2007). Thus, alcohol has been classified as "no ordinary commodity" due to its wide availability and usage, social acceptability, and harm profile (Babor et al., 2022).

1.1.1. Health consequences

Alcohol use is a leading risk factor for communicable diseases, non–communicable diseases, and injuries (Shield et al., 2020). Health consequences partially or wholly attributed to alcohol use include (for example) cancer (e.g., oral, breast, liver, intestinal), cardiovascular disease (e.g., hypertensive heart disease, ischaemic heart disease, ischemic and haemorrhagic stroke), metabolic disorders (e.g., diabetes mellitus), respiratory disease (e.g., tuberculosis, pneumonia), mental disorders (e.g., depression, anxiety), cirrhosis of the liver, foetal alcohol syndrome, road traffic accidents, and others (Brick, 2004).

Current global estimates suggest that alcohol is attributable to approximately 3 million deaths and 131 million Disability–Adjusted Life–Years (DALYs, i.e., the loss of the equivalent of one year of full health) (Shield et al., 2020). This is equivalent to around 5% of all global deaths and DALYs. This level of harm corresponds to an estimated financial cost of 2.6% of global gross domestic product (GDP)

Figure 1.2 Schematic overview representing the relationships between patterns of alcohol consumption (upper rectangles), mechanisms of action (ovals), and harm to individual drinkers and others (bottom boxes). Single–headed arrows represent direct effects and double–headed arrows represent reciprocal relationships. Reproduced from Babor et al. (2022) under the Creative Commons Attribution 4.0 International License.



with the majority of these costs being attributable to lost productivity and around one–third incurred directly (Manthey et al., 2021). More specifically, recent statistics show that 980,000 alcohol–related hospital admissions were recorded in England in 2018/19 (NHS Digital, 2022b), and that the number of alcohol–related deaths in the UK reached a record high in 2021 (9,641 deaths, 14.8 per 100,000 people) (Office for National Statistics, 2022). This corresponds to an estimated societal cost of alcohol in the UK of £54 billion[1].

Notwithstanding the clear physical harms caused by alcohol, the idea that moderate drinking is good for health is ingrained in many cultures and the belief that low–dose alcohol use can protect against mortality remains controversial among scholars (e.g., Hawkins & McCambridge, 2021). This is because

[1] This figure was calculated using the methodology found in Rehm et al. (2009) which suggests that the weighted average total cost of alcohol to society in high–income countries is equivalent to 2.5% of GDP. GDP data was sourced from the Office for National Statistics (Office for National Statistics, 2023).

several studies demonstrate J–shaped risk curves (whereby low levels of alcohol consumption seem to be associated with protection and high doses with increased risk); particularly when considering deaths caused by heart disease (Fillmore et al., 2006; Roerecke & Rehm, 2012). However, recent systematic reviews and meta–analyses have demonstrated that these effects disappear once several study–level biases are accounted for in the analysis (Zhao et al., 2017, 2023). Therefore, all levels of alcohol consumption increase the risk of harm to the user, with risk compounding at higher levels of use.

1.1.2. Socioeconomic consequences

The extent of alcohol–related harm does not stop at the individual consumer, extending into the family home and society more broadly. There are three major categories of alcohol–related harm to others (rather than the individual consumer): (1) harm to children, (2) unintentional injuries, and (3) intentional injuries / violence (Rehm et al., 2017). Regarding harm to children, alcohol can harm a child before birth, predominantly through foetal alcohol spectrum disorders and foetal alcohol syndrome which are the leading causes of preventable birth defects and mental disabilities (Rehm et al., 2017). For instance, Thanh & Jonsson (2016) estimate the life expectancy among people with foetal alcohol syndrome at just 34 years of age. Furthermore, the risk of unintentional injuries to children increases as a function of caregiver alcohol use (Damashek et al., 2009) and caregiver alcohol misuse is strongly associated with traumatic brain injury among children (Winqvist et al., 2007). In terms of unintended injury outside of the family, the impact of alcohol on road traffic accidents has been studied most (Rehm et al., 2017). For example, blood alcohol concentration (BAC) levels as low as 0.05% impairs driving ability (Martin et al., 2013) and up to 35% of all global road deaths involve alcohol (World Health Organization, 2018b). With regard to intentional injuries, the link between alcohol and violence is also well–established, though complex (Bushman & Cooper, 1990; Parrott & Eckhardt, 2018). For instance, alcohol has been shown to be related to increased levels of intimate partner violence in meta–analysis (Foran & O'Leary, 2008) and it is estimated that alcohol is involved in up to 50% of all violent crimes in England each year (Office for National Statistics, 2017). Taken together, alcohol use has both severe intended and unintended socioeconomic consequences.

1.1.3. Low–risk Drinking Guidelines (LRDGs)

Alcohol control measures can be implemented by policy makers in an attempt to reduce alcohol consumption and alcohol–related harm (Babor et al., 2022). One such measure is Low–Risk Alcohol Drinking Guidelines (LRDG), which are implemented by several governments worldwide (Kalinowski & Humphreys, 2016). LRDGs provide guidance about how people should limit their daily and weekly alcohol consumption and heavy episodic drinking (HED; i.e., consuming large amounts of alcohol in a short space of time). Therefore, LRDGs aim to empower drinkers to moderate their own consumption and to inform primary care practitioners on what constitutes high–risk drinking. For instance, the World Health Organization defines a standard drink as 10g of ethanol and recommends that neither men nor women should consume more than two standard drinks per day (World Health Organization, 2001). However, on a global scale, there is high country–to–country variability in how much ethanol represents a standard drink (8–20g) and in LRDGs, with daily limits ranging from10–42g for women and 10–56g for men, and weekly limits ranging from 98–140g for women to 150–280g for men (Kalinowski & Humphreys, 2016).

The UK Chief Medical Officer recommends that neither men nor women should drink more than 14 units[2] per week spread over three or more days (Department of Health England et al., 2016). Despite this clear public health message, data from the Health Survey for England, 2021 (HSE–21) suggests that 43% of people (age 16+) in England frequently drink more than 14 units per week and approximately 11% of individuals regularly consume alcohol on 5 days or more per week (NHS Digital, 2022a). These individuals are at an increased risk of alcohol–related harm (Department of Health England et al., 2016). Therefore, the importance of a greater understanding of what drives this level of alcohol consumption among these individuals cannot be understated.

1.1.4. Alcohol dependence and alcohol use disorder

Long-term alcohol misuse (e.g., frequently drinking > 14 units per week) can result in an alcohol–related clinical diagnosis for some individuals. The two main international systems used to provide alcohol–related clinical diagnoses are the International Classification of Diseases (ICD), published by

[2] 1 UK alcohol unit equates to 8g of ethanol.

the World Health Organization, and the Diagnostic and Statistical Manual of Mental Disorders (DSM), published by the American Psychiatric Association (Saunders et al., 2019). By design, there is significant overlap between the ICD–11 diagnosis of alcohol dependence and the DSM–5 diagnosis of alcohol use disorder (AUD) (Saunders et al., 2019). However, there are key differences in the overall classification of diagnostic entries in each system due to both the differences in the objectives of each system and the knowledge available at the time that the systems were created (see Clark et al., 2017 for review).

The diagnostic criteria for both alcohol dependence and AUD are presented in Table 1.1. In summary, both ICD–11 and DSM–5 diagnoses include: a loss of control over drinking, failure to fulfil prior commitments due to an increased priority given to alcohol, persistent social and interpersonal problems, tolerance, and withdrawal. Despite the relatively high qualitative overlap between both conditions, a key difference is that DSM–5 AUD is graded from mild (2–3 symptoms), through moderate (4–5 symptoms), to severe (6+ symptoms). Moreover, quantitative data suggests that alcohol dependence and AUD are distinct conditions. For instance, in a large representative sample, Lago et al. (2016) found low overall concordance between ICD–11 and DSM–5 ($\kappa = 0.30$). However, the concordance rates improved as a function of AUD severity (mild $\kappa = 0.06$, moderate $\kappa = 0.24$, severe $\kappa = 0.67$), suggesting that ICD–11 alcohol dependence is most similar to severe DSM–5 AUD. Finally, an AUD diagnosis can be given on the basis of 2,048 possible combinations of DSM–5 criteria (i.e., 2+ criteria) (Lane & Sher, 2015). Therefore, two individuals could receive the same diagnosis despite presenting with totally different symptoms. Nevertheless, despite these differences and issues regarding diagnostic heterogeneity, researchers typically adopt DSM operational criteria over ICD (Clark et al., 2017). Therefore, herein, AUD will be used to refer to a DSM–5 diagnosis of alcohol use disorder and the term alcohol use disorders (AUDs) will comprise both AUD and alcohol dependence.

Table 1.1 Diagnostic criteria for Alcohol Dependence and Alcohol Use Disorder in ICD–11 and DSM–5. Adapted from Saunders et al. (2019) under the Creative Commons Attribution 4.0 International License.

	ICD–11 Alcohol Dependence	DSM–5 Alcohol Use Disorder
Stem	Alcohol dependence is a disorder of regulation of alcohol use arising from repeated or continuous use of alcohol. The characteristic feature is a strong internal drive to use alcohol, which is manifested by impaired ability to control use, increasing priority given to use over other activities and persistence of use despite harm or negative consequences. These experiences are often accompanied by a subjective sensation of urge or craving to use alcohol. Physiological features of dependence may also be present, including tolerance to the effects of alcohol, withdrawal symptoms following cessation or reduction in use of alcohol, or repeated use of alcohol or pharmacologically similar substances to prevent or alleviate withdrawal symptoms. The features of dependence are usually evident over a period of at least 12 months but the diagnosis may be made if alcohol use is continuous (daily or almost daily) for at least 3 months.	A problematic pattern of alcohol use leading to clinically significant impairment or distress, as manifested by at least 2 of the following occurring within a 12-month period
1.	Impaired control over alcohol use (i.e., onset, frequency, intensity, duration, termination, context)	Craving or a strong desire or urge to use alcohol. There is persistent desire or unsuccessful efforts to cut down or control alcohol use. Alcohol is often taken in larger amounts or over a longer period than was intended.

2.	Increasing precedence of alcohol use over other aspects of life, including maintenance of health, and daily activities and responsibilities, such that alcohol use continues or escalates despite the occurrence of harm or negative consequences (e.g., repeated relationship disruption, occupational or scholastic consequences, negative impact on health)	Important social, occupational or recreational activities are given up or reduced because of alcohol use. Recurrent alcohol use resulting in a failure to fulfil major role obligations at work, school, or home (e.g., repeated absences or poor work performance). A great deal of time is spent in activities necessary to obtain alcohol, use alcohol, or recover from its effects. Alcohol use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by alcohol. Continued alcohol use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of alcohol.
3.	Physiological features indicative of neuroadaptation to the substance, including: 1) tolerance to the effects of alcohol or a need to use increasing amounts of alcohol to achieve the same effect; 2) withdrawal symptoms following cessation or reduction in use of alcohol, or 3) repeated use of alcohol or pharmacologically similar substances to prevent or alleviate withdrawal symptoms.	Tolerance is defined by either of the following: (i) a need for markedly increased amounts of alcohol to achieve intoxication or desired effect or (ii) a markedly diminished effect with continued use of the same amount of alcohol. Withdrawal, as manifested by either of the following: (i) the characteristic withdrawal syndrome for alcohol, or (ii) alcohol (or a closely related substance, such as a benzodiazepine) is taken to relieve or avoid withdrawal symptoms.
		Recurrent alcohol use in situations in which it is physically hazardous

Note. The shading indicates the nearest equivalent set of criteria in each system. ICD–11 = International Classification of Diseases 11th edition (World Health Organization, 2019); DSM–5 = Diagnostic and Statistical Manual of Mental Disorders 5th edition (American Psychiatric Association, 2013). The DSM–5 further classifies Alcohol Use Disorder into three categories depending on the number of symptoms present: mild = 2–3 symptoms, moderate = 4–5 symptoms, and severe = 6+ symptoms

Comorbidity

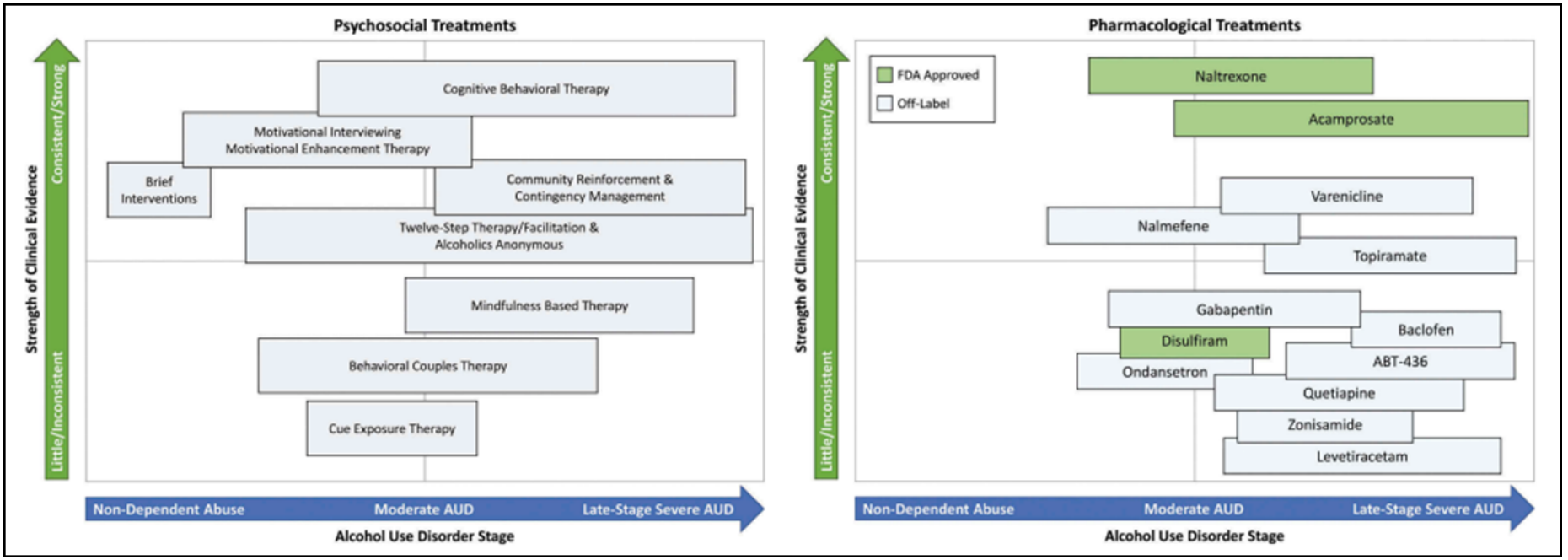
AUDs have considerable comorbidity (i.e., diagnostic co–occurrence) with a plethora of other psychiatric disorders (see Castillo-Carniglia et al., 2019 for review), These include externalising disorders (e.g., Krueger et al., 2002), internalising disorders (e.g., Fernandes et al., 2020), personality disorders (Newton-Howes & Foulds, 2018), and other substance use disorders (SUDs) (e.g., Glass et al., 2014). Possible reasons for this high level of comorbidity may be diagnostic arteifacts (e.g., poor construct validity, whereby arbitrary boundaries between disorders are drawn where, in reality, they do not exist) (Boness et al., 2021), shared causal mechanisms (Krueger et al., 2002), causal associations between disorders (Krueger & Markon, 2006), or dysregulation in some higher-order factor (e.g., the p–factor) (Conway et al., 2021).

Current treatments

Treatment rates for AUDs are generally low despite prevalence estimates indicating a significant public health concern. For instance, a recent meta–analysis suggests that, globally, only one in six people suffering with AUDs receive treatment (Mekonen et al., 2021). Nevertheless, several behavioural (i.e., psychotherapeutic) and pharmacological interventions for AUDs exist (see Figure 1.3). In a recent review, Ray et al. (2019) overview the evidence in favour of the available AUD interventions, suggesting that current treatments are only modestly effective. The efficacy of each treatment is highly dependent on individual–level factors, such as the motivation to reduce or stop drinking, misconceptions about treatments (e.g., inaccurate perceptions about the side effects of pharmacological intervention), social barriers (e.g., worries about stigma), access to treatment services, disorder severity, and disorder comorbidity (Mann et al., 2004; Mellinger et al., 2018; Morgenstern et al., 2016; Ray et al., 2019). Thus, treatment for AUDs should be prescribed in an individual, graded fashion. Despite this, in their "best practice" guidance document, the National Institute for Health Care Excellence[3] highlights that current practice and service access varies wildly across England, and that current care

[3] The National Institute for Health Care Excellence provide current best practice in health and social care in England and Wales.

Figure 1.3 A summary of the available psychological and pharmacological treatments for Alcohol Use Disorder (AUD). The strength of evidence in favour of a particular treatment is presented on the y–axis and the recommended use of a treatment across the AUD severity spectrum is shown on the x–axis. Reproduced from Ray et al. (2019) under Creative Commons Attribution 4.0 International License.



pathways are often ill defined (National Institute for Health Care Excellence, 2011). Overall, there is a large scope for improvement with regards to the treatment of AUDs.

A mechanistic approach to diagnosis and management

Personalised intervention (i.e., precision medicine) appears to be a goal that the alcohol research community has been working towards in an effort to improve the diagnosis and treatment of AUDs (Litten et al., 2015). Personalised intervention involves identifying the best treatment matches for individual patients through their unique profile of dysfunction (Wakefield, 1992). Therefore, attempting to find a solution to the issues surrounding the development, diagnosis, and management of AUDs, research has shifted towards identifying and understanding both etiologic (i.e., related to the development of a disorder) and maintenance (i.e., associated with the chronicity of a disorder) mechanisms (Boness et al., 2021; Keyser-Marcus et al., 2021; Kotov et al., 2017; Kwako et al., 2016; Sanislow et al., 2010; Sher, 2015). This effort has culminated in the development of the Etiologic, Theory–based, Ontogenetic Hierarchical Framework (ETOH) (see Boness et al., 2021 for review). Briefly, the ETOH contains three broad domains of reward (e.g., habit, incentive salience, and discounting), cognitive control (e.g., impulsivity and compulsivity), and negative valence and emotionality (e.g., negative emotionality and issues with coping). Each domain comprises several subdomains (examples are given in parentheses above) which are thought to describe processes which cause or maintain AUDs. Organising related mechanisms into higher–order domains in this way is hoped to improve the diagnosis and treatment of AUDs. In practice, this may involve administering a battery of psychometric tests to a patient to better understand the specifics of their dysfunction (e.g., the patient may present as impulsive with high levels of negative emotionality due to trauma), and then prescribing interventions designed to target these specific issues. Furthermore, data–driven approaches could be applied to aid clinicians in selecting the most effective intervention(s) (Fisher et al., 2019), thus reducing costs in terms of both time and money. However, if this new era of diagnosis and management is to be successful, further empirical work enhancing our understanding of the mechanisms behind AUDs is required (Boness & Witkiewitz, 2022).

1.2. Theories of addiction

1.2.1. The Brain Disease Model of Addiction (BDMA)

For over a quarter of a century, the dominant theory in addiction science has been Brain Disease Model of Addiction (BDMA) (Leshner, 1997). When initially proposed, the BDMA attempted to combat the previously established non-scientific view that addiction is a result of a moral failing, thus putting the blame on the addicted individual (Matano & Wanat, 2000). The BDMA posits that addiction is a chronic, relapsing disorder, characterised by compulsive drug use, increased negative emotionality during withdrawal, and a loss of control over drug intake (e.g., Koob & Volkow, 2016). More specifically, addiction is thought to arise as a result of dysregulated motivational systems in the brain, which over time worsen through repeated drug use and the cycle / spiral of addiction (Koob & Le Moal, 1997).

Koob and Le Moal (1997) describe how the cycle begins with binge–intoxication, which is characterised by heavy drug use and the experience of substance–induced hedonic effects. Next, comes withdrawal–negative affect which involves increases in stress and anxiety during withdrawal and subsequent surges in drug use to lessen said effects of abstinence (i.e., negative reinforcement). Finally, during preoccupation–anticipation, the addicted person is thought to experience intense cravings in relation to the substance in question after a period of abstinence. Subsequently, due to compromised executive functions (e.g., reduced self–control) in the preoccupation–anticipation phase, the individual will circle back to the binge–intoxication phase. Addiction is then thought to spiral out of control (i.e., progress) due to repeated failures in self–regulation (e.g., drug use) at any given phase, resulting in increased allostatic load and subsequent drug use (i.e., an opponent–process whereby drugs are taken in greater amounts to compensate for increasing levels of withdrawal–associated distress and drug tolerance) (for reviews, see: Everitt & Robbins, 2016; Koob & Le Moal, 2001).

1.2.2. Is addiction a disorder of choice?

Since being advanced, the BDMA has been routinely criticised by other scientists (e.g., Hall et al., 2015; Hart, 2017; Heather et al., 2018, 2022; Heyman, 2013; Pickard et al., 2015). Key criticisms of the BDMA include the arguments that: addiction does not fit the criteria of a disease; people who are

addicted do not act compulsively, instead retaining the ability to make more advantageous choice (e.g., not using drugs); high remission rates are observed among addicts; and a specific brain pathology of addiction has not been identified (Heilig et al., 2021). Overall, critics of the BDMA propose that the disease model is deterministic and reductionist. Instead, an alternative prevailing theory suggests that addiction is a *disorder of choice*, by which addiction is a myopic and self–destructive operant behaviour that is determined by contextual variables (e.g., an individual's perceived value of drug use vs. other rewards, negative emotionality, and cumulative lifetime stress) (Heyman, 2009, 2013).

1.2.3. An integrated perspective

Attempting consensus, Heilig et al. (2021) offer an update on the brain disease view of addiction by acknowledging and addressing the criticisms mentioned earlier. Importantly, they assert that the BDMA and disorder of choice models are not mutually exclusive but complementary. For instance, the disorder of choice model does not totally discount the neurophysiological basis of addiction (Kurti & Dallery, 2012) and brain disease viewpoint advocates consider contextual factors in their approach (Heilig et al., 2016). Under this contemporary view, pre–existing vulnerabilities and chronic drug use disrupt areas in the brain associated with reward. In turn, these dysregulated motivational systems leave individuals with a profoundly compromised ability to make long–term advantageous choices.

1.2.4. Relevance to this thesis

Regardless of which framework is applied to addiction, it is clear that further research on the contextual factors which result in the development and maintenance of alcohol addiction is required. A good starting point is to further investigate factors which are considered across the theoretical playing field. For example, *stress* (see section 1.3.2) is a central tenant of both the BDMA and the disorder of choice viewpoints. In the BDMA, intense distress is experienced by an addicted individual during the addiction spiral (e.g., during the withdrawal–negative affect phase) (e.g., Koob & Le Moal, 2001). Furthermore, the overvaluation of short–term rewards underpins the disorder of choice theory and extends to negative reinforcers (e.g., the relief from stress following drug consumption). For instance, proponents of the disorder of choice model use arguments that the neuroscientific view of addiction (i.e., the BDMA) is

undermined by the fact that addicted individuals typically present with a *"deep seated intolerance of stress or boredom"* (e.g., Satel & Lilienfeld, 2014, p. 5).

Impulsivity (see section 1.3.1) is a core executive function (Diamond, 2013). Like stress, impulsivity is central in both the BDMA and disorder of choice theories. For example, in the BDMA model, executive functions are undermined by drug use (e.g., Koob & Le Moal, 1997; Koob & Volkow, 2016). Therefore, addicted individuals are more likely to act impulsively and make unwise *choices* (i.e., consume drugs). Moreover, in the seminal book, *Addiction: A Disorder of Choice* (2009), Heyman recognises impulsivity as a "predrug individual difference". Arguably however, impulsive choice is underlined throughout the disorder of choice model. For instance, in a more recent paper updating the disorder of choice model, Heyman (2013) describes how several empirical choice principles (e.g., delay discounting[4]) – which are known to reflect impulsivity (De Wit, 2008) – apply in the field of addiction.

Notwithstanding, alcohol use is harmful to the individual much sooner than the onset of addiction (e.g., Zhao et al., 2017, 2023). Thus, reducing alcohol misuse is a public health issue. Therefore, a greater understanding of central premorbid factors, such as impulsivity and stress, is important outside of the context of addiction.

1.3. Key risk factors for alcohol misuse and addiction

1.3.1. Impulse control

"Impulsivity" has existed in human vernacular for more than 500 years, playing a crucial role in our understanding of psychopathology since the genesis of psychology, psychiatry and philosophy (Bari & Robbins, 2013). Overall, impulsivity is implicated in almost all externalising behaviours (Beauchaine et al., 2017) and perhaps every psychiatric condition, including substance use and addiction (Caspi & Moffitt, 2018). Impulsivity is broadly defined as the tendency to act in haste and without foresight (Dalley et al., 2011). However, research has indicated that impulsivity is better explained in a multidimensional fashion: as a set of distinct personality traits and/or behaviours (e.g., Dalley & Robbins, 2017; Strickland & Johnson, 2020). For instance, clear, unambiguous operational definitions

[4]Delay discounting is the reduction in the present value of a future reward as the delay to that reward increases (Odum, 2011).

of impulsivity have proven elusive: both self–report and behavioural measures exist, however, the inter–correlations both within and between these self–report and behavioural assessments are typically low (Dalley & Robbins, 2017; Strickland & Johnson, 2020), despite each facet contributing significantly to relevant behaviours outside of the laboratory or clinic (Moeller et al., 2001). Though the reason for this incongruence is not clear (Dang et al., 2020), the considerable diversity in impulsivity measurement suggests that a triangulation approach (i.e., using multiple methods of assessment) is the best course of action if future research aims to identify the most important facet of impulsivity in the context of alcohol use (and elsewhere).

Self–report (i.e., questionnaire) assessments are based on a person's own evaluation of how they view themselves relative to others and are typically considered to measure the level of an individual's trait impulsivity (Vassileva & Conrod, 2019). In contrast, behavioural measures (i.e., neurocognitive tasks) can be used to measure both trait and state impulsivity (Cyders & Coskunpinar, 2011; Dougherty et al., 2009; Moeller et al., 2001). In other words, unlike questionnaires, neurocognitive tasks require continuous responding and therefore facilitate the assessment of within–person, moment–to–moment, fluctuations in (for example) impulsive choice or response inhibition. Applying this to addiction, questionnaire measures could be used to identify who is at future risk, whereas behavioural measures may better predict who is at immediate risk (Cyders & Coskunpinar, 2011).

Self–report measures

Historical theoretical models of personality generally propose that behaviour is directed by at least two independent systems that have a heritable biological basis: the avoidance system, which is broadly associated with trait anxiety; and the approach system, which reflects impulsivity (Cloninger, 1987; Costa & McCrae, 1985; Evenden, 1999; Eysenck, 1985). Some self–report measures of personality developed around this period conceptualised a multifaceted view of impulsivity. For example, the Barratt Impulsivity Scale, which was originally created by Barratt (1959), is one of the most widely utilised self–report impulsivity assessments (Strickland & Johnson, 2020). The current version of the scale, the Barratt Impulsivity Scale – 11 (BIS–11) fractionates impulsivity into three second–order

facets (motor impulsivity, non–planning impulsivity, and attentional impulsivity) as well as six other first order factors (attention, motor, self–control, cognitive complexity, perseverance, and cognitive instability) (Patton et al., 1995; Stanford et al., 2009).

Attempts to consolidate this array of measurements have been made. For example, during the development of the UPPS Impulsive Behaviour Scale[5], Whiteside & Lynam (2001) applied factor analysis to several already existing personality measurements, resulting in a four–factor structure, consisting of negative urgency (i.e., the tendency to act rashly under extreme negative emotions), lack of premeditation (i.e., the tendency to act without thinking), lack of perseverance (i.e., the inability to remain focused on a task), and sensation seeking (i.e., the tendency to seek out novel and thrilling experiences). More recent work (i.e., the UPPS– P) has updated the model to also include a positive urgency (i.e., the tendency to act rashly under extreme positive emotions) component (Cyders et al., 2007, 2014).

Behavioural measures

The creation of behavioural measures of impulsivity followed a trajectory that was largely independent from the development of questionnaire–based assessments (e.g., Vassileva & Conrod, 2019). Theoretical models have proposed as little as two, and as many as five, distinct impulsive behaviours (Strickland & Johnson, 2020). For example, Dalley and Ersche (2019) highlight that even at the most basic level, impulsivity is made up of at least two separate processes: moment–to–moment self–restraint (i.e., suppressing inappropriate anticipatory behaviour) and foresight (i.e., weighing up the cost of rash decisions). Consequently, a large and diverse battery of behavioural tasks have been developed (e.g., Cyders & Coskunpinar, 2011; Hamilton, Littlefield, et al., 2015; Hamilton, Mitchell, et al., 2015). Subsequently, in a recent review, Strickland & Johnson (2020) consider theoretical, empirical and sociocultural evidence surrounding impulsivity as a construct. In line with other work in this area (e.g., Cyders, 2015; Reynolds et al., 2008; Sharma et al., 2014), they identified four core behaviours related to impulsivity and their appropriate measurement (see Table 1.2 for overview).

[5] UPPS = Negative Urgency, Lack of Premeditation, Lack of Perseverance, and Sensation Seeking.

Table 1.2 Impulsive behaviour constructs and their commonly used measures. Adapted from Strickland & Johnson (2020) under the Creative Commons Attribution 4.0 International License.

Construct	Definition	Assessment
Response inhibition	Ability to withhold a prepotent response.	Go / No Go
		Stop Signal Task
		5-CSRTT (premature responses)
		Anti-saccade task
Delayed consequence sensitivity	Devaluation of a consequence as a function of its delay.	Delay discounting tasks
Attention	Capacity to selectively control allocation of attention and avoid interference.	Continuous Performance Test
		5-CSRTT (accuracy)
		Stroop Task
		Immediate or delayed memory task
		Anti-saccade task
Risk sensitivity	Sensitivity to risky or probabilistic decisions.	Probability discounting
		Balloon Analogue Risk Task

Impulsivity and alcohol use

Impulsivity (broadly defined) is a central tenant in contemporary theories of addiction (e.g., Boness et al., 2021; Heyman, 2013; Leshner, 1997), and it is considered as both a risk–factor for, and consequence of, drug use (De Wit, 2008). For example, in early work with stimulants, Ersche et al. (2010) found that non–drug–using siblings of stimulant–dependent individuals had elevated levels of impulsivity

compared to non–siblings, and that stimulant–dependent individuals had significantly higher levels of impulsivity than either group. These findings suggest that impulsivity represents a behavioural endophenotype (i.e., a measurable, heritable, intermediate phenotype that is detectable prior to the full-blown "disease" phenotype; Gottesman & Gould, 2003) that is, in this case, exacerbated by the use of drugs. Moreover, pre–clinical experimental work has shown that rodents with high levels of trait impulsivity (assessed using the 5–CSRTT) are more likely to self–administer cocaine and develop addiction–like symptoms (e.g., responding for drugs despite contingent adverse consequences [i.e., foot–shock]) compared to a low–impulsivity control group (Belin et al., 2008). Finally, neuroimaging and neurophysiological studies have identified functional differences related to impulsivity (e.g., Dalley & Ersche, 2019; Vassileva & Conrod, 2019; Voon et al., 2020). More specifically, from the neurobiological perspective, impulsivity is thought to arise from dysfunctional corticostriatal circuitry, due to over engagement from the striatum (nucleus accumbens, putamen / caudate), and poor top–down control from the cortices (anterior cingulate, orbitofrontal and inferior frontal regions) (Fineberg et al., 2014). For instance, in the context of addiction, the ability to wait (in both the short– and long–term) is mediated by pathways in the ventral striatum, and in terms of neurotransmission, abnormalities in dopamine, γ-aminobutyric acid, and serotonin transmission are linked to addiction propensity (Dalley & Ersche, 2019).

When considering alcohol use specifically, Khemiri et al. (2016) used a large national sample of twins to demonstrate the substantial genetic overlap between impulsivity and alcohol dependence (e.g., a bivariate heritability of 80% for males and 53% for females). Furthermore, Courtney et al. (2012) used structural equation modelling to investigate the associations between risk–sensitivity (measured using the Balloon Analogue Risk Task [BART]), delayed consequence sensitivity (assessed using a delay discounting task [DDT]), response inhibition (evaluated using a Stop Signal Task [SST]), and alcohol misuse. They found that both BART and DDT scores positively predicted alcohol–related outcomes (i.e., use and pathology). These findings are largely in–line with the self–report literature, whereby meta–analyses of UPPS– P traits indicate significant positive relationships between each trait and alcohol use (Coskunpinar et al., 2013; Stautz & Cooper, 2013). More specifically, drinking quantity

is most highly associated with lack of perseverance; drinking problems are most strongly predicted by urgency (both positive and negative); and alcohol dependence is most closely related to negative urgency and lack of premeditation (Coskunpinar et al., 2013) (see section 1.3.1 for construct definitions).

The studies reviewed thus far have focused on trait, rather than state impulsivity. However, experimental psychology studies suggest that alcohol–induced deficits in impulse control are associated with increased craving for alcohol and subsequent *ad libitum* alcohol consumption (Field & Jones, 2017; Jones et al., 2013; Weafer & Fillmore, 2008). More recent work has used ecological momentary assessment (Moskowitz & Young, 2006) methods to investigate relationships concerning within–person fluctuations in state impulsivity and alcohol use. Nevertheless, the findings are mixed. For instance, Stamates and Lau-Barraco (2020) suggest that day–to–day changes in impulsivity do not predict alcohol use or alcohol–related problems. However, increased levels of alcohol use and alcohol related problems were related to greater levels of impulsivity the next day. In contrast, Wonderlich et al. (2022) found that negative urgency increased prior to drinking, while positive urgency and sensation seeking decreased following alcohol consumption. Similarly, findings from Griffin and Trull (2021) suggest that moment–to–moment premeditation and sensation seeking are related to drinking and that the only trait–level facet of impulsivity related to alcohol use in daily life was premeditation. Regardless of the heterogeneity in these results, it is clear that impulsivity is central to alcohol use and addiction.

1.3.2. Stress

Stress is an umbrella term that is generally defined as any stimulus which promotes allostatic change through the disruption of homeostasis (i.e., the biological processes that keep an organism's internal environment within its limits) (Sterling & Eyer, 1988). Therefore, stressors can be physical (e.g., injury), chemical (e.g., alcohol), biological (e.g., pathogens), environmental (e.g., prison), or event–based (e.g., a relationship ending). Importantly, stress is thought to be experienced when an individual's perceived or actual ability to cope, either psychologically or physically, is in question (Cohen et al., 2016).

The type of stress experienced by an individual is predominantly determined by duration and severity (for reviews, see: Crosswell & Lockwood, 2020; Epel et al., 2018). *Acute stressors* are intense short–term exposures. For example, during the Trier Social Stress Test (TSST), which is a standardised laboratory procedure, participants are asked to give a speech and then perform a mental arithmetic task in front of a panel of judges (Kirschbaum et al., 1993). *Daily events* (i.e., daily hassles) are relatively minor difficulties that occur in daily life, such as being stuck in traffic or meeting a tight deadline. Such life events can be considered as chronic stressors depending on the frequency that a person experiences event–related threat (Epel et al., 2018). *Life events* are time–limited and episodic. For example, losing a job or a relationship ending. *Traumatic life events* include particularly dangerous life events which threaten the psychological or physical safety of an individual, such as the death of someone close or the experience of violence. Finally, *chronic stress* involves stressors that are present for longer periods of time, such as being unemployed or serving a prison sentence. However, the specific duration that defines the limits of chronic stress varies in the field from as little as four weeks to at least six months (Epel et al., 2018).

Measuring stress

Stress can be assessed using self–report questionnaires, behavioural data, or physiological assessment. For example, the Perceived Stress Scale measures how overwhelming a person finds their current life situation (Cohen et al., 1983); stress is accompanied by nonverbal behaviour across a diverse range of species (Whitehouse et al., 2022); and glucocorticoids, such as cortisol, are regularly used as a stress biomarker in research (Hellhammer et al., 2009). Importantly, however, there is generally a weak association between psychological and physiological responses to stress. For instance, in a review, Campbell and Ehlert (2012) found psychophysiological correspondence in only approximately 25% of studies which used the TSST to induce an acute stress response. As the stress response in a complex phenomenon involving cognitive, emotional, physiological, and behavioural response systems, this apparent desynchrony is thought to be caused by each component of the stress response representing an independent factor, rather than a single synchronised system (Campbell & Ehlert, 2012). Therefore, researchers interested in investigating stress should consider how both the timescale and severity of

stress is captured in their measurements. Furthermore, multiple stress measurements, covering the breadth of the stress response, should be considered to ensure that stress exposure measurement is accurate. Finally, the experience of stress can result in further stress from a different cause (Epel et al., 2018). For example, life events such as losing a job could result in chronic stress related to financial strain. This financial strain may be managed by moving to a cheaper, unsafe area. In turn, the chronic stress related to living in a more dangerous environment could give rise to further life events, such as being a victim of crime. Though such events do not always cascade in such a way, the cumulative effect of stress should be considered by researchers when investigating the effects of stress on health and behaviour.

Stress and alcohol use

Stress is a well–known risk–factor for alcohol use and addiction (for reviews, see: Jose et al., 2000; Ruisoto & Contador, 2019; Sinha, 2001). Among non–dependent drinkers, results from experimental human research have shown that acute stress increases craving for alcohol (Clay et al., 2018; Clay & Parker, 2018; Field & Powell, 2007), the perceived reinforcing value of alcohol (Owens et al., 2015), and voluntary drinking (Clay & Parker, 2018; De Wit et al., 2003; Magrys & Olmstead, 2015; McGrath et al., 2016). Similarly, Thomas et al. (2011) found that alcohol–dependent individuals were twice as likely to finish an alcoholic beverage, following acute stress, during an *ad libitum* drinking task.

Previous work has also found a link between daily hassles, alcohol use (e.g., Baer et al., 1987), and problem drinking (e.g., Takeshita et al., 1998). More recent work has used ecological momentary assessment methods to investigate the effect of day–to–day or moment–to–moment fluctuations in mood on alcohol–related outcomes. For instance, several studies suggest that daily alcohol use is associated with drinking to cope with changes in affect (Carney & Armeli, 2000; Duif et al., 2020; Dvorak et al., 2014). Similarly, Waters et al. (2020) demonstrate how negative affect predicts temptation to relapse among alcohol–dependent individuals attempting to maintain abstinence. However, in contrast, results from O'Donnell et al. (2019) suggest that momentary changes in affect are not related to drinking behaviour. Taken together, this body of work provides evidence that day–to–day increases in stress are positively associated with drinking behaviour.

Significant evidence suggests that life–events and chronic stressors are causally linked to addiction. For instance, in a review of the literature, Enoch (2011) posits that there is a direct route between chronic stress exposure during childhood (e.g., neglect, mistreatment) to problem drinking and ultimately dependence. However, as a substantial proportion of individuals do not go on to develop addictions, this pathway is likely moderated by gene–environment interactions which either bolster vulnerability or resilience. *Perhaps endophenotypes, such as impulsivity, are crucial here.* However, this hypothesis is yet to be tested substantively. Nevertheless, childhood is not the only period whereby adversity can influence substance use. For instance, service personnel are at a greater risk of exposure to traumatic life events and chronic stress due to the nature of their occupation, and are therefore more likely to misuse alcohol to cope with the experience post–traumatic stress disorder (e.g., Debell et al., 2014).

Some of the mechanisms by which stress increases individual propensity to consume drugs are known. For instance, extensive theoretical and empirical work affirms that the link between stress and the risk for alcohol misuse are the result of dysfunction (including both hypo– and hyper–activation) of the hypothalamic–pituitary–adrenal (HPA) axis (al'Absi, 2018; Koob & Kreek, 2007; Koob & Schulkin, 2019; Milivojevic & Sinha, 2018). Repeated stressor exposure results in neurophysiological changes to areas associated with emotional processing, stress reactivity, and reward regulation (Casement et al., 2015; Kim et al., 2013). For example, individuals who are dependent on drugs, such as alcohol, show hypercortisolism and a blunted stress response (for reviews, see: Blaine & Sinha, 2017; Sinha, 2001). Ultimately, these neurophysiological changes leave individuals in a dysregulated state and *unable to regulate their own emotions*. Therefore, such individuals are left at an increased risk of compensating through alcohol use.

Somewhat counterintuitively, alcohol is a powerful trigger for activation of stress systems, including the HPA axis (Armario, 2010; Milivojevic & Sinha, 2018) and an important underlying loop exists in which individuals are left unable to regulate their emotions due to stressful life experiences. In turn, increased emotional dysregulation (i.e., a person's inability to regulate their own emotions) means that individuals may choose to misuse alcohol, attempting to self-regulate (Wemm et al., 2022). Finally,

following periods of prolonged alcohol misuse (e.g., HED), neuroadaptations and the experience of more stressful life events (e.g., failing to fulfil commitments and interpersonal issues) may occur, exacerbating the emotional dysregulation and subsequent inability to cope (Carbia et al., 2021). Additional evidence for this is found in the clinical literature, where alcohol-dependent individuals (versus non-dependent social drinkers) commonly present with such neuroadaptations (Sinha, 2012). In sum, like suggested by the BDMA, a cycle of dysregulation seems to occur throughout addiction.

1.3.3. Sociocultural factors

There are several inter–related sociocultural factors that are strongly associated with alcohol use and alcohol–related harm, such as an individual's age, gender, ethnicity, socioeconomic status (SES), and place of residence (Room, 2013). Such influences should be considered in alcohol research wherever possible as they are critical to the onset and maintenance of addiction. However, these contributions are unlikely to be medically controllable (e.g., one cannot change their age). Therefore, outside of controlling for these aspects in experimental design and data analyses, the relative possible clinical utility of such factors is less than, for example, understanding the contribution of impulse control and stress to alcohol misuse.

Age

Historical data suggests that the total volume of alcohol consumed per week increases with age, while HED typically declines (NHS Digital, 2022a; Office for National Statistics, 2018). These changes are presumably driven by differences in circumstances and attitudes over the life course. However, there is evidence that older people of today may be relatively heavier drinkers compared to previous generations. For instance, data from the HSE–21 shows an upward trend in the total volume of alcohol consumed among those aged 75+, from approximately 6.5g of ethanol per week in 2016 to 13.3g in 2021 (NHS Digital, 2022a). In contrast, emerging evidence suggests a devaluation of alcohol among Generation Z (born between 1996 and 2015) (Kraus et al., 2020), suggesting that the disparity in alcohol use between generations may grow over time.

Gender

Men typically drink more than women. Large multinational surveys show that men are more likely than women to consume > 23g of alcohol per week, drink on 5 or more occasions per week, and binge drink (i.e., participate in HED) more frequently (Wilsnack et al., 2009). This is corroborated by data from HSE–21 which shows that, on average in 2021, men consumed 117.6g of ethanol per week, whereas women consumed 68.0g per week (NHS Digital, 2022a). Although it is unclear why these gender differences have persisted over time, several hypotheses have been put forward by Wilsnack & Wilsnack (2013). First, biological differences between men and women may play a role. For instance, there is evidence of the existence of sex–differences in the heritability of alcohol dependence (Prescott, 2002). Moreover, differences in metabolism and/or typical body mass may result in women needing to consume less alcohol to experience the same effects (Kerr et al., 2006; Kwo et al., 1998; Mumenthaler et al., 1999), which may explain why women are more sensitive to hangover symptoms (Slutske et al., 2003). Second, men may use alcohol to assert power over others as (for example) consuming large quantities of alcohol, especially without displaying signs of intoxication, is sometimes viewed as a masculine behaviour (Iwamoto et al., 2011; Lindsay & Lyons, 2018; Slutske et al., 2003). Third, intoxicated men may be viewed as sexually assertive, whereas women could be viewed as sexually disinhibited and promiscuous – making alcohol more rewarding for men and less rewarding for women (due to drinking potentially creating a moral or physical hazard for women) (Wilsnack & Wilsnack, 2013). Fourth, men are typically more risk–taking than women (Byrnes et al., 1999; Sapienza et al., 2009). Finally, stereotypical gender roles may facilitate drinking among men and limit drinking among women (Holmila & Raitasalo, 2005)

Ethnicity

Though not a homogenous group, rates of abstention from alcohol and other drugs are typically higher among those from minority ethnic backgrounds, compared to white individuals (Alcohol Change UK, 2019), and heavy episodic drinking rates are usually lower (Twigg & Moon, 2013). Research aiming to understand the effects of ethnicity on drinking is generally sparse. However, these differences are thought to be partly attributable to the stigmatisation of alcohol use among minority ethnic groups.

(Room, 2005; Zapolski et al., 2014). For instance, where there is a religious restriction on alcohol (e.g., Islam) (Institute of Alcohol Studies, 2020). However, due to (for example) this stigmatisation of alcohol and systemic racial discrimination, individuals that belong to these cultural groups who do drink may be disproportionately affected by alcohol–related harm. For instance, Zapolski et al. (2014) suggests that black men are more likely to face legal problems related to drinking, compared white men, at the same level of alcohol consumption. Moreover, people belonging to minority ethic groups may be less likely to seek help until they have faced serious health consequences, perhaps due to the fear of within–group social disapproval when alcohol use and alcohol–related problems occur (Institute of Alcohol Studies, 2020). Finally, some research suggests that younger people who belong to minority ethnic groups can experience increased acculturative stress related to the navigation of parental tradition and peer norms of their host country, which may paradoxically increase the risk of substance use (Marsiglia et al., 2012).

Socioeconomic status (SES)

SES reflects an individual's social position within society, usually encompassing income level, education level, occupational prestige, and place of residence (Baker, 2014). Those with lower SES (i.e., who are more disadvantaged) tend to consume less alcohol than their more privileged counterparts, yet typically experience more alcohol–related harm, reflected by consistently greater alcohol–related morbidity and mortality rates in these groups (Mackenbach et al., 2008; Probst et al., 2020). This observation is known as the alcohol–harm paradox (Bellis et al., 2016; Lewer et al., 2016). Although the alcohol–harm paradox is not fully understood, one theory it is caused by the link between socioeconomic disadvantage and stress exposure (e.g., financial concerns, unemployment, issues with inter–personal relationships, discrimination, and isolation) (Baum et al., 1999; Lantz et al., 2005). Biological evidence supports this theory, suggesting that that deprivation is positively associated with elevated stress hormone levels (Cohen et al., 2006). Therefore, as the relationship between stress and poor health is well–established (Thayer et al., 2012), the effects of alcohol consumption on health may be compounded by disadvantage. In addition, some disadvantaged people may drink to cope as a means of emotional regulation (Lantz et al., 2005; Wilkinson, 2002). Another explanation may be that

deprivation is associated with worse access to essential services e.g., health care (Butler et al., 2013). Therefore, individuals living in deprived areas who are suffering with alcohol–related conditions may not be able to access the care that they need. Finally, HED, which is known to be more harmful than spreading out alcohol consumption over multiple occasions, is more prevalent among lower SES groups (Bellis et al., 2016; Probst et al., 2020).

Place of residence

Place of residence is closely related to deprivation. For instance, the distribution of deprivation in England is highly skewed, whereby Northern areas are typically more deprived than Southern locations – Figure 1.4 (Ministry of Housing, Communities & Local Government, 2019). Related to this, Twigg & Moon (2013) report a clear north–south divide in rates of HED in the UK, suggesting that these differences are likely driven by differences in both area–based deprivation level and regional drinking cultures. Furthermore, when considering rural vs. urban areas, a recent international review shows that the majority of research completed between 1990 and 2019 found that rural (compared to urban) populations are at greater risk of hazardous alcohol use and alcohol–related harm (Friesen et al., 2022). However, outlet density, which is associated with deprivation level, alcohol consumption, and alcohol–related harm, is typically greater in urban populations (Alcohol Research UK, 2018).

1.4. Chapter summary

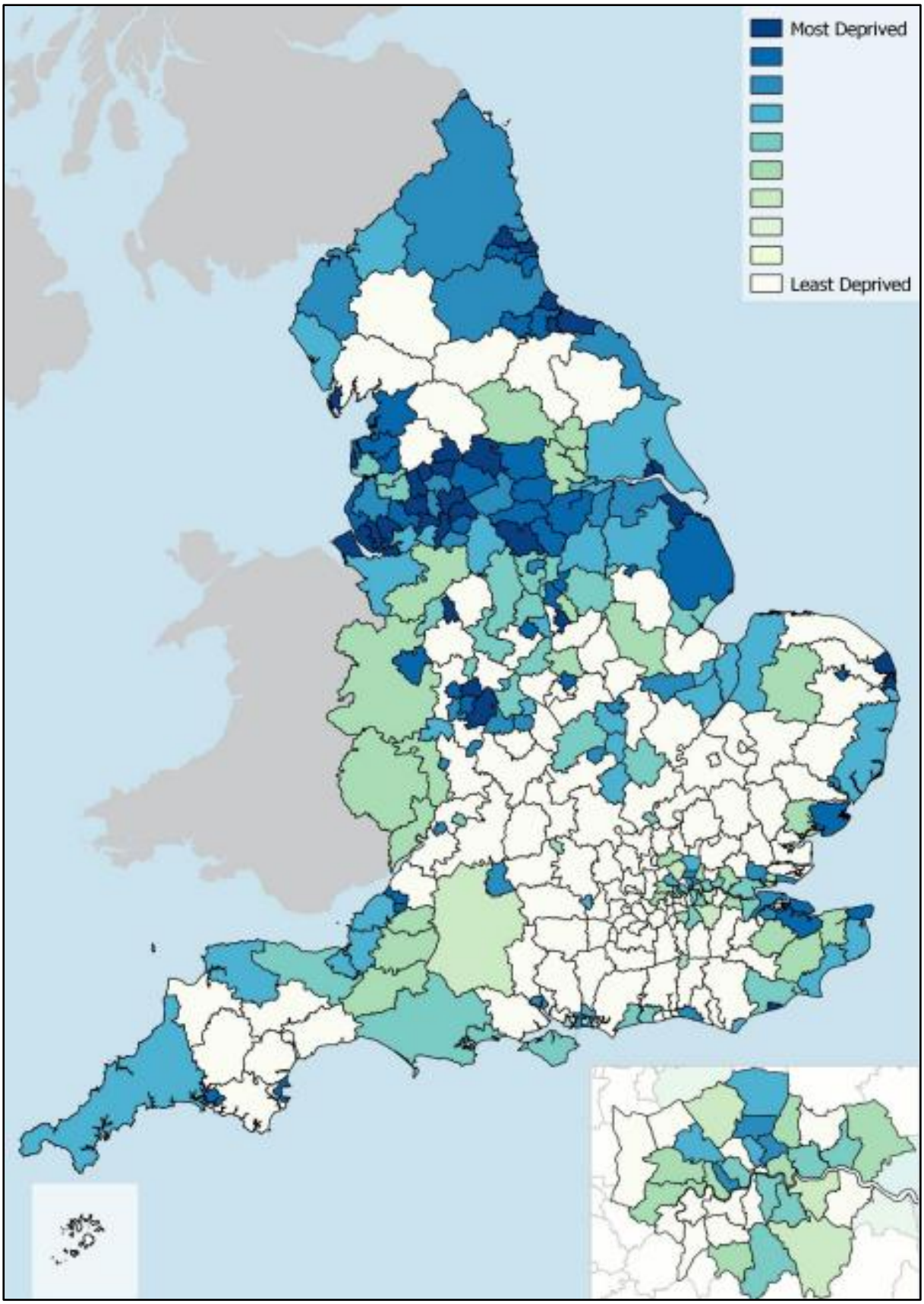
Alcohol is a powerful psychoactive drug that is readily available and regularly consumed. Despite intoxication often being pleasurable for many and alcohol consumption being embedded in society, the public health, sociocultural impacts, and economic impacts of alcohol–related harm are substantial. For instance, unbridled alcohol consumption can escalate into an addiction, resulting in many people suffering with AUDs. Despite this, current diagnostic approaches (e.g., ICD–11 and DSM–5) are not reliably detecting distinguishing characteristics of AUDs, as reflected by the high comorbidity between AUDs and other disorders, high treatment self–referral rates, and the general underdiagnosis of AUDs by clinical professionals. Furthermore, treatment rates for AUDs are low and current interventions are only modestly effective. Therefore, it is hoped that a better understanding of key mechanisms which

underly the development and maintenance of alcohol misuse, such as impulse control and stress, will improve efficacy of the diagnosis and treatment of alcohol misuse and AUDs.

1.4.1. Overarching aims

The overall aim of this thesis is to better understand the impact of impulsivity on alcohol use in the context of acute, chronic, and cumulative lifetime stress. Specifically, the hypothesis that the effects of stress on alcohol use behaviours will be strengthened by impulsivity will be tested across several studies ranging from experimental studies to large–scale national cohort approaches.

Figure 1.4 The distribution of the English Index of Multiple Deprivation 2019 by local authority. Reproduced from Ministry of Housing, Communities & Local Government (2019) under the UK Open Government Licence.



Chapter 2. The Role of Impulse Control on Craving and Consumption of Alcohol Following Acute Stress: A Laboratory Study

Chapter Foreword

Results from a laboratory study are presented here in the first empirical chapter of this thesis. This study was conducted to investigate the role of impulse control on stress–induced craving and consumption of alcohol in the context of acute stress, are reported. This study builds on work completed during my Bachelor's and Master's degrees. Specifically, in Clay et al. (2018) and Clay & Parker (2018), we demonstrated how acute psychosocial stress increases craving for, and consumption of, alcohol, respectively. We also found that these effects were strengthened by risk–taking. This chapter contributes to the overall aims of the thesis by investigating the impact of impulsivity on alcohol craving and consumption following several commonly used, laboratory–based, acute stress tasks. Data collection for this study began in 2019 and was therefore interrupted by the COVID–19 pandemic. Following the pandemic, several procedural changes were necessary to ensure participant safety. Each amendment had to then be reviewed by the University of Portsmouth Science Faculty Ethics Committee. Together, these delays resulted in a lower–than–expected sample size.

Abstract

Impulsivity and stress are both well–established risk–factors for the onset and maintenance of alcohol dependence and alcohol use disorder (AUD). Previous research has demonstrated that the laboratory–based acute stress tasks result in increases in craving for alcohol, the perceived reinforcing value of alcohol, and voluntary drinking in samples of social drinkers. There is also evidence to suggest that poor impulse control strengthens these relationships. However, no work has directly compared the role of impulse control in the response to different types of acute stress in terms of alcohol use behaviour. To address this, we exposed 107 participants (65 females) aged 18–47 years old (M = 23.44, SD = 5.55) to either the Trier Social Stress Test (TSST; n = 22), Maastricht Acute Stress Test (MAST; n = 31), a Cold Pressor Task (CPT; n = 24), or a no stress control (n = 30). Prior to completing these tasks, participant's trait impulsivity was characterised using a battery of tests. Physiological and psychological stress responses and craving were measured before and after each manipulation. Participants then completed a voluntary drinking task, whereby they were asked to consume a single alcoholic beverage (50mL of 37.5% ABV vodka diluted with 250mL of mixer). The time to finish the drink was covertly measured. The stress manipulation was most robust in the TSST group, followed by the MAST group. The CPT task did not successfully induce stress. In the TSST group, negative urgency and negative affect were associated with increased stress–induced craving. However, like in previous work, craving was not associated with drinking duration. In conclusion, data from the present study suggests that that clinical interventions which aim to reduce negative urgency and negative affect may be potential targets for personalised interventions for individuals who are at risk of developing alcohol dependence in the future.

Ethnics Approval Reference: SHFEC 2019–123A (Appendix A)

2.1. Introduction

Impulsivity is generally defined as the tendency to act in haste and without foresight (Dalley et al., 2011). However, recent work has shown that impulsivity is better characterised as a multifaceted construct which is made up by several traits (assessed using questionnaires) and behaviours (measured using computer tasks) (for reviews, see: Dalley & Robbins, 2017; Strickland & Johnson, 2020). Despite this complexity, there is a clear link between poor impulse control and addiction (including AUDs), in terms of both onset and maintenance (Dalley & Ersche, 2019; Lee et al., 2019). For example, in a sample of 457 young adults, "impulsive sensation seeking" and "behavioural disinhibition" traits were linked to an increased risk of a SUD diagnosis six years later (Sher et al., 2000). Similarly, Kirisci et al (2007) show that high levels of childhood (at 10–12 years of age) "neurobehavioural disinhibition" predicts SUD diagnosis at age 22. This link is further evidenced by numerous pre–clinical (e.g., Belin et al., 2008; Kreek et al., 2005), neuroimaging (e.g., Bosker et al., 2017; Voon et al., 2020), and heritability (e.g., Karlsson Linnér et al., 2021) studies.

In terms of alcohol use specifically, there is a substantial genetic overlap (e.g., a bivariate heritability of 80% for males and 53% for females) between impulsivity and alcohol dependence (Khemiri et al., 2016). Furthermore, in terms of impulsive behaviour, both risk–sensitivity and delayed consequence sensitivity (see section 1.3.1) significantly predict alcohol use and AUD pathology (Courtney et al., 2012). Meanwhile, meta–analyses of UPPS– P traits (see section 1.3.1) shows that drinking quantity is most highly associated with lack of perseverance; drinking problems are most strongly predicted by urgency (both positive and negative); and alcohol dependence is most closely related to negative urgency and lack of premeditation (Coskunpinar et al., 2013).

Stress is also a well–established predictor of alcohol use and addiction (for reviews, see: Jose et al., 2000; Ruisoto & Contador, 2019; Sinha, 2001). Laboratory–based stressors are frequently employed to study the psychobiological systems that underlie the relationship between *acute stress* exposure and alcohol use (for review, see: Thomas et al., 2012). Acute stressors are intense and short–lived exposures (Crosswell & Lockwood, 2020; Epel et al., 2018): for example, an argument with a romantic partner or delivering an important presentation at work. Previous research has demonstrated

that the laboratory–based acute stress tasks result in increases in craving for alcohol (Clay et al., 2018; Clay & Parker, 2018; Field & Powell, 2007), the perceived reinforcing value of alcohol (Owens et al., 2015), and voluntary drinking (Clay & Parker, 2018; De Wit et al., 2003; Magrys & Olmstead, 2015; McGrath et al., 2016) among social drinkers. Similarly, laboratory–based acute stress has been shown to increase voluntary drinking among alcohol–dependent individuals (Thomas et al., 2011).

Common methods of acute stress induction rely on pain (e.g., the cold pressor task [CPT] Mitchell et al., 2004), psychosocial stress (e.g., the Trier Social Stress Test [TSST] Kirschbaum et al., 1993), or pharmacological manipulation (e.g., insulin Costa et al., 1996) to stimulate the stress response. However, there are nuanced differences in the neuroendocrine response to each phenomenon. For instance, regardingtake pain and stress; both could be considered as two sides of the same coin, whereby pain is conceptualised as a form of acute stress (Abdallah & Geha, 2017). However, the processing of acute pain and acute stress can be different.

Acute stress triggers the hypothalamic–pituitary–adrenal (HPA) axis, resulting in adrenal glucocorticoid release (McEwen, 2007). These hormones bind to receptors which are concentrated in the limbic system (Morimoto et al., 1996; Sánchez et al., 2000). The limbic system and HPA axis form an interconnected loop, whereby the limbic feeds information back to the hypothalamus to regulate the stress response and future glucocorticoid release (Lupien et al., 2009). Furthermore, autonomic system activation during the response to stress results in physiological changes, such as increased blood pressure and diversion of blood from the gastrointestinal system to the brain and muscles (McEwen, 2007; Saper, 2002). Ultimately, these changes result in altered emotional states (e.g., increased anxiety) and behavioural adjustments (e.g., fight or flight).

The response to acute pain begins with the conscious perception of nociceptive information, which is sent to the brainstem and thalamus, and then relayed to several cortical and subcortical areas (Coizet et al., 2010; Klop et al., 2005; Newman et al., 1996). Like stress, pain activates the autonomic nervous system (McEwen, 2007; Saper, 2000). Moreover, there is significant overlap between acute stress and pain at the brain level (Sinha et al., 2016; Tracey & Mantyh, 2007). Unlike stress, however, there is no clear link between acute pain, HPA axis activation and subsequent adrenal glucocorticoid

release (Abdallah & Geha, 2017). Therefore, due to the existence of neurophysiological differences between stress and pain, it would be sensible to assume that stress and pain could differentially affect craving for, and consumption of, alcohol. However, to the best of our knowledge, no study has investigated this specifically.

The first aim of this study was, therefore, to compare craving and drinking following an acute pain task, a psychosocial stress task, and a mixed stress (pain + psychosocial stress) task. Here, we hypothesised that craving and drinking would increase as a function of stress intensity. Furthermore, we have previously shown how poor impulse control acts as a moderator for craving and drinking, in times of acute stress, by strengthening the relationship between stress and alcohol use behaviour in social drinkers (Clay et al., 2018; Clay & Parker, 2018). Therefore, the second aim of this study was to test the hypothesis that impulse control would influence stress–induced craving and drinking, with more impulsive individuals showing higher levels of stress–induced craving and faster alcohol consumption during a voluntary drinking task.

2.2. Methods

2.2.1. Design

A mixed design with both between–subject and within–subject independent variables was used. There was one between–subject independent variable with four levels (group allocation: either TSST, the Maastricht Acute Stress Test [MAST], a CPT, or the control group). There was one within–subject independent variable with three levels (time: T1 = baseline, T2 = during the stress manipulation, and T3 = immediately after the stress manipulation). The main dependent variables were response to stress (both physiological and psychological), craving for alcohol, and alcohol consumption during a voluntary drinking task. Impulsivity (assessed using both questionnaire–based and computer task measures) served as a potential explanatory variable. Several potential covariates were also assessed (e.g., sociodemographic information, sensory properties of the drink provided during the voluntary drinking task, and stimulant and sedative effects of the drink).

2.2.2. Transparency and openness

We report how we determined our sample size, all data exclusions, all manipulations, and all measures in the study. For the manipulation check, a sensitivity power analysis conducted in G* Power (version 3.1.9.7) revealed that with N = 107, power (1 – β) = 80%, and α = 0.05, we could reliably detect a time x group interaction with an effect size of ηp2 = 0.14 for analyses with two repeated measures. For the main analyses, a sensitivity power analysis conducted in G* Power revealed that with N = 107, power (1 – β) = 80%, and α = 0.05, we could reliably detect a one–way group difference with an effect size of ηp2 = 0.10. Similarly, with n = 22 (i.e., our smallest group size), power (1 – β) = 80%, and α = 0.05, we could reliably detect a group–wise correlation with an effect size of r = .56. As neither the study nor the analysis plan was pre–registered, the results should be considered exploratory. All data were analysed in IBM SPSS (version 28) and R (version 4.2.3) for Windows.

2.2.3. Sample

Participants were 107 adults (65 females, 42 males) ranging in age from 18–47 years old (M = 23.44, SD = 5.55) who were either studying at the University of Portsmouth or members of the local community. Participants were recruited using opportunity sampling (i.e., through advertising or word–of–mouth). The advert informed individuals that they would be required to take part in a mild stress test and consume an alcoholic beverage. However, specific details of the procedure were withheld. Participants were compensated for their time through course credits or via a payment of £15. To confirm the suitability, potential participants were sent a screening questionnaire via email. Participants were required to be aged between 18–55 years (self–report); in good physical and psychiatric health (self–report); fluent in English (determined by lead researcher); and consume alcohol at least monthly (self–report). Exclusion criteria included alcohol consumption less than 12 hours before the study session (self–report and confirmed via breathalyser); pregnancy or breastfeeding (self–report); treatment for psychiatric illness in the last year (self–report), ongoing physical illness or abnormality (self–report); heavy nicotine or caffeine use (self–report); a score greater than 5 on the Patient Health Questionnaire for Depression and Anxiety (PHQ–4; Kroenke et al., 2009), indicating possible anxiety and/or

depression; and a score greater than 20 on the Alcohol Use Disorders Identification Test (AUDIT; Babor et al., 2001), indicating possible alcohol dependence.

2.2.4. Impulsivity

Short UPPS–P Impulsivity Scale (SUPPS–P)

The S–UPPSP was used to assess negative urgency (i.e., the tendency to act rashly under extreme negative emotions), lack of premeditation (i.e., the tendency to act without thinking), lack of perseverance (i.e., the inability to remain focused on a task), sensation seeking (i.e., the tendency to seek out novel and thrilling experiences), and positive urgency (i.e., the tendency to act rashly under extreme positive emotions) (Cyders et al., 2014). The S–UPPSP is a 20–item questionnaire in which participants rate several statements related to their impulsive behaviour on a four–point Likert–type scale (1 = *Agree strongly*; 2 = *Agree some*; 3 = *Disagree some*; 4 = *Disagree strongly*). Each subscale is made up of four items; therefore, the maximum score on each subscale is 16, with higher scores reflecting greater impulsivity. In this study, the internal consistency (Cronbach's α) of each subscale ranged from 0.67 to 0.80.

Balloon Analogue Risk Task (BART)

The BART was used to establish risk sensitivity (Lejuez et al., 2002). The BART, which is a proxy measure of 'real world' risk–taking, requires participants to inflate a virtual balloon by pressing the spacebar. Each space bar press earns the participant £0.05 of virtual currency which can be "banked" by pressing the return key. Each balloon has a randomly allocated tolerance and over–inflation will cause the balloon to burst, losing the amount earnt (unbanked) in that trial. An array of 128 numbers were randomly sampled without replacement to set the tolerance of each balloon. As the probability of balloon exploding increases with successive pumps and the task was limited to 30 trials, a selection of trials with a mean burst point of 64 pumps was selected to match that of the original paper (Fernie et al., 2010; Lejuez et al., 2002). The dependent variable for this task is the average number of space–bar presses for unburst balloons, reflecting greater risk–taking.

Titrating Alternatives Delay Discounting Task (TADD)

Delay discounting (i.e., the reduction in the present value of a future reward as the delay to that reward increases) (Odum, 2011) was assessed using the TADD (Du et al., 2002; Rung et al., 2018). During this task, participants choose either 'smaller–sooner' or 'larger–later' (e.g., £250 now OR £1,000 in one year) by pressing the 'c' and 'm' keys, respectively. In each trial, the smaller–sooner reward was displayed on the left while the larger–later reward was shown on the right. The current delay interval (e.g., "The delay for the options on the right is now 1 WEEK") for that trial will be displayed at the top of the screen. When the smaller–sooner reward was chosen, the amount of the smaller–sooner reward was reduced by 50% in the subsequent trial. Whereas, if the larger–later reward was chosen, the smaller–sooner reward increased by 50% on the next trial. Overall, this titration procedure was repeated over seven blocks of eight trials, where each block represents a different delay interval (i.e., 1 week, 2 weeks, 1 month, 6 months, 1 year, 5 years, and 25 years). To quantify delay discounting, we calculated both area under the curve (AUC) (Myerson et al., 2001) and k (Gray et al., 2016). Unlike k, AUC provides a simple atheoretical measure of delay discounting (Bickel & Marsch, 2001; Field et al., 2007), with smaller values (between 0 and 1) reflecting grater delay discounting. Therefore, AUC values were reversed (1 – score), so that greater values represent greater discounting. Previous research has shown the quantification of delay discounting via AUC to be comparable to more conventional curve–fitting techniques (e.g., k) (Basile & Toplak, 2015; Odum & Rainaud, 2003). In the present study, a Spearman's rank correlation indicated a strong relationship between AUC and k, $r_s = .91$, $p < .001$. Therefore, AUC was used in the analysis.

Stop Signal Task (SST)

Participants completed the SST to assess response inhibition (i.e., the ability to withhold inappropriate action) (Lappin & Eriksen, 1966; Logan & Cowan, 1984; Verbruggen et al., 2019). Participants were instructed to *"Respond to an arrow, which is surrounded by a white circle, pointing either left or right, as fast as you can…However, if the arrow is surrounded by a RED CIRCLE, you should NOT RESPOND"*. Each trial began with a white fixation cross in the centre of the screen which remained for 500ms followed by a 500ms inter–stimulus interval. Next, an arrow pointing either left or right was

presented for 1000ms in the middle of the frame. In "go" trials, participants were required to respond by indicating if an arrow surrounded by a white circle (go–signal) was pointing left (with the 'c' button on a keyboard) or right (with the 'm' button). In "stop signal" trials, the arrow was surrounded by a red circle (stop–signal) after a brief delay, and participants were instructed to withhold their response.

During the first stop trial, the stop–signal occurred 50ms after the presentation of the go–signal and remained for the remaining duration of the trial (950ms). In subsequent trials, the latency of the onset and the corresponding duration of the stop–signal varied (by 100ms) based on how the participant performed during the previous stop–signal trial; where the maximum and minimum values for the latency of the onset of a stop–signal was 350ms and 50ms, respectively. Therefore, there were four possible values: 50ms, 150ms, 250ms, 350ms. Finally, participants were presented with visual feedback indicating how they performed on the previous trial. After a correct response, the feedback message said *"Correct"*, following an incorrect response to a go–signal (i.e., not responding), the feedback message said *"You should have pressed!"*, and after an incorrect response to a stop–signal, the feedback message said *"You should NOT have pressed!"*. Overall, participants completed an initial training block of 16 trials (12 go trials and 4 stop trials) followed by an experimental block of 200 trials (150 go trials and 50 stop trials). Each block was fully randomised. The main dependent variable for the SST is the stop signal reaction time (SSRT), which reflects the latency of the stop process (Verbruggen et al., 2019). Following Verbruggen et al. (2019), other relevant variables relating to the SST are reported in the Appendix A[6].

2.2.5. Stress response

Physiological

Physiological responses to stress were assessed using heart rate (HR), heart rate variability (HRV), systolic blood pressure (SBP), and diastolic blood pressure (DBP)[7]. HR and HRV data were collected continuously throughout the study using a Polar A300 Activity Tracker and a Polar H7 Heart Rate

[6] The task parameters indicated that this data was unreliable according to Verbruggen et al. (2019). Therefore, the task was omitted from analyses. Towards transparency and openness, we report the task parameters and rationale for omitting this measure from the analyses in Appendix A.

[7] We had planned to additionally measure salivary cortisol. However, due to the ongoing COVID–19 pandemic at the time of data collection, it was deemed unsafe to collect saliva samples by the University of Portsmouth.

Sensor (Polar Electro, Finland). HRVanalysis (version 1.2) for Windows was used to process the HRV data and calculate the standard deviation of normal–to–normal intervals (SDNN) and the root mean square of successive differences between normal heartbeats (RMSSD) (Pichot et al., 2016). Blood pressure was measured an Omron M2 Upper Arm Monitor (Omron, The Netherlands). Mean arterial pressure (MAP) was calculated using the SBP and DBP data, where:

$$\mathrm{MAP} = \mathrm{DBP} + 1 / 3(\mathrm{SBP} - \mathrm{DBP}) \quad (1)$$

Psychological

Psychological responses to stress were assessed using two questionnaires with good psychometric properties: the Positive Negative Affect Schedule (PANAS; Watson & Clark, 1988) and the six–item short–form version of the state scale of the State–Trait Anxiety Inventory (STAI; Marteau & Bekker, 1992). The PANAS consists of two ten–item subscales designed to measure both positive affect (PA) and negative affect (NA) at the time of administration. When completing the PANAS, participants are required to rate a number of words relating to their mood on a five–point Likert–type scale (1 = *Very slightly or not al all*; 2 = *A little*; 3 = *Moderately*; 4 = *Quite a bit*; 5 = *Extremely*). Therefore, the maximum score on each subscale is 50, with higher scores reflecting greater positive or negative affect. The internal consistency in this study ranged from acceptable to excellent, Cronbach's $\alpha = 0.75–0.89$. When completing he state anxiety subscale of the STAI, participants must rate six items related to how they feel on a four–point Likert–type scale (1 = Not al all; 2 = Somewhat; 3 = Moderately; 4 = Very much). Therefore, the maximum score is 24, where higher scores reflect greater levels of state anxiety. The internal consistency in this study ranged from acceptable to excellent, Cronbach's $\alpha = 0.76–0.85$.

2.2.6. Alcohol use behaviour

Alcohol Use Disorders Identification Test (AUDIT)

Participants completed the AUDIT (Babor et al., 1992, 2001) to assess hazardous drinking. The AUDIT has excellent psychometric properties when used to assess AUDs (Claussen & Aasland, 1993; Fleming et al., 1991). The AUDIT is a ten-item scale, scored on a scale from 0–40, where scores between 0–7 indicate low-risk drinking, scores between 8–15 indicate increasing risk of harm, scores between 16–

19 higher risk drinking, and a scores > 20 suggest alcohol dependence. Internal consistency of the AUDIT in this study was acceptable, Cronbach's $\alpha = 0.70$.

Desires for Alcohol Questionnaire (DAQ)

Craving for alcohol was measured using the 14-item version of the Desires for Alcohol Questionnaire (Kramer et al., 2010). When completing the DAQ participants rate a series of statements on a five–point Likert–type scale (1 = *Not at all* : 5 = *Strongly agree*) which are related to their desire to consume alcohol at the point in time when the scale is administered. Therefore, the maximum score on the DAQ is 70. The scores attained from the DAQ provide a single measure of craving for each participant, where greater scores specify a greater desire for alcohol consumption. Internal consistency of the DAQ in this study was acceptable, Cronbach's $\alpha = 0.76–0.81$.

Voluntary Drinking Task

Alcohol consumption was assessed using a voluntary drinking task based on the tasks reported in Higgs et al. (2008) and Stafford & Dodd (2013). Participants were offered a single alcoholic beverage consisting of 50mL of Smirnoff Vodka (Smirnoff, USA)[8] diluted in 250mL of a mixer of their choice (either coke, lemonade or tonic). Prior to consuming the alcoholic beverage, participants were asked to drink a small shot glass (40mL) of chilled water, described as a "thirst quencher". The participants were then instructed to *"consume all of the drink at a rate that feels comfortable"* and they were also told that "*there is no advantage in drinking the drink as quickly as possible as the study session will last the same amount of time regardless"*. While the alcoholic beverage was being consumed, participants watched a DVD program (Blue Planet, Series 1, Episode 1) and were filmed using a JVC GZ–R15 Camcorder (JVC, Japan). Participants were required to watch the DVD program to distract them from the study aims and to provide us with a relatively ecologically valid measurement of alcohol use (vs. a progressive ratio schedule, for example). The video recordings were coded and converted to total drinking time, total "sipping" duration, total interval between sips, number of sips, mean sip duration, and mean inter–sip–interval by two independent coders using BORIS for Windows (version 8.13) (Friard & Gamba, 2016). Inter–coder reliability was established by calculating the intraclass correlation

[8] Smirnoff Vodka is 37.5% ABV. Thus, 50mL is equivalent to 1.875 UK alcohol units or 15g of ethanol.

coefficient (ICC) between the two coders, using data from nine randomly selected subjects[9] (average ICC = 0.92, 95% CI = 0.66–0.98). Once the alcoholic beverage was consumed, participants continued to watch the DVD program for a further five minutes before being breathalysed (AlcosSense Pro, AlcoSense, UK). They were then then asked to complete the Brief Biphasic Alcohol Effects Scale (B–BAES; Rueger & King, 2013) and a series of visual analogue scales (VAS).

The B–BAES was used to assess whether any stimulant or sedative effects had taken place due to the consumption of alcohol. Each subscale of the B–BAES contains three items related to how the participant feels at the time of administration which are rated on an 11–point Likert–type scale (1 = Not at all : 11 = Extremely). Therefore, the maximum score on each subscale is 33, with higher scores reflecting greater level of stimulation or sedation. The internal consistency of the B–BAES subscales in this study was excellent, Cronbach's $\alpha = 0.89–0.92$. Participants used the VAS to rate the sensory properties of the drink and their attitudes towards the drink. Each VAS was anchored with "low" or "not at all", followed by the relevant adjective, and "high" or "very", followed by the relevant adjective. The descriptors used were presented in the following order: "alcohol strength", "like", "bitter", "cold", "dislike", "sweet", "similarity to drinks normally consumed", "fizzy", and "similar to drinks consumed on a night out". Comparable descriptors have been used in previous research (e.g., Higgs et al., 2008; Stafford & Dodd, 2013).

2.2.7. Procedure

Phase 1: Baseline assessments

After informed consent was obtained, the participant's hight and weight was measured. Height and weight data were used to calculate body mass index (BMI), where:

$$\text{BMI} = \frac{\text{weight (kg)}}{\text{height}^2\ \text{(m)}} \qquad (2)$$

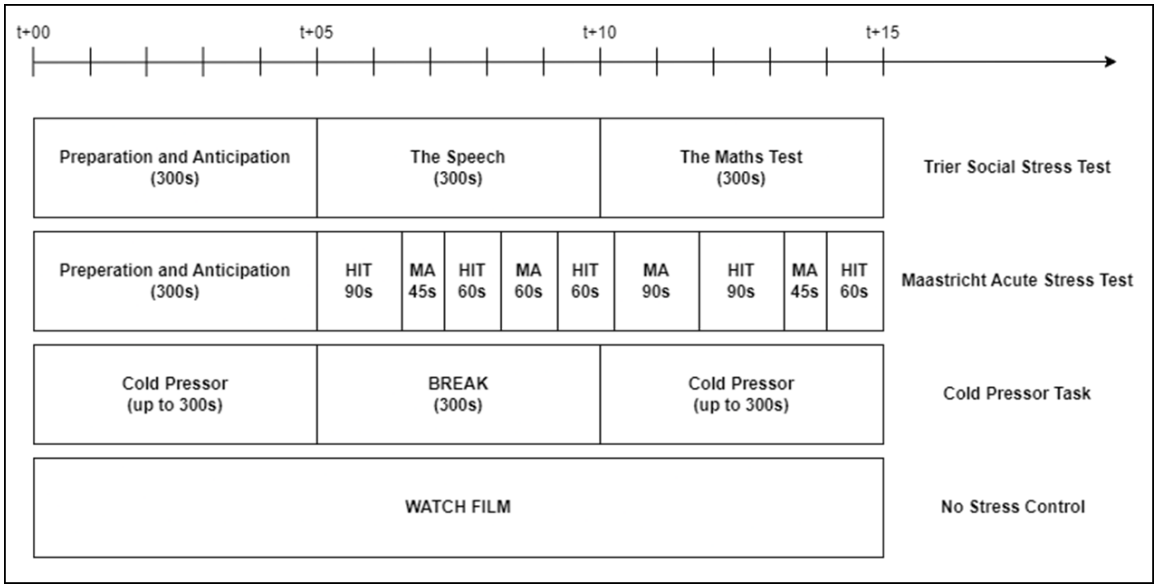
[9] A power analysis using the methods found in Zou (2012) indicated that data from eight participants was required to reliability detect an ICC as low as 0.798 with power $(1 - \beta) = 80\%$ and $\alpha = 0.05$. Therefore, nine participants were randomly selected to err on the side of caution.

The heart rate monitor (Polar A300 and H7, Polar, Finland) was fitted, and the recording was started. Participants were then asked whether they had consumed alcohol in the last 12 hours (confirmed using an AlcoSense Pro breathalyser, AlcoSense, UK), nicotine in the last hour, or caffeine in the last hour. The first blood pressure measurement was then taken using the Omron M2 Upper Arm Monitor (Omron, The Netherlands). Next, participants then completed the PANAS, the state subscale of the STAI, the DAQ, and the S–UPPSP in counter balanced order using Qualtrics (Provo, Utah). Subsequently, participants completed the three computer tasks (TADD, BART, SST) in counter balanced order. Computer tasks were programmed and administered using PsychoPy (Peirce et al., 2019, 2022). Finally, participants completed two "taste tests" (see Appendix A). The taste test data was collected for a separate project which falls outside of the scope of this thesis, thus it will be reported elsewhere.

Phase 2: Stress manipulation

Participants were randomly allocated to take part in either the TSST (Kirschbaum et al., 1993; Kudielka et al., 2007), the MAST (Shilton et al., 2017; Smeets et al., 2012), a CPT (Mitchell et al., 2004), or a no stress control condition. Each condition lasted approximately 15 minutes (see Figure 2.1). Cold pressor equipment was required for the MAST and CPT conditions. Here, a circulating and cooling water bath, Grant Instruments model TC120 (Cambridge, UK), was used as the cold pressor stimulus. This model of water bath has a 38L water capacity and maintains temperature within the ± 0.05°C range.

Figure 2.1 A schematic of the procedures used in Phase 2 of the study. HIT = Hand Immersion Task; MA = Maths Assessment.



Those allocated to the TSST condition were told *"you have 5-minutes to prepare a five-minute speech about your dream job and what makes you the ideal candidate. You will deliver this speech to a group of strangers without any notes".* Once the five–minute preparation and anticipation stage was complete, the participant was led to a room containing two or three people wearing lab coats who were sat behind a desk, and a video camera (JVC GZ–R15 Camcorder, JVC, Japan) aimed at the position where the participant would deliver their speech. If a participant stopped speaking for more than ten–seconds during their speech, they were encouraged to continue and were reminded how much time was remaining. Once the speech was complete, participants were asked to complete a mathematics test by counting backwards in 17s for five minutes, beginning at 2,043. If the participant made an error during the mathematics test, they were asked to start again at 2,043.

The MAST consists of a five–minute preparation and anticipation stage and a ten-minute stress stage. During the first stage, participants are shown the task instructions on a computer screen. The instructions told the participants that *"…there will be trials where you have to immerse your hand in ice cold water. The duration of these trials will be randomly chosen by the computer but will never exceed 90s. In between hand immersion trials, you will place your arm on a towel and engage in a mental athematic test which consists of counting backwards from 2043 in 17s until the computer signals you to begin the next hand immersion trial"*. They were also told that *"the procedure could be very uncomfortable, and you can remove their hand from the ice-cold water at their own discretion without consequence".* During the hand immersion trials (HIT), participants placed their hand, up to and including the wrist, in ice cold water (3 °C). During the mathematic assessment (MA), participants continued counting while resting their arm on a towel beside the water bath. If a mistake was made during the MA, or if the participant did not give a response within five-seconds, they were asked to start again at 2,043. In reality, the duration of each trial was pre-determined, and the same protocol was used for each participant (see Figure 2.1). Therefore, there were five HITs alternated with four MAs with the following order and duration: HIT (90s), MA (45s), HIT (60s), MA (60s), HIT (60s), MA (90s), HIT (90s), MA (45s), HIT (60s).

Those in CPT condition were instructed to immerse their hand up to and including their wrist in ice cold (3 °C) water for as long as possible, with a maximum duration of 5-minutes per trial. They were told that *"the procedure could be very uncomfortable, and you can remove your hand from the ice-cold water at your own discretion without consequence".* After the first trial, there was a five–minute break followed by a second hand immersion. Between trials, participants rested their arm on a towel beside the water bath.

Participants in the control condition were asked to watch the same DVD program (Blue Planet, Series 1, Episode 1) used in the voluntary drinking task for the 15–minute duration of Phase 2.

Phase 3: Post–manipulation

Following completion of one of the assigned conditions, a second blood pressure measure was taken before participants completed a second PANAS, DAQ and the state subscale of the STAI in counterbalanced order using Qualtrics (Provo, Utah). Subsequently, participants completed the voluntary drinking task before being partially debriefed. We chose a partial debrief method due to the sensitive nature of our variables. For instance, if participants were to notify their peers about the study aims, subsequent participants may alter their responses, thus rendering the study invalid. A full debrief was emailed to participants at the end of the data collection period. Participants were breathalysed a final time at the end of the study using an AlcoSense Pro (AlcoSense, UK). In concordance with the National Institute on Alcohol Abuse and Alcoholism (NIAAA) Guidance for Conducting Alcohol Administration Studies with Human Participants (NIAAA, 2023), participants with a BAC ≈ 0.04% were advised to rest in the waiting area until their BAC level decreased. Once a participant's BAC had dropped to this level, they were released with a cautionary reminder to avoid activities that entail risk while under the influence of alcohol. Participants who insisted on leaving immediately after debriefing were reminded that their current their estimated BAC may be in excess of the legal limit for driving (i.e., BAC > 0.08%).

2.2.8. Analysis

Both univariate and multivariate outliers were screened prior to analysis following Tabachnick and Fidell (2014). Univariate outliers were assessed using z-scores, where a z-score >3.29 and < -3.29 ($p <$

.001, two-tailed test) was considered a univariate outlier. Univariate outliers were assigned a score that was one unit larger (or smaller) than the next most extreme score in the distribution to reduce their influence while conserving statistical power (Tabachnick & Fidell, 2014). The assessment of multivariate outliers was based on a Mahalanobis distance that is significant at the $p < .001$ level, assuming that the test statistic follows a chi-square distribution (Verardi & Dehon, 2010). Overall, 0.38% of data were replaced as they were univariate outliers. No multivariate outliers were detected. No missing data were present in the analysed data. Reactivity and recovery variables were calculated for stress response and craving data (Clay & Parker, 2018). For variables measured at multiple timepoints (i.e., heart rate and heart rate variability), reactivity = T2 (recorded during the task) – T1 (recorded at baseline) and recovery = T3 (recorded immediately after the task) - T2. For variables measured at two timepoints (i.e., blood pressure, affect, state anxiety and craving), reactivity = T3 - T2. Results were considered significant when $p < 0.05$ or when the 95% CI did not contain zero.

2.3. Results

2.3.1. Sample characteristics

Table 2.1 presents the sociodemographic characteristics of the sample and descriptive statistics (means and standard deviations) for selected study variables in terms of impulsivity, alcohol use behaviour, and the sensory properties of the drink consumed during the voluntary drinking task. A series of Kruskal-Wallis (for continuous variables) and chi-square (for categorical data) tests were used to test for any between–group differences. No statistically significant differences were identified (all *p*s > .05). Descriptive data for stress and craving by timepoint and group is presented in Appendix A.

2.3.2. Bivariate analysis

The intercorrelations between measures of impulsivity, the stress response – broken down into cardiac function (HR, HRV), blood pressure (SBP, DBP, MAP), and psychological (positive and negative affect, and state anxiety) assessments – and drinking behaviour were examined to help determine the reliability of our measures. Here, Spearman's rank correlations were calculated for each subset of variables previously listed.

Table 2.1 Sociodemographic characteristics of the sample and descriptive statistics (*M* and *SD*) for selected variables.

Variable	TSST (*n* = 22)	MAST (*n* = 31)	CPT (*n* = 24)	Control (*n* = 30)
Demographics				
Age	26.14 (7.46)	22.87 (5.20)	21.79 (4.09)	23.37 (4.81)
Sex=Female	11 (50.00%)	22 (70.97%)	13 (54.17%)	19 (63.33%)
Height (m)	168.03 (7.48)	166.35 (8.27)	165.78 (8.86)	167.30 (6.58)
Weight (kg)	69.05 (12.58)	69.01 (13.84)	71.19 (14.31)	69.24 (12.91)
BMI (kg/m^2)	24.63 (5.38)	24.92 (4.39)	25.87 (4.68)	24.64 (3.75)
Smoke = Yes	1 (4.55%)	7 (22.58%)	2 (8.33%)	3 (10.00%)
Vape = Yes	1 (4.55%)	4 (12.90%)	0 (0.00%)	3 (10.00%)
PHQ-4	1.23 (1.19)	1.10 (1.33)	1.58 (1.64)	0.70 (1.12)
Impulsivity				
SUPPS-P Negative Urgency	7.95 (2.65)	8.35 (2.96)	9.67 (2.87)	7.57 (2.39)
SUPPS-P Premeditation	6.36 (1.84)	6.26 (1.93)	7.00 (1.74)	6.23 (1.76)
SUPPS-P Perseverance	7.23 (1.90)	6.61 (1.71)	7.13 (2.09)	6.57 (1.63)
SUPPS-P Sensation Seeking	11.13 (3.03)	11.19 (2.65)	11.29 (2.62)	10.73 (2.42)
SUPPS-P Positive Urgency	7.23 (2.60)	6.87 (1.67)	7.96 (2.77)	6.43 (2.65)
BART	34.69 (12.22)	31.50 (11.46)	32.76 (9.41)	33.14 (13.08)
1 - AUC	0.75 (0.18)	0.77 (0.12)	0.81 (0.11)	0.69 (0.30)
Alcohol use behaviour				
AUDIT	5.86 (2.55)	6.94 (3.88)	7.08 (3.23)	5.90 (2.62)
Total drinking time (sec)	596.35 (308.14)	769.21 (441.01)	770.38 (413.98)	576.25 (264.32)
Total sipping duration (sec)	40.48 (26.12)	48.76 (46.92)	42.33 (22.10)	36.10 (22.72)
Total interval between sips (sec)	555.87 (298.50)	720.46 (425.75)	662.79 (369.52)	541.63 (270.41)
Number of sips	16.04 (9.26)	16.61 (8.91)	17.75 (9.08)	13.57 (5.24)
Mean sip duration (sec)	2.63 (1.50)	2.72 (1.08)	2.47 (0.77)	2.81 (1.79)
Mean inter-sip-interval (sec)	39.78 (18.30)	55.10 (38.00)	50.24 (34.78)	48.87 (27.58)

B-BAES Stimulated	14.36 (7.31)	16.10 (5.71)	14.38 (6.39)	14.97 (5.58)
B-BAES Sedated	11.36 (7.96)	10.48 (6.22)	10.83 (5.67)	11.80 (5.99)
Sensory VAS				
Like	53.32 (29.20)	59.39 (23.78)	54.79 (23.00)	59.00 (23.96)
Bitter	32.36 (28.97)	19.81 (20.60)	27.38 (26.80)	21.33 (23.21)
Cold	47.55 (34.06)	44.68 (30.57)	43.38 (28.85)	49.03 (22.14)
Dislike	26.95 (31.15)	18.29 (22.71)	29.50 (27.85)	23.37 (24.01)
Sweet	54.50 (28.29)	54.97 (25.89)	45.83 (26.69)	54.40 (23.08)
Normal	39.95 (39.21)	62.23 (32.74)	56.88 (32.19)	50.33 (28.26)
Fizzy	72.59 (21.40)	70.32 (25.33)	66.21 (26.19)	68.60 (17.55)
Night out	62.45 (39.57)	72.58 (30.73)	67.25 (36.51)	68.47 (28.17)
Strength	55.68 (22.48)	42.39 (22.48)	52.08 (19.43)	47.13 (18.52)

Note. TSST = Trier Social Stress Test; MAST = Maastricht Acute Stress Test; CPT = Cold Pressor Task; BMI = Body Mass Index; PHQ–4 = Patient Health Questionnaire for Depression and Anxiety; SUPPS-P = The Shortened Urgency, Premeditation (lack of), Perseverance (lack of), Sensation Seeking, Positive Urgency, Impulsive Behaviour Scale; BART = average number of space bar presses for unburst balloons during the Balloon Analogue Risk Task; 1 – AUC = 1 minus the area under the curve score (so that greater scores reflect greater delay discounting) for the Titrating Alternatives Delay Discounting Task; AUDIT = Alcohol Use Disorder Identification Test; B–BAES = Brief Biphasic Alcohol Effects Scale; VAS = Visual Analogue Scale.

As shown in Table 2.2, several measures of impulsivity were intercorrelated. Negative urgency was positively correlated with premeditation positive urgency; premeditation was positively correlated with perseverance and positive urgency; and sensation seeking was positively correlated with positive urgency. No other relationships between impulsivity variables were significant (all *p*s > .05).

Table 2.2 Inter-correlations (Spearman's rank values) between impulsivity measures.

	1	2	3	4	5	6
1. SUPPS-P Negative Urgency	-					
2. SUPPS-P Premeditation	.39***	-				
3. SUPPS-P Perseverance	.16	.40***	-			
4. SUPPS-P Sensation Seeking	-.07	-.05	-.07	-		
5. SUPPS-P Positive Urgency	.51***	.20*	.08	.30**	-	
6. BART	-.02	.08	0.07	.16	-.00	-
7. 1 - AUC	.06	.09	-.03	.03	.03	.03

Note. SUPPS-P = The Shortened Urgency, Premeditation (lack of), Perseverance (lack of), Sensation Seeking, Positive Urgency, Impulsive Behaviour Scale; BART = average number of space bar presses for unburst balloons during the Balloon Analogue Risk Task; 1 – AUC = 1 minus the area under the curve score (so that greater scores reflect greater delay discounting) for the Titrating Alternatives Delay Discounting Task.

* $p < .05$, ** $p < .01$, *** $p < .001$

In terms of stress, all measures of blood pressure were intercorrelated (see Table 2.3) and all measures of cardiac function were intercorrelated, (see Table 2.4). However, not all measures of psychological stress were correlated (see Table 2.5). Here, positive affect at T1 was positively correlated with positive affect at T3 and negatively correlated with state anxiety at T3; positive affect at T3 was negatively correlated with state anxiety at both T1 and T2; and all measures of negative affect and state anxiety were positively intercorrelated, apart from state anxiety at T1 and negative affect at T3. Taken together, these results suggest that including multiple measures of blood pressure, cardiac function, and negative affect in subsequent analyses is redundant.

Correlations between drinking behaviour measures are shown in Table 2.6. All measures were intercorrelated except mean sip duration, suggesting that using multiple measures of alcohol use in following analyses would be unnecessary.

Table 2.3 Inter-correlations (Spearman's rank values) between measures of blood pressure.

	1	2	3	4	5
1. SBP–T1	-				
2. SBP–T3	.65***	-			
3. DBP–T1	.51***	.43***	-		
4. DBP–T3	.38***	.68***	.52***	-	
5. MAP–T1	.83***	.60***	.89***	.52***	-
6. MAP–T3	.55***	.88***	.52***	.94***	.61***

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; T1 = baseline measurement; T3 = measurement taken immediately after manipulation.

* $p < .05$, ** $p < .01$, *** $p < .001$

Table 2.4 Inter-correlations (Spearman's rank values) between measures of cardiac function.

	1	2	3	4	5	6	7	8
1. HR–T1	-							
2. HR–T2	.68***	-						
3. HR–T3	.83***	.80***	-					
4. SDNN–T1	-.59***	-.48***	-.62***	-				
5. SDNN–T2	-.57***	-.46***	-.54***	.67***	-			
6. SDNN–T3	-.58***	-.51***	-.63***	.79***	.75***	-		
7. RMSSD–T1	-.58***	-.46***	-.60***	.89***	.67***	.73***	-	
8. RMSSD–T2	-.46***	-.70***	-.59***	.66***	.75***	.79***	.74***	-
9. RMSSD–T3	-.52***	-.51***	-.63***	.72***	.71***	.88***	.82***	.89***

Note. HR = Heart Rate (BPM) = heart rate; SDNN (ms) = standard deviation of normal–to–normal intervals; RMSSD (ms) = root mean square of successive differences between normal heartbeats; T1 = baseline measurement; T2 = measurement taken during the manipulation; T3 = measurement taken immediately after manipulation.

* $p < .05$, ** $p < .01$, *** $p < .001$

Table 2.5 Inter-correlations (Spearman's rank values) between measures of psychological stress.

	1	2	2	4	5
1. Positive Affect (PANAS)–T1	-				
2. Positive Affect (PANAS)–T3	.76***	-			
3. Negative Affect (PANAS)–T1	.10	-.02	-		
4. Negative Affect (PANAS)–T3	.16	.08	.32***	-	
5. State Anxiety (STAI)–T1	-.35***	-.27**	.51***	.17	-
6. State Anxiety (STAI)–T3	-.11	-.24*	.21*	.66***	.41***

Note. PANAS = Positive Negative Affect Schedule; STAI = State–Trait Anxiety Inventory; T1 = baseline measurement; T3 = measurement taken immediately after manipulation.

* $p < .05$, ** $p < .01$, *** $p < .001$

Table 2.6 Inter-correlations (Spearman's rank values) between drinking behaviour measures.

	1	2	3	4	5
Total drinking time (sec)	-				
Total sipping duration (sec)	.27**	-			
Total interval between sips (sec)	.95***	.17	-		
Number of sips	.36***	.73***	.28**	-	
Mean sip duration (sec)	.02	.53***	-.07	-.10	-
Mean inter-sip-interval (sec)	.56***	-.40***	.65***	-.47***	.00

* $p < .05$, ** $p < .01$, *** $p < .001$

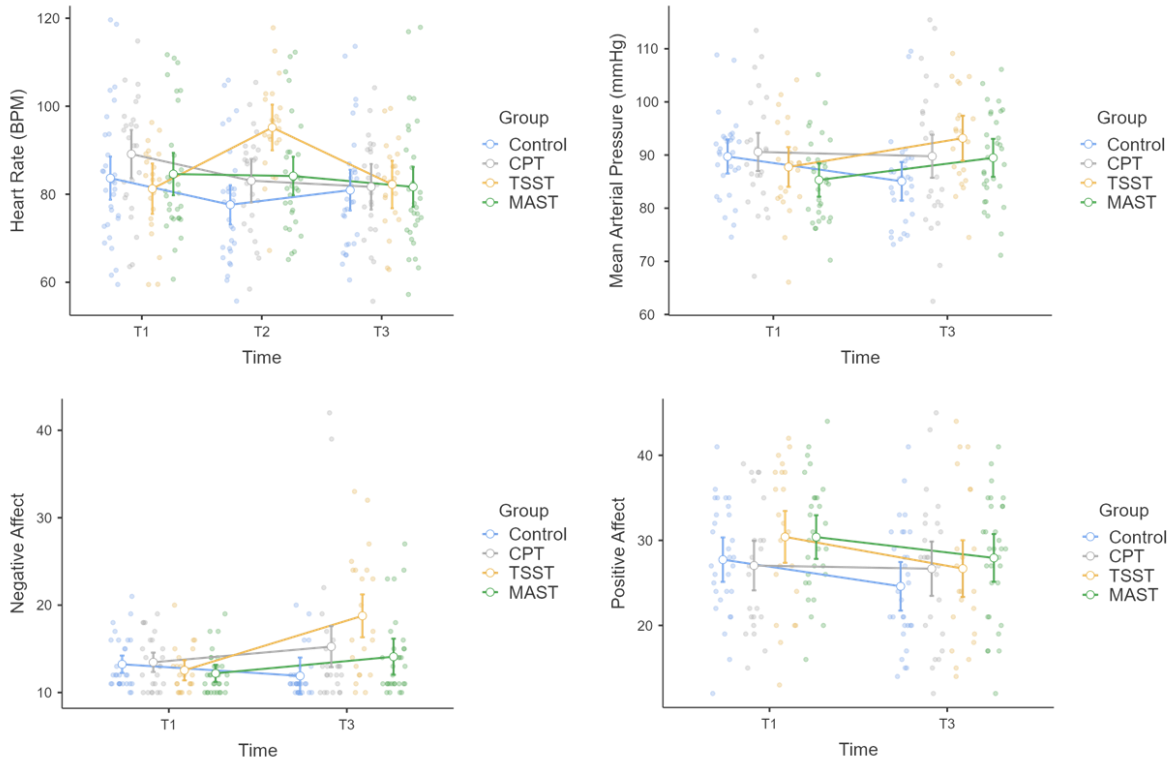
2.3.3. Manipulation check

Descriptive statistics (means and standard deviations) for repeated measures variables (stress and craving) are presented in Table A2. Several time x group mixed ANOVAs were used to check our manipulations worked. As shown in Figure 2.2, there were statistically significant interactions for HR (F [6,206] = 19.6, $p < .001$, $\eta p^2 = 0.36$), MAP (F [3, 103] = 13.15, $p < .001$, $\eta p^2 = 0.28$), and negative affect (F [3,103] = 8.08, $p < .001$, $\eta p^2 = 0.19$), but not for positive affect (F [3, 103] = 1.89, $p = 0.137$, $\eta p^2 = .137$). However, the main effect of time for positive affect was significant, F (1, 103) = 22.69, $p < .001$, $\eta p^2 = 0.18$).

Significant interactions were probed using planned Bonferroni-corrected pairwise comparisons. In the Control group HR (M = 83.66, SD = 15.27 vs. M = 77.62, SD = 13.18, t (103) = 4.01, p = .008, d = 0.78 [95% CI 0.37-1.19]) and MAP (M = 89.73, SD = 7.76 vs. M = 85.06, SD = 8.59, t (103) = 3.81, p = .007, d = 0.98 [95% CI 0.53-1.41]) decreased. In the CPT group, HR (M = 89.14, SD = 13.34 vs. M = 83.07, SD = 11.67, t (103) = 4.49, p = .032, d = 0.81 [95% CI 0.34-1.27]) decreased from T1 to T2. In the TSST group, HR increased from T1 (M = 81.26, SD = 10.45) to T2 (M = 95.17, SD =10.89), t (103) = 7.92, $p < .001$, d = 1.22 (95% CI 0.65-1.76), and decreased from T2 to T3 (M = 82.22, SD = 9.74), t (103) = 9.11, $p < .001$, d = 1.47 (95% CI 0.86-2.07). Meanwhile, MAP (M = 87.75, SD = 9.01 vs. M = 93.17, SD = 8.74, t (103) = 3.78, p = .007, d = 0.79 [95% CI 0.30-1.27]) and negative affect (M = 12.55, SD = 2.58 vs. M = 14.09, SD = 7.05, t (103) = 5.34, $p < .001$, d = 1.00 [95% CI 0.48-1.51]) increased. Finally, in the MAST group, MAP increased (M = 85.31, SD =7.92 vs. M = 89.48, SD = 8.63, t (103) = 4.17, p = .022, d = 0.60 [95% CI 0.21-0.97]). Taken together, these results suggest that the

manipulation of stress worked in the TSST and MAST groups. Though, the TSST stress response was more robust than the MAST.

Figure 2.2 Heart rate, mean arterial pressure, negative affect, and positive affect in the Control (*n* = 30), Cold Pressor Task (CPT; *n* = 24), Trier Social Stress Test (TSST; *n* = 22), and Maastricht Acute Stress Task (MAST, *n* = 31) conditions at baseline (T1), during the tasks (T2), and immediately after each task (T3).



2.3.4. Main analysis

Changes in craving and drinking

One-way ANOVAs were used to test whether a change in craving (DAQ–T3 - DAQ–T1) and drinking (total drinking time) varied across groups. Neither were statistically significant (Fs < 2, ps > .155).

The combined effects of impulsivity on craving and drinking

Group–wise correlations (Spearman's rank values) were calculated between measures of impulsivity, stress, craving, and drinking to test the hypothesis that higher levels of impulsivity would be associated with increased levels of stress–induced alcohol craving and use (i.e., total drinking time). These data are presented in Table A3 and Table A4. Negative urgency (r_s = .46, p = .030) and increased negative affect (r_s = .54, p = .009) were positively associated with increased levels of stress–induced craving in

the TSST group. Surprisingly, no other significant relationships were found, and craving change was not significantly associated with drinking in any group (all $p_s > .05$). In terms of drinking, poor perseverance was positively associated with total drinking time ($r_s = .43$, $p = .048$) in the TSST group; higher levels of positive affect were negatively associated with total drinking time in the MAST group ($r_s = -.46$, $p = .009$); and in the CPT group, increased levels of positive affect ($r_s = .48$, $p = .017$) and higher levels of negative urgency ($r_s = -.43$, $p = .034$) were significantly associated with total drinking time.

2.4. Discussion

The present study aimed to assess the impact of impulse control on craving and consumption of alcohol following either acute pain (CPT), psychosocial stress (TSST), or a mixed stressor (MAST). We tested the hypothesis that heightened stress would increase craving and reduce total drinking time. We also tested the hypothesis that impulsivity would moderate the effect of stress on craving and drinking, whereby increased levels of impulsivity would be associated with increased stress–induced craving and faster stress–induced alcohol consumption.

Statistically significant between–group changes in craving or drinking were not detected; thus, the findings here were at odds with the majority of the existing literature (e.g., Clay et al., 2018; Clay & Parker, 2018; De Wit et al., 2003; Field & Powell, 2007; Magrys & Olmstead, 2015; McGrath et al., 2016; Owens et al., 2015). Instead, therefore, these findings were likely driven by insufficient stress responses following the CPT and MAST.

Previous research indicates that cold pressor pain reliably activates the HPA system (e.g., Bullinger et al., 1984; Edelson & Robertson, 1986; Velasco et al., 1997). Though, this response is typically less than that elicited by the TSST (McRae et al., 2006) or MAST (Smeets et al., 2012)[10]. Therefore, finding that the CPT did not elicit a stress response was surprising. One explanation may be the length of our CPT protocol. For example, Mitchell et al (2004) investigated how pain tolerance and

[10] There were some notable differences between the CPT used in the present study and that used in Smeets et al. (2012): (1) the temperature of the water used in Smeets et al. (2012) was 2°C and (2) the maximum duration per hand immersion was 3 min. As explained in the main text, variations in water temperature and hand immersion duration may affect HPA responses.

intensity change as a function of water temperature (1°C, 3°C, 5°C, and 7°C) during the CPT. They found that 3°C water was tolerated for approximately 55s on average. Therefore, as our CPT protocol was broken into three 5-minute blocks (hand immersion, break, hand immersion), it may be that stress responses stabilised before the post–stress measures were taken or that the pain stimulus was no longer stressful upon the second immersion. Alternatively, for both the CPT and MAST, our sample may have been particularly resistant to stress. For instance, higher HRV is associated with stress resilience (An et al., 2020; Chen et al., 2023) and participants in the CPT and MAST groups had higher than normal HRV. Put somewhat differently, the SDNN is an overall index of HRV which reflects both sympathetic and parasympathetic influences on HR (Shaffer et al., 2014), and typical normative SDNN values from short–term recordings (~ 5 min) at rest are 50ms ± 16 (Shaffer & Ginsberg, 2017). However, the baseline SDNN values here in the CPT and MAST groups were 84.13ms ± 43.09 and 88.51 ± 37.35, respectively. Despite this, our stress response results in the MAST group are mostly in line with previous work. For instance, data from Shilton et al. (2017) suggest that the MAST effects blood pressure and state anxiety but not HR. Therefore, the most likely explanation is that the CPT and MAST manipulations were not sufficiently intense enough.

In contrast, the TSST caused robust changes in HR (d = 1.22), MAP (d = 0.79), and negative affect (d = 1.00) and our predictions were partially supported by data from this group. For instance, among those assigned to this group, both negative affect (rs = .54) and negative urgency (i.e., the tendency to act rashly under extreme negative emotions) (r_s =.46) were associated with increased levels of craving. These findings are in line with previous work which suggests that acute psychosocial stress causes increased alcohol craving among non–dependent individuals and that this increase in craving is strengthened by impulsivity (Clay et al., 2018). However, a key difference between the findings reported here and those described in Clay et al. (2018) was the type of impulsivity associated with increased craving. Specifically, in Clay et al. (2018), we found that BART score was positively correlated with craving, whereas here it was negative urgency[11]. One explanation may be subtle differences in the BART tasks administered in each study. For instance, the BART used in Clay et al.

[11] Negative urgency was not measured in Clay et al. (2018).

(2018) had only 20 trials (i.e., balloons) whereas, here we had 30. The more likely reason is that the sample size was too low in this study. For example, there was a non–significant positive association between BART scores and craving in the TSST group in this study of r_s = .29 but with the 22 participants in the TSST group, we were only able to reliably detect a correlation as low as r_s = .56. Taken together, these results and findings from previous research suggest that negative affect, negative urgency and possibly risk–taking (i.e., BART score) are important in understanding the link between acute stress and craving.

Data from previous empirical work supports these ideas. For instance, Bresin et al. (2018) conducted a systematic review and meta–analysis of the literature investigating craving and consumption of alcohol following laboratory–based manipulations of negative affect. Across the 41 studies analysed (N = 2,403), they identified that increased negative affect (vs. control) was positively associated with increased craving (d = .39) and alcohol use (d = .31). Importantly, they did not find evidence to suggest that negative affect was altered by AUD status. Thus, suggesting that the impact of negative affect on craving and consumption of alcohol among alcohol dependent individuals is predicated on other risk–factors, such as poor impulse control (Bresin et al., 2018).

Similarly, negative urgency has been identified in meta–analyses as the most important UPPS–P trait for alcohol–related problems and dependence (Coskunpinar et al., 2013; Stautz & Cooper, 2013). Relevant to the discussion about negative affect, Simons et al. (2010) investigated within–person associations between positive affect, anxiety, sadness, hostility, frequency of alcohol intoxication, and alcohol dependence symptoms in a sample of 102 university students. The authors found that anxiety and sadness (i.e., negative affect) were both linked to dependence symptoms. Importantly, they also found that the association between anxiety and intoxication was only significant among those high in negative urgency or low in positive urgency (Simons et al., 2010). Correspondingly, in a sample of 675 community–dwelling adults, Um et al. (2019) found that negative, but not positive, urgency was a unique mediator between depressive symptoms and problematic alcohol use. Thus, negative urgency seemingly moderates the relationship between negative affect and alcohol use.

With regards to negative urgency and craving specifically, VanderVeen et al. (2016) investigated negative urgency, mood induction, and alcohol seeking behaviours among a sample of 34 community–dwelling individuals. Specifically, participants were tested in two counterbalanced intravenous alcohol self–administration conditions: one with negative mood induction and one without. They found that greater negative urgency predicted increased negative affect and craving for alcohol in the condition where negative mood was manipulated. Therefore, demonstrating that negative urgency moderates alcohol self–administration via increased craving and negative affect (VanderVeen et al., 2016).

When considering risk–taking, several studies have shown that BART scores are positively associated with alcohol use and related outcomes (see Canning et al., 2021 for review). For example, several studies have found a positive relationship between BART and craving for alcohol (Clay et al., 2018; Heinz et al., 2016; Rose et al., 2014). However, Clay and Parker (2018) and Padovano et al. (2019) did not detect a significant association between BART and craving. Nevertheless, both of these studies had relatively small sample sizes ($N < 40$), which could hinder the ability to detect significant effects. Taken together, it is clear that BART score is predictive of alcohol–related outcomes. However, the link between BART scores and craving requires more evidence to be confirmed.

Craving was not significantly associated with drinking in this study which was surprising. However, similar results were also found in Clay and Parker (2018). Though craving is a hallmark feature of addiction (e.g., Addolorato et al., 2005), the samples analysed in both studies were non–dependent as reflected by the relatively low AUDIT scores: 6.46 ± 3.16 in this study, and 9.17 ± 5.65 in Clay and Parker 2018. Therefore, craving may only be an important predictor of alcohol use behaviour among those who drink excessively. This is corroborated by Grüsser et al. (2006) who compared craving and several craving–related variables between two groups of occasional drinkers ($n = 50$) and problem drinkers ($n = 50$), finding that craving was unimportant in understanding drinking behaviour among the group of occasional drinkers (e.g., occasional drinkers reported little alcohol craving). Despite this, craving is a key component of the addiction cycle (e.g., Koob & Le Moal, 1997). Thus, those who are more likely to experience craving under acute stress, such as individuals who are

high in negative urgency or prone to experience greater levels of negative affect, may be more likely to transition from social drinking to alcohol dependence. Moreover, as increased negative urgency was also associated with a quicker time–to–consume an alcoholic beverage in the CPT group (r_s = -.43) in this study, screening individuals for traits such as negative urgency and then offering treatment to reduce the influence of such traits may prove beneficial in the future.

Significant relationships between positive affect and drinking (i.e., total drinking time) were observed in the MAST (which did invoke a stress response) and CPT groups (which did not cause a stress response). In the MAST group, positive urgency was associated with quicker drinking durations (*r*s = -.46). However, the opposite was observed in the CPT group (*r*s = .48). Previous literature suggests that among university students, the relationship between positive affect, stemming from social stressors, and alcohol use is moderated by deficits in the regulation of positive emotions (Weiss et al., 2019). However, among alcohol dependent individuals, positive affect seems to aid in stress resilience, and therefore is associated with lower levels of stress–related alcohol consumption, in some studies (McHugh et al., 2013). While in others positive affect was found to be a positive predictor of alcohol use among dependent individuals (Bresin & Fairbairn, 2019). Therefore, further research is required to fully understand the role of positive affect in the development and maintenance of alcohol dependence.

Finally, our results suggest that increased levels of lack of perseverance is associated with taking an increased amount of time to consume an alcoholic beverage following an acute psychosocial stressor (i.e., the TSST). This is a novel and surprising finding which is at odds with much of the literature. For instance, two separate meta–analyses found that all UPPS–P traits are positively associated with alcohol use (Coskunpinar et al., 2013; Stautz & Cooper, 2013). Therefore, it is likely that this finding is a statistical artefact or due to our methodological choices. For instance, the *p*–value associated with this result was relatively large (.048) which therefore may suggest that this finding is a type I error. Alternatively, we chose to use the time to consume a single alcoholic beverage as our primary dependent variable, whereas others have used a progressive ratio schedule (e.g., Clay & Parker, 2018) or an *ad libitum* alcohol consumption task (e.g., Field et al., 2007). Therefore, choosing another drinking task may have altered our results.

2.4.1. Limitations

There were several limitations to the present study that must be considered. First, the less than optimal sample size, which resulted from the data collection period being interrupted by the COVID–19 pandemic, meant we were only able to reliably detect large associations (r = .56), whereas our previous work has found medium effect sizes (Clay et al., 2018; Clay & Parker, 2018). Nevertheless, we did detect predicted effects smaller than r = .56 (e.g., the relationship between negative urgency and craving in the TSST group, r = .46). Second, the generalisability of our findings is limited to our sample which was predominantly made up of relatively young female participants. Third, the CPT and MAST did not invoke the expected stress response, further limiting our sample size for hypothesis testing. Therefore, future research should endeavour to understand the exact conditions required to stimulate the stress response during both tasks. This research should also aim to quantify the level of stress caused by each task relative to robust tasks, such as the TSST, across several parameters. Finally, Dickerson and Kemeny (2004) suggest that cognitive tasks can stimulate the HPA axis enough to detect changes in cortisol. Therefore, ideally stress manipulations and the completion of cognitive tasks should be completed on separate occasions. However, in this study we had to measure craving and affect before and after the stress manipulation. Thus, completion of the cognitive tasks on the same day as the stress manipulation was required. Similarly, we were under strict time constraints due to the aforementioned interruptions, so multiple testing sessions were not feasible as they would likely have resulted in greater attrition rates and even lower statistical power.

2.4.2. Conclusion

We demonstrated that the TSST is the most robust stress task compared to the CPT and MAST. Furthermore, our findings highlight the importance of negative urgency and negative affect in the link between stress and alcohol craving following acute psychosocial stress. These results suggest that clinical interventions which aim to reduce negative urgency and negative affect may be potential targets for personalised interventions for individuals who are at risk of developing alcohol dependence in the future.

Chapter 3. Drinking During Social Isolation: A Birth Cohort Study

The research reported in this chapter has been published as:

Clay, J. M., Stafford, L. D., & Parker, M. O. (2021). Associations between self-reported inhibitory control, stress, and alcohol (mis) use during the first wave of the COVID-19 pandemic in the UK: A national cross-sectional study utilising data from four birth cohorts. *International Journal of Mental Health and Addiction, 21*(1), 350–371. https://doi.org/10.1007/s11469-021-00599-8

Chapter Foreword

This chapter presents an analysis of data collected in May 2020 during the first wave of the Centre for Longitudinal Studies COVID–19 survey. The COVID–19 pandemic had a profound impact on both my personal and professional life. For example, having to remain at home meant that I was unable to collect data in–person as originally planned. However, as new challenges arose, so did novel opportunities. For instance, between April 2020 – July 2020, my supervisors and I began discussing how we could adapt my PhD studies. As the pandemic and associated lockdowns represented a period of long–term uncertainty, and were stressful for many, a unique opportunity to test our ideas using a naturally occurring chronic stressor was unfolding. Therefore, during this time, we wrote and published a correspondence piece in *The Lancet Public Health* outlining our concerns that long periods of social isolation may lead to increased alcohol misuse and relapse among vulnerable individuals (Clay & Parker, 2020). I was also able to complete the analysis presented in this chapter alongside collecting data for my own survey, which utilised more detailed measures, and is reported in the next chapter. Overall, this chapter contributes to the aims of this thesis by investigating the impact of chronic stress and impulse control on drinking behaviour during a period of national social isolation using data from four nationally representative birth cohorts. The work was published as an original research article in the *International Journal of Mental Health and Addiction* (Clay et al., 2021). The format of the original article has been modified to match the other chapters in this thesis. However, the content largely remains the same to that which was published.

Abstract

The COVID–19 pandemic caused governments around the world to respond by imposing "lockdowns" (i.e., orders to remain at home and socially isolate) on their populations as a way of mitigating the spread of disease. Subsequently, scholars raised their concerns about the impact of lockdowns on mental health and well–being. For example, concerns that lockdowns may increase alcohol misuse among vulnerable populations. An example of such individuals are those who have high trait impulsivity. For instance, prior work demonstrates that an experimentally induced acute psychosocial stressor increased craving for alcohol and voluntary alcohol consumption. Moreover, the strength of these stress–induced behaviours were predicated on individual differences in risk–taking personality traits, stress–reactivity, and stress–recovery. Therefore, in the present study, we explored self–reported changes in alcohol use during the first wave of the pandemic in the UK, and the extent to which self–reported impulsivity and/or stress were associated with any change in pandemic drinking behaviour. Data from a UK–based cross–sectional online survey administered to four nationally representative birth cohorts (N = 13,453), aged 19–62 years, were analysed. A significant minority of 30– (29.08%) and 50–year–olds (26.67%) reported drinking more, and between 32.23–45.02% of respondents reported feeling more stressed depending on the cohort. Stress was associated with hazardous drinking among 30–year–olds (OR = 3.77, 95% CI 1.15 to 12.28). Impatience was associated with both increased alcohol use (1.14, 95% CI 1.06, 1.24) and hazardous drinking (1.20, 95% CI 1.05, 1.38) among 19–year–olds. Risk–taking was associated with hazardous drinking for 30–year–olds (OR = 1.18, 95% CI 1.05, 1.32). Therefore, these data highlight concerns for those at risk of alcohol misuse and alcohol–related harm during future lockdowns. In particular, those who may go on to develop an alcohol use disorder in the future.

Ethnics Approval Reference: ETHICS–10155 (Appendix B)

3.1. Introduction

Since being first identified in Wuhan, China, in December 2019, severe acute respiratory syndrome coronavirus 2 (SARS–CoV–2), the virus that causes COVID–19, has caused a significant threat to global health (Sohrabi et al., 2020). Governments around the world responded by imposing 'lockdowns' (orders to remain at home, and socially isolate) on their populations, and available evidence supports this action as a means of mitigating the rate of spread of the virus (Anderson et al., 2020). However, the indirect impact of lockdown on public health has raised concern, particularly relating to mental health and wellbeing (Bhattacharjee & Acharya, 2020; Gavin et al., 2020; Ornell et al., 2020; Pfefferbaum & North, 2020).

Concerns that the lockdowns may increase alcohol misuse have been raised, particularly concerning people at high–risk of developing, or re–establishing, hazardous alcohol use (Clay & Parker, 2020; Finlay & Gilmore, 2020; Rehm et al., 2020). An example of individuals who are at high risk of alcohol misuse are people that display poor impulse control (i.e., high levels of impulsivity) (Dalley & Ersche, 2019; Lee et al., 2019). Impulse control is generally conceptualised as one of the core executive functions (Diamond, 2013). It is a complex and multifaceted construct made up of several subcomponents. For example, response inhibition (i.e., action inhibition, action cancellation), sensitivity to delay (i.e., delay discounting, patience), sensitivity to risk/reward (risk–taking, sensation seeking), and attention (i.e., capacity to focus and avoid interference) (Strickland & Johnson, 2020). Indeed, several lines of evidence from pre–clinical translational work (e.g., Belin et al., 2008; Kreek et al., 2005), neuroimaging studies (e.g., Bosker et al., 2017; Voon et al., 2020), and heritability studies (e.g., Ersche et al., 2010; Khemiri et al., 2016) converge to suggest that poor impulse control is both a risk factor for the development, and consequence, of substance misuse and addiction.

The association between stress and alcohol use is also well established (Jose et al., 2000; Ruisoto & Contador, 2019; Sinha, 2012). Similar to impulsivity stress plays a critical role in both the onset and maintenance of alcohol misuse and addiction (Becker, 2017). On the one hand, the acute anxiolytic properties of alcohol motivate some individuals to drink (Kwako & Koob, 2017). On the other, perhaps counterintuitively, alcohol acts as a physiological 'stressor': acute exposure to alcohol

stimulates the HPA axis through direct activation of the paraventricular nucleus (Armario, 2010). Finally, exposure to either chronic stress or chronic alcohol misuse both lead to blunted stress responses, including dysregulation of the HPA axis – a known risk factor for hazardous drinking and addiction (Milivojevic & Sinha, 2018).

Recently we have demonstrated a complex interplay between impulse control, stress, and alcohol use, where an experimentally induced acute psychosocial stressor increased craving for alcohol (Clay et al., 2018), and voluntary alcohol consumption (Clay & Parker, 2018) in healthy (non–addicted) individuals. We found that the strength of these stress–induced increases in alcohol craving and consumption were predicated on individual differences in risk-taking personality traits, stress–reactivity, and stress–recovery. Collectively, our findings suggest these innate (e.g., trait impulsivity), and environmental (e.g., state induced stress) factors may combine to make particular individuals more at risk of alcohol misuse.

Here, we analysed the first sweep of the Centre for Longitudinal Studies (CLS) COVID–19 survey (University of London Institute of Education Centre for Longitudinal Studies, 2020a) – which was answered by individuals from five nationally representative cohorts who have been providing data since childhood – to explore: (1) self–reported changes alcohol use during the pandemic in the UK; and (2) the extent to which self–reported impulse control and/or stress were associated with any change in drinking behaviour.

3.2. Methods

3.2.1. Data source

We used data from the first wave of the CLS COVID–19 survey (University of London Institute of Education Centre for Longitudinal Studies, 2020a). The survey design, recruitment procedure, and fieldwork processes have been described in detail elsewhere (Brown et al., 2020). Briefly, the survey was administered between 2 and 31 May 2020, using Qualtrics (Provo, Utah), to 50,479 individuals from five nationally representative UK birth cohorts. These included: (1) the Millennium Cohort Study (MCS), who are part of 'Generation Z', and were aged 19; (2) Next Steps, who are part of the 'Millennial' generation, who were aged 30; (3) the 1970 British Cohort Study (BCS70), who belong to

‘Generation X’ – aged 50; (4) the National Child Development Study (NCDS), who were aged 62 and were born in the latter part of the ‘Baby Boomer’ generation; and (5) the National Study of Health and Development (NSHD), who were born at the beginning of the ‘Baby Boomer’ era, and were aged 74. Due to the nature of the survey, only those who had their email address previously recorded were approached. Overall, 18,042 of those invited responded, achieving a response rate (RR) of 35.7%. This response rate is similar to comparable national web surveys conducted at this time, such as the Understanding Society COVID–19 survey (Institute for Social and Economic Research, 2020). Ethnicity data was linked from previous survey waves (Kelly, 2008; University of London Institute of Education Centre for Longitudinal Studies, 2016, 2020c, 2020b). All data used in this study are available from the UK Data Service Website (https://ukdataservice.ac.uk/) under the “Safeguarded” data access policy.

3.2.2. Study sample

Due to data availability at the time of analysis, four of the five cohorts included in the COVID–19 survey were analysed. Namely, the MCS cohort members (n = 2,645, RR = 26.59%), Next Steps (n = 1,907, RR = 20.33%), the BCS70 (n = 4,223, RR = 40.38%), and the NCDS (n = 5,178, RR = 57.90%). The study was restricted to UK–based respondents; thus emigrants (n = 500) were excluded prior to analysis. This left 13,453 cases for analysis. A detailed overview of the study sample is presented in the Appendix B. Selected sample characteristics are shown in Table 3.1.

3.2.3. Outcome measures

Alcohol use behaviour was assessed using five questions from the AUDIT (see Chapter 2 for details) – a tool developed by the World Health Organisation as a brief assessment of alcohol misuse (Babor et al., 2001). The original AUDIT has been shown to have excellent psychometric properties when used to assess alcohol use disorders in a variety of settings including both college students (Fleming et al., 1991) and during routine health examinations (Claussen & Aasland, 1993). Subsequently, several short versions of the AUDIT have been developed and shown to perform similarly to the original instrument (Gual, 2002; Kim et al., 2013).

Table 3.1 Sociodemographic characteristics of the sample and descriptive statistics (*M* and *SD*) for selected variables.

	MCS (*n* = 2,644)			Next Steps (*n* = 1,852)			BCS70 (*n* = 3,997)			NCDS (*n* = 4,960)		
Variable	**Statistic**	**LL**	**UL**	**Statistic**	**LL**	**UL**	**Statistic**	**LL**	**UL**	**Statistic**	**LL**	**UL**
Age in years	19			30			50			62		
Sex, %												
Male	49.46	46.47	52.45	43.14	39.19	47.18	51.05	48.49	53.62	50.44	48.24	52.64
Female	50.54	47.55	53.53	56.86	52.82	60.81	48.95	46.38	51.51	49.56	47.36	51.76
Ethnicity %												
White	85.79	82.21	88.75	87.32	85.05	89.29	96.74	95.83	97.45	96.16	94.65	97.25
Black	5.15	3.49	7.55	2.13	1.38	3.27	1.35	0.88	2.06	1.45	0.78	2.67
Indian/Pakistani	4.93	3.29	7.32	4.85	3.75	6.26	1.18	0.78	1.78	1.60	0.92	2.74
Mixed Race	1.24	0.43	3.48	2.37	1.71	3.27	0.44	0.25	0.76	0.34	0.19	0.61
Other/Unsure	2.89	1.88	4.43	3.34	2.23	4.97	0.30	0.15	0.59	0.46	0.23	0.92
Relationship status, %												
Cohabiting relationship	6.88	5.56	8.48	65.40	61.81	68.82	68.76	66.06	71.34	67.55	65.32	69.70
Non–cohabiting relationship	33.75	30.72	36.93	11.73	9.77	14.04	10.63	9.06	12.44	13.39	11.85	15.10
Single	59.37	56.18	62.48	22.87	19.79	26.28	20.61	18.26	23.17	19.07	17.26	21.01
COVID–19 Status, %												
Yes, confirmed	0.32	0.13	0.80	0.57	0.29	1.13	0.68	0.24	1.90	0.33	0.20	0.53
Yes, unconfirmed	5.17	4.11	6.48	10.26	8.08	12.95	9.54	7.94	11.41	5.42	4.62	6.35
Unsure	21.31	18.73	24.14	23.57	20.71	26.69	25.44	23.18	27.85	19.91	18.28	21.65
No	73.20	70.33	75.89	65.60	62.19	68.86	64.34	61.71	66.88	74.34	72.47	76.13

Economic activity, %												
Employed	62.61	57.17	67.75	80.82	77.42	83.81	69.34	66.61	71.93	44.05	41.85	46.27
Self–employed	2.43	1.41	4.17	6.32	4.67	8.49	12.81	11.36	14.43	12.13	10.52	13.93
Unpaid/voluntary work	0.11	0.02	0.48	0.21	0.09	0.52	0.14	0.07	0.28	0.48	0.31	0.73
Apprenticeship	6.16	4.32	8.73	0.11	0.03	0.38	-	-	-	-	-	-
Unemployed	20.40	15.79	25.94	4.10	2.82	5.92	3.96	2.69	5.78	3.56	2.65	4.78
Permanently sick or disabled	0.44	0.17	1.10	0.75	0.37	1.51	6.02	4.33	8.33	5.59	4.34	7.17
Looking after home or family	1.13	0.49	2.59	4.27	2.83	6.39	5.08	4.05	6.35	4.58	3.89	5.39
In education	1.43	0.67	3.03	-	-	-	0.03	0.01	0.15	-	-	-
Retired	-	-	-	-	-	-	1.02	0.61	1.70	28.04	26.29	29.86
Uncategorised	5.29	3.39	8.16	3.43	2.26	5.19	1.61	0.91	2.81	1.56	0.86	2.82
Key worker, %												
Yes	9.36	7.50	11.61	33.57	30.03	37.29	31.63	29.45	33.89	18.88	17.30	20.57
No	90.64	88.39	92.50	66.43	62.71	69.97	68.37	66.11	70.55	81.12	79.43	82.70
NS–SEC analytical classes, %												
Higher managerial	0.78	0.45	1.34	16.80	14.14	19.84	15.83	14.10	17.72	7.31	6.27	8.51
Lower managerial	3.05	1.90	4.86	29.71	26.35	33.30	20.48	18.73	22.35	12.23	10.87	13.73
Intermediate occupations	5.56	4.48	6.88	17.46	14.61	20.74	13.42	12.11	14.83	9.60	8.61	10.69
Small employers and self–employed	1.09	0.68	1.75	2.80	1.86	4.20	5.19	4.21	6.38	4.48	3.57	5.62
Lower supervisory and technical	2.39	1.55	3.68	3.19	1.86	5.43	4.87	3.94	5.99	3.84	3.04	4.86
Semi–routine occupations	11.35	9.29	13.81	9.23	7.35	11.53	9.66	8.35	11.14	9.59	8.27	11.09
Routine occupations	5.65	4.50	7.07	3.71	2.56	5.35	7.44	6.13	9.00	6.03	5.07	7.16

Uncategorised	70.13	66.38	73.62	17.09	14.56	19.96	23.12	20.56	25.91	46.92	44.72	49.12
Change in drinking, %												
Less	49.45	46.00	52.90	21.03	18.04	24.37	11.52	9.96	13.30	16.29	14.55	18.18
Same	36.43	33.34	39.63	49.89	45.93	53.85	61.81	59.28	64.28	65.43	63.25	67.54
More	14.12	11.49	17.24	29.08	25.65	32.77	26.67	24.49	28.96	18.28	16.78	19.89
Risk of alcohol-related harm at time of survey, %												
Low risk	77.87	74.33	81.04	70.68	66.79	74.29	59.99	57.48	62.45	62.29	60.16	64.38
Increasing risk	17.29	14.76	20.15	23.65	20.36	27.30	32.25	30.01	34.58	31.55	29.62	33.55
High risk	1.49	0.90	2.46	2.10	1.16	3.78	2.74	2.21	3.39	2.63	2.15	3.22
Highest risk	3.35	2.04	5.47	3.56	2.20	5.72	5.02	3.85	6.51	3.52	2.73	4.53
Change in stress, %												
Less	17.86	15.19	20.88	9.94	7.84	12.52	10.86	9.40	12.52	7.13	6.18	8.22
Same	44.97	41.73	48.25	45.04	41.32	48.82	50.28	47.68	52.88	60.64	58.49	62.75
More	37.17	34.37	40.06	45.02	41.30	48.80	38.85	36.33	41.44	32.23	30.25	34.28
Risk-taking, M (SD)	7.01 (2.18)	6.86	7.15	6.64 (2.22)	6.48	6.80	5.99 (2.53)	5.84	6.14	5.91 (2.64)	5.78	6.04
Impatience, M (SD)	4.30 (2.60)	4.14	4.46	4.27 (2.83)	4.04	4.51	4.03 (2.58)	3.89	4.17	3.88 (2.87)	3.73	4.03

Note. MCS = Millennium Cohort Study, BCS70 = 1970 British Cohort Study, NCDS = National Child Development Study; NS-SEC = National Statistics Socio–economic class prior to the outbreak. Economic activity reflects activity during the pandemic.

The questions administered during the survey were:

1. *"How often have you had a drink containing alcohol?"*
2. *"How many standard alcoholic drinks have you had on a typical day when you were drinking?"*
3. *"How often have you found you were not able to stop drinking once you had started?"*
4. *"How often have you failed to do what was expected of you because of drinking?"*
5. *"Has a relative, friend, doctor, or health worker been concerned about your drinking or advised you to cut down?"*

Questions one and two were repeated, prefaced by either "in the month before the Coronavirus outbreak", or "since the start of the Coronavirus outbreak". This provided an assessment of alcohol use prior to, and during, the pandemic. Questions one to five were posed in the context of the pandemic, thus were worded using the latter phrasing, offering an assessment of hazardous drinking during the outbreak.

Each item was scored in line with the original AUDIT. Scores which represented alcohol use prior to and during the pandemic were calculated by summing questions one and two. A change score was calculated by subtracting the pre–pandemic from intra–pandemic score. Thus, values equal to zero reflected no change, values greater than zero represented an increase, and values less than zero denoted a reduction in alcohol use. A score representing risk of alcohol–related harm due to hazardous drinking during the pandemic was calculated by summing all items which used the latter wording. The hazardous drinking score was categorised proportionally to the original AUDIT. Whereby, a score between zero and three was coded as "Low risk"; a score between four and six was classified as "Increasing risk"; scores between seven and eight were labelled "Higher risk"; and scores of nine or greater were classed as "Highest risk".

3.2.4. Stress

Perceived stress was assessed using a single question: "Since the Coronavirus outbreak, please indicate how the following have changed… The amount of stress I've been feeling". The possible responses included "More than before", "Same – no change", and "Less than before". As it is well–known that

experiencing symptoms of depression and/or anxiety is associated with increased psychological stress (Crawford & Henry, 2003; Heinen et al., 2017), we used linear regression models for each cohort to determine the relationship between scores on the PHQ–4 (Kroenke et al., 2009) – an ultra–brief tool, with good psychometric properties, designed to screen for anxiety and depression in both clinical and non–clinical settings – and the stress item used here (see Appendix B). After controlling for potential confounders (see section 3.2.6), individuals who said they were feeling more stressed than before the pandemic scored approximately two points higher (range = 1.95 – 2.68, *p*s < .001) than those who said they felt the same.

3.2.5. Impulse control

Two self–report measures of impulse control were administered in the survey: patience and risk–taking. Each was measured using a single ten–point Likert scale item. The questions were phrased "On a scale from 0 – 10, where 0 is 'never' and 10 is 'always', how willing to take risks/patient would say you are?". A similar single–item scale of risk preference, known as the General Risk Question (GRQ) (Dohmen et al., 2011), has been used extensively and has been included in several widely analysed surveys, such as the Household, Income and Labour Dynamics in Australia Survey (Watson & Wooden, 2012), and the Understanding Society Survey (Institute for Social and Economic Research, 2018). Recent work suggests that the self–report (e.g., GRQ) assessment of risk–taking oftentimes outperform behavioural assessments (e.g., laboratory lotteries) due to self–report assessments taking subjective internal states, such as regret or need, into account (Arslan et al., 2020). Moreover, during the development of the Global Preferences Survey (Falk et al., 2018) – which was conducted to investigate risk and time (patience) preferences – Falk et al. (Falk et al., 2016) experimentally validated their measures by (among other things) assessing the association between single–item assessments and behavioural measures of the same constructs through Spearman's correlations and linear regression models. Their analysis shows that the single–item assessments were moderately correlated with the behavioural measures (see Appendix B). The "patience" item was reverse scored to reflect greater impatience.

3.2.6. Potential confounders

Potential confounding variables were identified using the authors' substantive knowledge about established risk factors that could plausibly be related to our outcome variables. These included respondent's sex, ethnicity, National Statistics Socio–economic Class (NS–SEC) prior to the outbreak of Coronavirus, and economic activity during the pandemic. Further information on these measures is presented in the Appendix B.

3.2.7. Analysis

Statistical analysis was conducted using Stata IC (version 16.1). Figures were generated using *ggplot2* (version 3.3.2) for R (version 3.6.2) was used to create figures. Inverse probability weighting was used to account for bias introduced due to missing data, and to ensure the results were as representative as possible (Seaman & White, 2013). The overall percentage of missing data was 23.43%. The median percentage of missing data by variable was 5.29% (IQR = 8.01%). See Table B3 for a detailed description of missing data. Separate analyses were conducted for each cohort due to differences in sampling methods and therefore design weights. Descriptive statistics (mean and standard deviation or proportion alongside 95% CIs) were calculated our variables of interest and select demographic variables. Prevalence estimates (with 95% CIs) split by sex, ethnicity, economic activity, and NS–SEC were calculated for our outcome measures and change in stress. Ordinal regression models were used to assess whether sociodemographic sub–group membership was associated with change in alcohol use, risk of alcohol–related harm due to hazardous drinking, or a change in stress levels, and to investigate associations between impulse control, stress, and alcohol use. We first regressed our outcome measures and change in stress on sex, ethnicity, economic activity, and NS–SEC. We then added parameters for impulse control, stress, and the interaction between impulse control and stress to our models containing our outcome variables. Given that most respondents across all cohorts were White, and since some ethnic groups made up less than one percent of the sample, a dichotomous White/non–White variable was used in regression analyses. We also noticed that the standard error among fifty–year–olds that reported being in education during the pandemic was inflated, leading to implausible results, due to only two fifty–year–olds females falling into this category. These two cases were omitted for all

regression analyses, which had no impact on the final results of the models. For brevity, model estimates for potential confounders are reported in the Supplementary Tables B4–B15. Finally, as neither the study nor analysis plan were pre–registered on a publicly available platform, the results should be considered exploratory.

3.3. Results

3.3.1. Changes in alcohol use during the first lockdown

Across all cohorts, most respondents reported drinking the same amount of alcohol or less since the start of the pandemic (Table 3.1). Thirty–year–olds and fifty–year–olds were most likely to report increased drinking with around one–third and one–quarter reporting an increase respectively.

Figure 3.1 shows change in alcohol use by subgroup. In all cohorts except for sixty–two–year–olds, being employed was associated with reporting increased alcohol use (Tables B4–B6). Fifty–year–old and sixty–two–year–old females had 1.27 (95% CI 1.08 to 1.50) and 1.23 (95% CI 1.02 to 1.50) times the odds of reporting increased alcohol use, respectively (Tables B6 and B7). Regarding socio–economic class, fifty–year–olds who worked in intermediate occupations (OR = 0.70, 95% CI 0.54 to 0.92), semi–routine occupations (OR = 0.62, 95% CI 0.46 to 0.85), and routine occupations (OR = 0.62, 95% CI 0.39 to 0.98), and sixty–two–year–olds in lower supervisory and technical occupations (OR = 0.45, 95% CI 0.24 to 0.84) were less likely to report an increase in alcohol use compared to those in higher managerial positions (Tables B6 and B7). Finally, among thirty–year–olds, non–White ethnicity was associated with a 29% (OR = 0.71, 95% CI 0.55 to 0.93) reduction in the odds of reporting increased drinking (Table B4).

3.3.2. Risk of alcohol–related harm due to hazardous drinking during the first lockdown

Most participants fell into the low–risk category regardless of age or sub-group membership since the start of the lockdown (Table 3.1, Figure 3.2). Approximately one-fifth of nineteen-year–olds, one–third of thirty–year–olds, and two–fifths of both fifty–year-olds and sixty–two–year-olds were at an increased risk of alcohol–related harm or worse. Of these, approximately 60.50% (95% CI 48.73 to 71.17) of nineteen–year–olds, 59.93% (95% CI 52.51 to 66.92) of thirty–year–olds, 68.11% (95% CI 63.14 to

72.71) of fifty–year-olds, and 69.28% (95% CI 49.96, 73.29) of sixty–two-year–olds reported an increase in alcohol use since the start of the pandemic.

Figure 3.2 shows risk of alcohol–related harm due to hazardous drinking by sub–group. Among nineteen–year–olds, being employed or in education was associated with an increase in the odds of being more at risk of alcohol–related harm (Table B8). For thirty, fifty, and sixty-two–year–olds (Tables B9–B11), being female and non–White ethnicity was associated with decreased odds of alcohol–related harm due to hazardous drinking. Finally, some effects were cohort specific. Being a permanently sick or disabled fifty–year–old was associated with a 76% (OR = 0.24, 95% CI 0.10 to 0.58) decrease in the odds of alcohol related harm compared to those who were employed (Table B10). Similarly, sixty–two–year–olds who worked in routine occupations (OR = 0.56, 95% CI 0.33 to 0.96) were less likely to drink hazardously (Table B11).

3.3.3. Change in stress during the first lockdown

Across all cohorts, most participants reported experiencing the same amount or less stress since the start of the pandemic (Table 3.1). Approximately two–fifths of nineteen–year–olds, half of thirty–year–olds, two–fifths of fifty–year–olds, and one–third of sixty–two–year–olds reported feeling more stressed. Of those, females were disproportionately affected (Figure 3.3). More specifically, among nineteen–year–olds, being female was associated with 1.54 (95% CI 1.08 to 2.20) times the odds of reporting an increase in stress (Supplementary Table 12). For thirty–year–olds, being female was associated with 1.93 (95% CI 1.39 to 2.70) times the odds of reporting an increase in stress (Table B13). For fifty–year–olds, being female was associated with 1.62 (95% CI 1.37 to 1.92) times the odds of reporting an increase in stress (Table B14). For sixty–two–year–olds, being female was associated with 2.03 (95% CI 1.66 to 2.48) times the odds of reporting an increase in stress (Table B15). Additionally, for nineteen–year–olds being either self-employed (OR = 5.53, 95% CI 1.56 to 19.57) or unemployed (OR = 1.75, 95% CI 1.08 to 2.83) was associated with an increase in the odds of reporting an increase in stress (Table B12). Similarly, for thirty–year–olds, being unemployed (OR = 2.14, 95% CI 1.15 to 3.98) was also associated with an increase in the odds of reporting an increase in stress (Table B13).

Figure 3.1 Change in alcohol use during the first wave (May 2020) of the COVID–19 pandemic in the UK, utilising data from four birth cohorts: The Millennium Cohort Study (*n* = 2,645), Next Steps (*n* = 1,907), the 1970 British Cohort Study (*n* = 4,223), and the National Child Development Study (*n* = 5,178) by sex (panel A), ethnicity (panel B), economic activity during the pandemic (panel C), and National Statistics Socio–economic Class (panel D). Point estimates represent weighted percentages, error bars represent 95% confidence intervals.

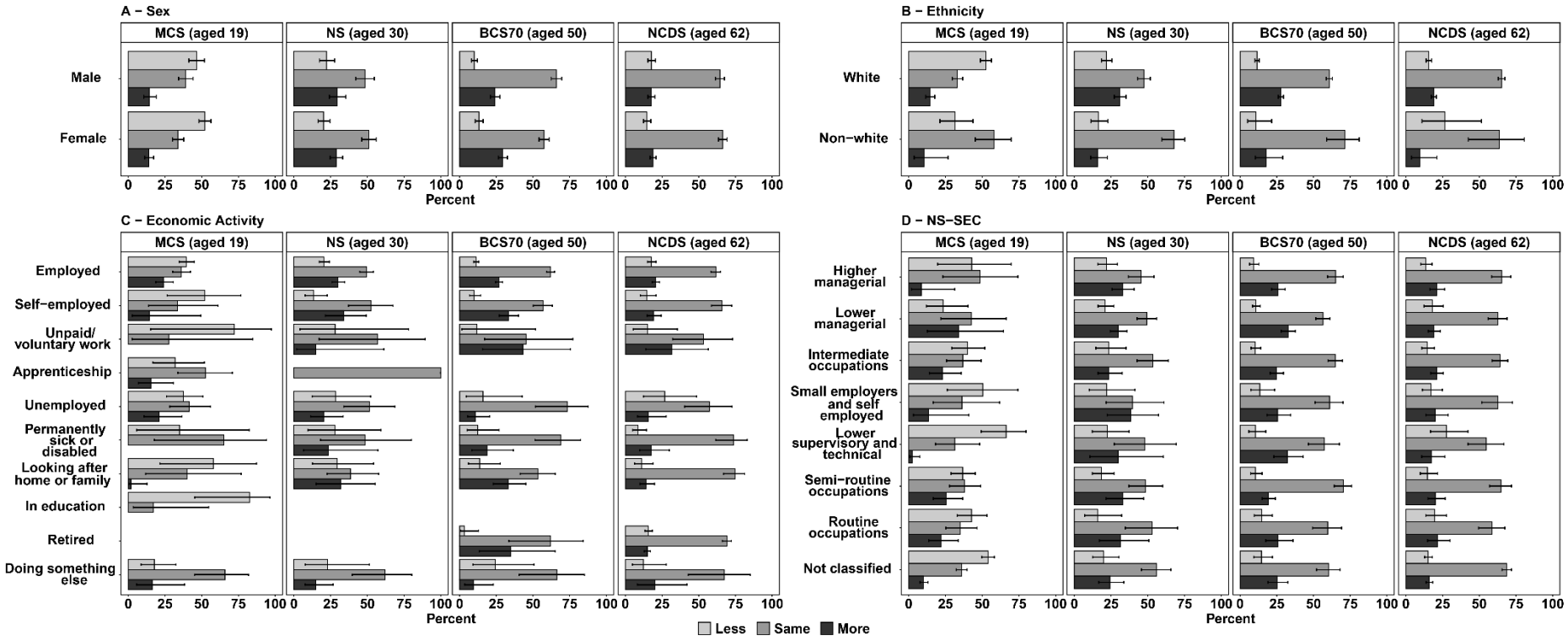


Figure 3.2 Risk of alcohol–related harm due to hazardous drinking during the first wave (May 2020) of the COVID–19 pandemic in the UK, utilising data from four birth cohorts: The Millennium Cohort Study (*n* = 2,645), Next Steps (*n* = 1,907), the 1970 British Cohort Study (*n* = 4,223), and the National Child Development Study (*n* = 5,178) by sex (panel A), ethnicity (panel B), economic activity during the pandemic (panel C), and National Statistics Socio–economic Class (panel D). Point estimates represent weighted percentages, error bars represent 95% confidence intervals.

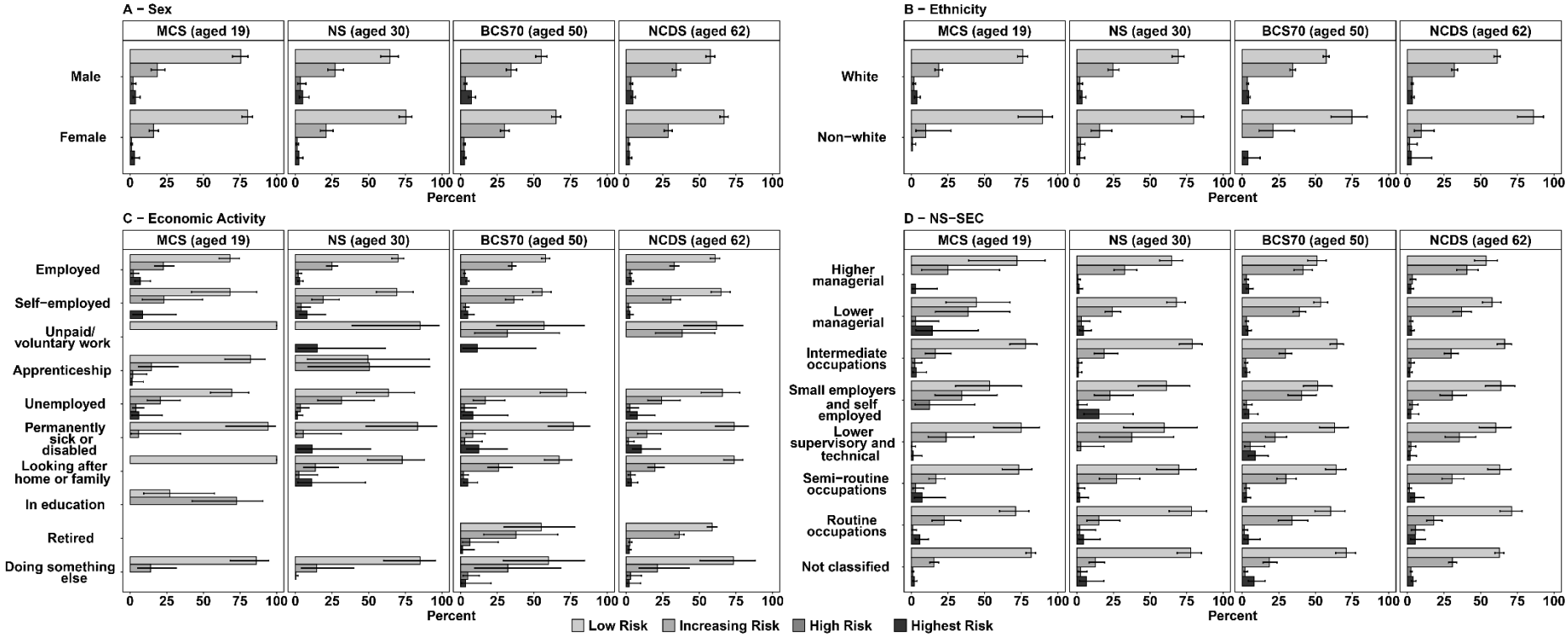
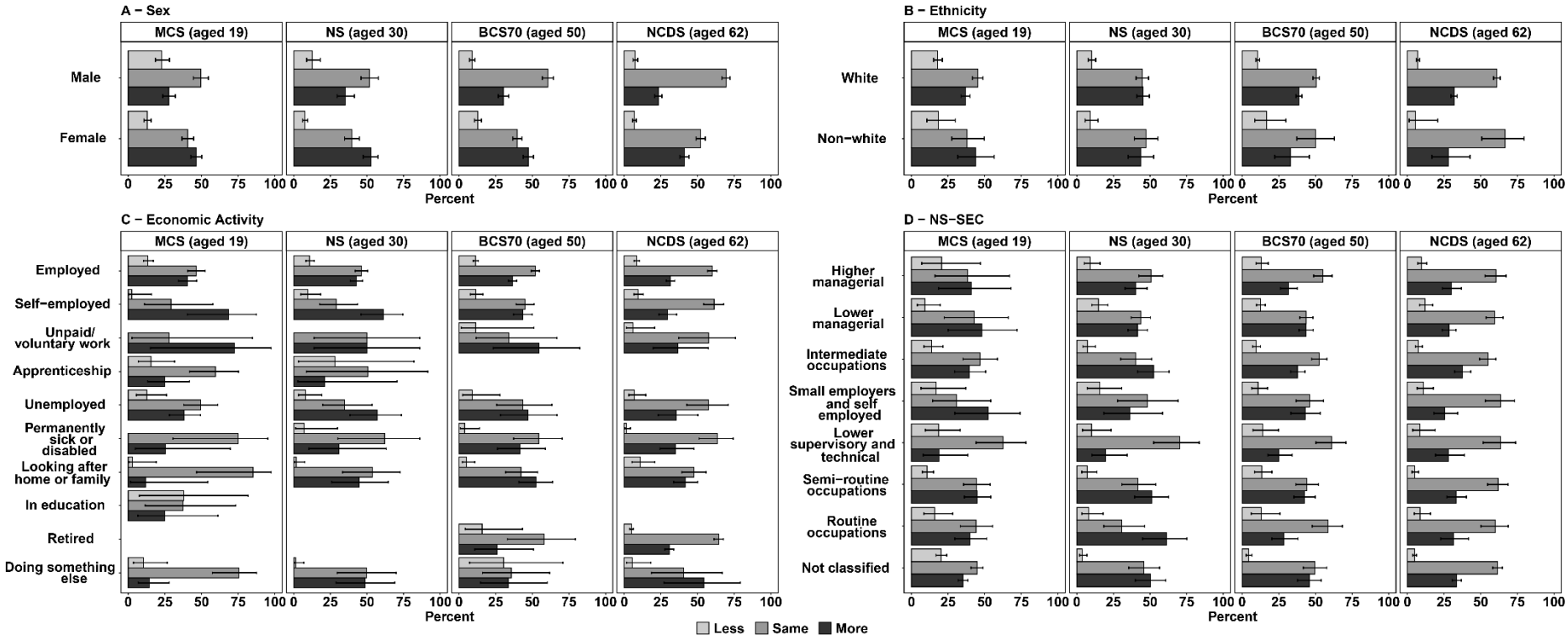


Figure 3.3 Change in perceived stress during the first wave (May 2020) of the COVID–19 pandemic in the UK, utilising data from four birth cohorts: The Millennium Cohort Study (*n* = 2,645), Next Steps (*n* = 1,907), the 1970 British Cohort Study (n = 4,223), and the National Child Development Study (*n* = 5,178) by sex (panel A), ethnicity (panel B), economic activity during the pandemic (panel C), and National Statistics Socio–economic Class (panel D). Point estimates represent weighted percentages, error bars represent 95% confidence intervals.



3.3.4. Associations between stress, impulse control, and drinking behaviour

Stress

After adjusting for potential confounders, thirty–year–olds who reported feeling more stressed since the start of lockdown were at 3.77 (95% CI 1.15 to 12.28) times greater odds of being at increasing, high, or highest (versus low) risk of alcohol–related harm, compared to those that reported feeling no change in stress (Table 3.2). There was no evidence to suggest that this effect was present in other cohorts.

Impatience

Among nineteen–year–olds, a one unit increase in impatience was associated with 1.14 (95% CI 1.06 to 1.24) times the odds of reporting an increase in alcohol use, and 1.20 (OR = 1.20, 95% CI 1.05 to 1.38) times the odds of alcohol–related harm due to hazardous drinking after controlling for potential confounders (Table 3.2). There was no evidence to suggest that this effect was present in other cohorts.

Risk–taking

After controlling for potential confounders, a one unit increase in risk–taking was associated with 1.18 (95% CI 1.05 to 1.32) times the odds of alcohol-related harm among thirty–year-olds (Table 3.2). Similarly, for fifty–year-olds, a one unit increase in risk-taking was associated with 1.06 (95% CI 1.01 to 1.12) times the odds of alcohol–related harm. This effect was not observed in other cohorts (Table 3.2).

Stress x personality interactions

There was evidence to suggest that, after controlling for potential confounders, individuals who were more impatient and less stressed tended to drink more and be at a greater risk of alcohol–related harm (Table 3.2). Specifically, for thirty–year–olds, a one unit increase in impatience was associated with a 22% (OR = 1.22, 95% CI 1.00 to 1.48) increase in the odds of reporting an increase in alcohol use among those who reported feeling less stressed, and a 12% (OR = 0.88, 95% CI 0.80 to 0.98) decrease in the odds of reporting an increase in alcohol use among those who reported feeling more stressed. Similarly, among nineteen–year–olds that reported feeling more stressed, a one unit increase in impatience was associated with a 13% (OR = 0.87, 95% CI 0.77 to 0.99) decrease in the odds of reporting an increase in alcohol use. In terms of risk of alcohol–related harm, for both thirty-year-olds

Table 3.2 Summary of the final ordinal regression models predicting change in drinking since the start of the pandemic (model A) and risk of alcohol¬–related harm due to hazardous drinking during the pandemic (model B), adjusting for sex, ethnicity, economic activity during the pandemic, and social class prior to the pandemic.

	MCS			Next Steps			BCS70			NCDS		
Variable	**OR (95% CI)**	**SE**	***p***	**OR (95% CI)**	**SE**	***p***	**OR (95% CI)**	**SE**	***p***	**OR (95% CI)**	**SE**	***p***
Model A: Change in drinking since the start of the pandemic												
Stress												
Same	Ref.			Ref.			Ref.			Ref.		
Less	0.21 (0.02, 1.98)	0.24	0.172	0.59 (0.09, 3.71)	0.55	0.574	1.40 (0.58, 3.38)	0.63	0.455	1.38 (0.42, 4.51)	0.83	0.590
More	1.47 (0.39, 5.61)	1.00	0.568	2.21 (0.99, 4.94)	0.90	0.053	0.87 (0.51, 1.47)	0.23	0.594	0.90 (0.54, 1.48)	0.23	0.670
Risk-taking	0.98 (0.88, 1.10)	0.06	0.775	1.03 (0.95, 1.13)	0.05	0.479	0.98 (0.94, 1.03)	0.02	0.533	0.99 (0.94, 1.03)	0.02	0.508
Risk-taking x Stress												
Same	Ref.			Ref.			Ref.			Ref.		
Less	1.2 (0.92, 1.57)	0.16	0.181	0.96 (0.74, 1.24)	0.13	0.760	0.96 (0.85, 1.08)	0.06	0.478	0.97 (0.82, 1.13)	0.08	0.674
More	1.05 (0.87, 1.26)	0.10	0.622	0.98 (0.87, 1.09)	0.06	0.676	1.06 (0.98, 1.15)	0.04	0.152	1.07 (0.98, 1.16)	0.04	0.118
Impatience	**1.14 (1.06, 1.24)**	**0.05**	**0.001**	1.05 (0.97, 1.14)	0.04	0.201	0.98 (0.93, 1.03)	0.02	0.370	0.99 (0.95, 1.03)	0.02	0.504
Impatience x Stress												
Same	Ref.			Ref.			Ref.			Ref.		
Less	0.92 (0.75, 1.12)	0.09	0.404	**1.22 (1.01, 1.48)**	**0.12**	**0.047**	1.01 (0.9, 1.14)	0.06	0.846	1.01 (0.89, 1.15)	0.07	0.869
More	**0.87 (0.77, 0.99)**	**0.06**	**0.030**	**0.88 (0.80, 0.98)**	**0.05**	**0.016**	1.05 (0.97, 1.13)	0.04	0.216	0.99 (0.93, 1.07)	0.04	0.875
Model B: Risk of alcohol-related harm due to hazardous drinking.												
Stress												

Same	Ref.			Ref.			Ref.			Ref.		
Less	0.25 (0.01, 4.98)	0.38	0.361	0.36 (0.06, 2.15)	0.33	0.259	1.00 (0.35, 2.89)	0.54	0.999	0.74 (0.25, 2.16)	0.41	0.585
More	0.82 (0.18, 3.65)	0.62	0.794	**3.77 (1.15, 12.28)**	**2.27**	**0.028**	1.29 (0.73, 2.25)	0.37	0.380	0.88 (0.49, 1.60)	0.27	0.680
Risk-taking	0.98 (0.8, 1.19)	0.10	0.836	**1.18 (1.05, 1.32)**	**0.07**	**0.006**	**1.06 (1.01, 1.12)**	**0.03**	**0.017**	1.00 (0.95, 1.05)	0.03	0.945
Risk-taking x Stress												
Same	Ref.			Ref.			Ref.			Ref.		
Less	1.32 (0.89, 1.96)	0.27	0.172	0.97 (0.77, 1.22)	0.11	0.813	0.95 (0.82, 1.1)	0.07	0.504	1.04 (0.89, 1.22)	0.08	0.631
More	1.13 (0.91, 1.41)	0.12	0.258	0.88 (0.77, 1.02)	0.07	0.098	1.01 (0.93, 1.09)	0.04	0.834	1.08 (0.99, 1.18)	0.05	0.091
Impatience	**1.20 (1.05, 1.38)**	**0.08**	**0.010**	0.97 (0.9, 1.06)	0.04	0.531	1.00 (0.95, 1.04)	0.02	0.859	1.02 (0.97, 1.06)	0.02	0.480
Impatience x Stress												
Same	Ref.			Ref.			Ref.			Ref.		
Less	0.9 (0.73, 1.12)	0.10	0.359	**1.31 (1.1, 1.57)**	**0.12**	**0.002**	**1.17 (1.04, 1.31)**	**0.07**	**0.007**	1.04 (0.94, 1.16)	0.06	0.435
More	0.95 (0.8, 1.14)	0.09	0.603	0.95 (0.83, 1.07)	0.06	0.396	1.00 (0.93, 1.08)	0.04	0.943	1.00 (0.92, 1.09)	0.04	0.972

(OR = 1.31, 95% CI 1.10 to 1.57) and fifty–year–olds (OR = 1.17, 95% CI 1.04 to 1.31), reporting feeling less stressed was associated with an increase in the odds of being at an increased risk of alcohol-related harm or worse. No stress x personality interactions were observed in the sixty-two-year-old cohort. No stress x risk-taking interactions were observed in any group.

3.4. Discussion

The present study utilised data from four nationally representative British birth cohorts to explore changes in alcohol use behaviour and stress since the start of the COVID–19 outbreak, during the first national lockdown in May 2020. Across all age–groups (cohorts), we found evidence to suggest that most respondents drank the same amount or less since the start of the pandemic. However, between approximately fourteen and thirty percent of respondents reported drinking more depending on age. Of these, thirty–year–olds and fifty–year–olds were most likely to report an increase in drinking. This supports recent emerging evidence which suggests that between one–fifth and one–third of individuals in the UK reported drinking more during the first wave of the pandemic (Institute of Alcohol Studies, 2020; Jacob et al., 2021; Niedzwiedz et al., 2020). Further, between twenty and forty percent of participants drank at levels of increasing risk of alcohol–related harm or worse, depending on age, with older participants displaying the greatest levels of risk due to alcohol misuse. Of these, approximately sixty percent of both nineteen–year–olds and thirty–year–olds, and seventy percent of both fifty–year–olds and sixty–two–year–olds reported drinking more since the start of the pandemic. Provisional data from the Office for National Statistics data suggests that alcohol–related deaths reached a 20–year high between quarter one (January to March) and quarter three (July to September) of 2020; with significant increases in mortality among those aged between thirty and forty–nine in quarter two and forty to sixty–nine in quarter three (Office for National Statistics, 2021). These data add concerning weight to our findings of the higher rates of harmful drinking in these age groups, supporting the public health concerns attributable to excess alcohol use in some at–risk individuals during lockdown (Clay & Parker, 2020; Finlay & Gilmore, 2020; Rehm et al., 2020). The increase in alcohol–related deaths could be, at least partly, attributable to changes in mental health service provision during the pandemic and therefore

increased psychological distress on top of that directly associated with stay–at–home orders (Columb et al., 2020; Da et al., 2020).

Similar to changes in drinking behaviour, most participants reported experiencing the same amount or less stress since the start of the pandemic. Nevertheless, between approximately thirty and forty–five percent of respondents reported an increase in their stress level. Of these, thirty–year–olds seemed to be most affected as more respondents from this group reported increased stress compared to the other cohorts. This group also had the highest proportion of individuals that reported increased alcohol use and there was evidence of an association between stress and hazardous drinking here too. Analogous to this finding, previous research suggests that the Millennial generation struggle with stress management considerably more than previous generations (Bland et al., 2012). Similarly, recent data from the UK Household Longitudinal Survey (Etheridge & Spantig, 2020) suggests that young individuals have seen larger declines in well–being during the first lockdown. Surprisingly, despite the well–established link between substance use and stress (Jose et al., 2000; Ruisoto & Contador, 2019), a main effect of stress was not observed in any other group. However, in all cohorts, being female was associated with an increased likelihood of reporting heightened stress; an effect which has consistently been reported elsewhere (Etheridge & Spantig, 2020; Niedzwiedz et al., 2020; Stanton et al., 2020). This may be due to (for example) an increased risk of psychiatric symptoms prior to, and after, suffering with COVID–19; an increased risk of domestic violence; and a disproportionate responsibility for domestic tasks including caring for family members (Almeida et al., 2020). In terms of drinking, our results suggest that for the fifty– and sixty–two–year–olds cohorts, being female was associated with an approximate twenty–five percent increase in the odds of reporting an increased alcohol use. Interestingly, however, across all cohorts, except the nineteen–year–olds, being female was associated with around a forty percent reduction in the odds of alcohol–related harm due to hazardous drinking.

Several sociodemographic characteristics were related to change in both stress and alcohol use behaviour. For instance, in all but the oldest cohorts, employment was related to reporting increased alcohol use; and in the youngest cohort, both being employed or in–education was associated with an increased likelihood of hazardous drinking and subsequent alcohol–related harm. Similarly, among

fifty–year–olds those in higher managerial positions were more likely to report increased alcohol use. Meanwhile, for those aged sixty–two, higher managerial positions were associated with an increased risk of alcohol related harm due to hazardous drinking. As off–premises alcohol consumption has been classified as 'essential' by the UK government (Reynolds & Wilkinson, 2020), this association is likely related to the physical and financial availability of alcohol (Babor et al., 2010; Rehm et al., 2020). In other words, those that are employed and/or high earners will generally be able to (financially) afford to drink more. Regarding changes in stress, unemployment was related to an increased likelihood of reporting heightened stress among both nineteen– and thirty–year–olds. Also, self–employed nineteen–year–olds were more likely to report increased stress. Again, this was most likely associated with financial stability. For instance, many people who rely on state welfare have been receiving Universal Credit which has been shown to be associated with psychological distress (Wickham et al., 2020), and recent research has shown that self–employed people have suffered a large and disproportionate reduction in income during the pandemic (Yue & Cowling, 2021). Finally, in all but the youngest cohorts, there was evidence to suggest that non–White ethnicity was associated with a decreased likelihood of alcohol–related harm due to hazardous drinking; and among thirty–year–olds non–White ethnicity was associated with a decreased likelihood of reporting and increase in alcohol use. This was unsurprising considering that results from several papers suggest that being White is a risk–factor for alcohol use and misuse (Bécares et al., 2011; Rao et al., 2015; Twigg & Moon, 2013).

Self–reported impulse control, and in some cases, a complex interaction between stress and personality were related to alcohol use and hazardous drinking during the lockdown. For example, in thirty– and fifty–year–olds, risk–taking personality was associated with an increased propensity to consume more alcohol and to have higher hazardous drinking scores. This corresponds to a large volume of literature which associates poor impulse control with substance misuse (Belin et al., 2008; Bosker et al., 2017; Dalley & Ersche, 2019; Ersche et al., 2010; Khemiri et al., 2016; Kreek et al., 2005; Lee et al., 2019; Voon et al., 2020). Moreover, the majority of nineteen–year–olds reported drinking less since the start of the pandemic. This was unsurprising considering the recent evidence of the 'devaluation of alcohol' among Generation Z (Kraus et al., 2020). This finding may also have been

driven by the closure of on–trade drinking locations since drinking at venues such as pubs and bars is more common among young people (Ally et al., 2016), and reduced exposure to environments related with alcohol consumption has been associated with a reduction in drinking among young individuals during the pandemic (Winstock et al., 2020). However, critically, for nineteen–year–olds, impatience was related to increased alcohol use and risk of alcohol–related harm due to hazardous drinking during the pandemic. This group also had the highest levels of impatience across all cohorts. Taken together, these findings raise a concern about the potential for adults who have poor impulse control to be at particular risk of an escalation of alcohol misuse following the pandemic situation.

It is clear from previous research that there is an interaction between stress and personality factors that influences drinking behaviour. For example, people who experience acute stress show increases in craving for, and consumption of, alcohol (Clay et al., 2018; Clay & Parker, 2018). Here, counterintuitively, we found that greater impatience and decreased stress was associated with increased alcohol use among thirty–year–olds and an increased hazardous drinking among both thirty–year–olds and fifty–year–olds. Similarly, among nineteen– and thirty–year–olds, those that rated themselves as more impatient and experienced increased stress were less likely to report increased alcohol consumption. As 'drinking to cope' was a prominent feature related to alcohol use during lockdown in the USA (Rodriguez et al., 2020), it may also be the case here. For instance, individuals with poor impulse control tend to use alcohol as a method of dealing with stress (Fede et al., 2020; Hamilton et al., 2013). Therefore, these individuals may have reduced stress levels due to their reported increased alcohol use. Alternatively, as the physiological response to long–term (chronic) and short–term (acute) stress differs (Stephens & Wand, 2012), it may be that the interaction between impulse control and chronic stress also differs. Therefore, future research should endeavour to investigate the impact of the interaction between different types of stress and impulse control in the context of alcohol use.

3.4.1. Limitations

We acknowledge several limitations in our study. First, the survey was designed to capture information across several domains other than those relevant here. Therefore, to mitigate known issues related to respondent burden (e.g., satisficing), brevity was prioritised, which inevitably resulted in less detail than

may be ideal in some of the measures used. For instance, single–item measures were used to assess risk–taking, impatience, and stress which may fail adequately to capture the full scope of these constructs (i.e., these measures may suffer from reduced content validity). This increases the uncertainty surrounding estimates calculated using these measures. Therefore, the use of single–item measures may also inflate standard errors and risk for type II error. Some of this potential error is offset by our large sample size; however, we found some effects that were not statistically significant despite relatively large effect sizes (e.g., among thirty–year–olds that reported increased stress, OR = 2.21, 95% CI 0.99 to 4.94). Second, there may be individual differences in the way each question was interpreted. For instance, feelings of stress are subjective and vary between–individuals (Sommerfeldt et al., 2019). Therefore, while some may find the pandemic and related period of social isolation as extremely stressful, others will find lockdown less stressful than pre–pandemic life. This may offer another explanation for why some that reported poor impulse control and lower levels of stress also reported increased alcohol use. Third, there is no way to independently verify self–report drinking; it is well–known that people under–estimate their alcohol consumption when asked on questionnaires due to social desirability bias, and often a lack of detailed memory of drinking episodes (Northcote & Livingston, 2011). It may, therefore, be that our data under–represent the true extent of drinking during the pandemic. Forth, as stress and alcohol use prior the pandemic were measured retrospectively, at the time of the survey, a 'true' baseline was not established. Thus, precluding the ability to infer causal relationships. Fifth, we realise that it is difficult to accurately assess determinants of change and these considerations informed our analysis. Therefore, we purposefully tried to avoid spurious findings by not adjusting for baseline measures in our models (Glymour et al., 2005). Finally, while the RRs were relatively low, as in comparable national COVID–19 web surveys (Institute for Social and Economic Research, 2020), the longitudinal nature of birth cohort data allows for attrition–related bias to be minimised using sample weights calculated by the CLS team (Brown et al., 2020). However, there is a possibility that unobserved predictors of missing data may still influence results.

3.4.2. Conclusion

In conclusion, we aimed to explore factors that influenced changes in alcohol use behaviour during the first COVID–19 lockdown in the UK, particularly concentrating on self–report stress and personality characteristics (risk–taking and impatience). We found that although most respondents drank either the same amount or less than prior to the pandemic, a significant minority, particularly of thirty– and fifty–year olds, drank more; often in amounts which could be classified hazardous, thus increasing their risk of potential alcohol–related harm. We also found that increases in drinking hazardously were predicted by personality (risk–taking, impatience) and environment (stress), although this was age specific. When considered in combination with recent data on alcohol–related deaths in the UK during the first three quarters of 2020, our findings suggest that hazardous drinking in a minority was strongly influenced by the pandemic and propose that this may be influenced by a combination of stress and personality factors, but also likely due to the availability of alcohol and inaccessible mental health services. We suggest that in future lockdowns, the government and public health officials pay particular attention to at–risk individuals, in terms of service provision, and consider critically the "essential" nature of off–premises alcohol sales.

Chapter 4. Drinking During Social Isolation: The ALCOVID–19 Project

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Chapter Foreword

The data analysed in the last chapter were collected as part of a large–scale survey administered to several cohorts. That survey was designed to capture information across several domains, other than those central to this thesis, by the Centre for Longitudinal Studies at University College London. Therefore, single–item measures of stress and impulsivity were used to (for example) reduce respondent burden. However, such measures may fail to adequately capture the full scope of these constructs. Therefore, in this chapter, this limitation was overcome by collecting primary survey data and by utilising commonly used, standardised, and psychometrically valid procedures across two studies.

In Study 1, results from a cross–sectional analysis are reported. Specifically, changes to alcohol use during the first wave of the COVID–19 pandemic (7 April–3 May 2020) – and whether impulsivity, negative affect (i.e., stress and boredom), or drinking motives were associated with these changes – was investigated. These results are published as an original research article in *Addiction Research & Theory* (Clay et al., 2022). The format of the original article has been modified to match the other chapters in this thesis. However, the content largely remains the same to that which was published.

In Study 2, a subset of respondents was followed up until September 2020. Therefore, the same relationships from Study 1 were investigated on a longitudinal basis, albeit with a smaller sample size. Furthermore, we also investigated the effectiveness of a personal feedback intervention (PFI) in Study 2. As previous research has shown that PFIs are an inexpensive and effective way to reduce alcohol use, it was hoped that they would also be useful for vulnerable people during periods of social isolation (e.g., lockdowns) as well. This also offered the opportunity to test the overarching hypothesis about stress and impulsivity interactions in a "real world" longitudinal context: it was predicted that impulsivity and stress would interact to predict aspects of alcohol misuse.

In both studies, incomplete and missing data was an issue due to respondents failing to answer all questions and attrition (i.e., participants dropping out of the study). Research has shown that using default techniques, such as listwise deletion, can result in biased parameter estimates in some instances, particularly when analysing cross–sectional data. Therefore, I had to learn how to deal with missing data appropriately using by, for example, using multiple imputation. Towards this end, I attended a

"hackathon" session at the annual virtual meeting of the Society for Improving Psychological Science in 2021. Subsequently, guidelines for addressing missing data were crowdsourced among attendees (see https://psyarxiv.com/mdw5r/) and we published a paper titled *"Best practices for addressing missing data through multiple imputation"* (Woods et al., 2023).

Overall, this chapter contributes to the aims of the thesis by testing the pre–registered hypotheses that poor impulse control, stress, and boredom would be positively associated with an increase in alcohol use during COVID–19 pandemic, and that the relationship between poor impulse control and alcohol use would be greater among those with higher stress and boredom.

Abstract

Impulsivity, negative affect (e.g., stress and boredom), and drinking motives are important moderators of alcohol–use behaviour. The recent COVID–19 pandemic resulted in an unprecedented period of social isolation (i.e., lockdowns), which caused increased chronic stress among some individuals. Therefore, in Study 1, analyses were carried out on questionnaire data from 337 individuals (243 female), aged 18–82 (*M* = 34.69, *SD* = 12.86), collected during the first wave of the COVID–19 pandemic (07 April – 03 May 2020), to assess changes in drinking behaviour, stress, and boredom. Drinking behaviour was then regressed on drinking motives, inhibitory control, stress, and boredom. Finally, interactions between negative affect and impulse control were also investigated. In Study 2, a subset of individuals (*N* = 60; 48 female), aged 18–62 (*M* = 31.40, *SD* = 10.40), were followed until September 2020 and the same questions were investigated on a longitudinal basis. In addition, the effectiveness of a personal feedback intervention (PFI) was also assessed. A minority of respondents reported increased alcohol use (units = 23.52%, drinking days = 20.73%, heavy days = 7.06%), alcohol-related problems (9.67%), and stress (36.63%). Meanwhile, most respondents reported increased boredom (67.42%). Those at–risk of increasing their alcohol use behaviour during this period included those who drink to cope with negative affect and those who were more impulsive (in terms of risk–taking, lack of premeditation, and sensation seeking). The PFI was successful in reducing the number of alcohol–related problems experienced by the participants. Therefore, such at risk individuals may benefit from similar interventions while they await more extensive treatment. Finally, the relationships between impulse control and alcohol were stronger among those who reported less pandemic–related boredom. Therefore, further research in understanding the complex interplay between impulsivity and boredom is required. Overall, these data provide a nuanced overview of changes in drinking-related behaviour during the COVID–19–induced period of social isolation and provide a potential solution (i.e., PFI) for reducing future alcohol–related harm.

Ethnics Approval Reference: SFEC 2020–030 (Appendix C)

4.1. Introduction

Increased mortality and morbidity have been linked to social isolation (e.g., loneliness) for decades (e.g., House et al., 1988). A large volume of theoretical and empirical work states that this effect ultimately results from increased activation of the HPA axis (Cacioppo et al., 2015). Chronic HPA axis activation results in dysfunctional stress responses and deficits in emotional regulation (Milivojevic & Sinha, 2018). In turn, these neuroadaptations contribute to the development and maintenance of addiction and offer an explanation as to why stress is a prominent risk factor for alcohol misuse (e.g., Jose et al., 2000; Ruisoto & Contador, 2019).

Impulsivity (i.e., impulse control) is a multifaceted construct (Strickland & Johnson, 2020) that has been established as a risk factor for alcohol misuse (e.g., Dalley & Ersche, 2019; Lee et al., 2019). Evidence for this is provided by pre–clinical experimental work (e.g., Belin et al., 2008; Kreek et al., 2005), neuroimaging studies (e.g., Bosker et al., 2017; Voon et al., 2020), and heritability studies (e.g., Karlsson Linnér et al., 2021). However, relatively little has been completed in the way of understanding the contextual conditions under which this effect may differ. However, recent work has shown that in times of acute stress, those who have lower impulse control tend to crave and consume more alcohol (Clay et al., 2018; Clay & Parker, 2018).

Boredom (i.e., the inability to find satisfaction or interest while participating in an activity) is consistently related to negative affect (Raffaelli et al., 2018). It has also been associated with addictive behaviours such as gambling (Eastwood & Mercer, 2010) and alcohol misuse (Biolcati et al., 2018). Those with reduced impulse control tend to have greater boredom proneness (Isacescu et al., 2017; Struk et al., 2016). Therefore, poor impulse control may moderate the relationship between boredom and alcohol use, whereby the impact of boredom on alcohol use is greater among those with poor impulse control.

Other well–researched moderators of drinking behaviour exist: so–called drinking motives (Cooper, 1994). Several general patterns emerge when examining the impact of drinking motives on alcohol use: social motives (i.e., drinking to improve social situations) tend to be related to drinking frequency; enhancement motives (i.e., drinking to increase positive affect) are related to heavy drinking;

coping motives (i.e., drinking to reduce negative affect) are associated with a greater number of alcohol–related problems; and conformity motives (i.e., drinking to fit in with a group) are typically negatively associated with frequency and quantity of alcohol use (Kuntsche et al., 2005, 2014; Lyvers et al., 2010). Drinking motives have also been shown to impact alcohol use following crisis. For example, after the 9/11 terrorist attack, Beseler et al. (2011) found that both drinking to cope and drinking for enjoyment (i.e., enhancement) were associated with increased alcohol use. Similarly, 'drinking to cope' has been highlighted as a prominent risk factor for increased alcohol use during the COVID–19 pandemic in the USA (Rodriguez et al., 2020) and Canada (Wardell et al., 2020).

The COVID–19 pandemic and associated 'lockdowns' (i.e., government mandated periods of social isolation characterised by orders to remain at home to mitigate the spread of disease; Anderson et al., 2020) have resulted in increased mental distress worldwide through (for example) social isolation, loss of income, increased childcare responsibilities, and monotony (Bhattacharjee & Acharya, 2020; Gavin et al., 2020; Ornell et al., 2020; Pfefferbaum & North, 2020). Thus, the pandemic presents a naturalistic source of chronic stress / negative affect.

Early in the pandemic, several scholars warned that long–term isolation may create an unforeseen public health crisis involving increased alcohol consumption (Clay & Parker, 2020; Finlay & Gilmore, 2020; Ramalho, 2020). As a result, attempts were made to synthesise work conducted in–relation to other crises involving trauma (e.g., the 9/11 attack), epidemic outbreaks (e.g., the 2002–03 SARS pandemic), and economic hardship (e.g., the 2008 recession) in relation to alcohol use (Gonçalves et al., 2020). Ultimately, two opposing scenarios were proposed (Rehm et al., 2020): (1) increased psychological distress may drive an increase in alcohol use and related harms; (2) alcohol policies which reduce the physical and financial availability of alcohol would cause a reduction in alcohol consumption and associated problems.

Following these predictions, recent work has tried to characterise those most at–risk of increased alcohol consumption, although this literature offers a somewhat mixed picture. Several studies provide evidence that increased distress was associated with increased drinking (Garnett et al., 2021; Jacob et al., 2021; Koopmann et al., 2020; Neill et al., 2020; Tran et al., 2020). Conversely, in a

large–scale study comprising data from 21 European countries, Kilian et al. (2021) found evidence that drinking decreased in most countries, and that this reduction was primarily driven by reduced availability of alcohol. Nevertheless, increased distress dampened this relationship. Additionally, recent work has shown that impulsivity acts as a moderator of stress–related pandemic drinking (Clay et al., 2021). However, that paper reports a secondary analyses of birth cohort data, and such surveys prioritise brevity and breadth. Thus, single-item measures of impulse control were utilised, which were not empirically validated and may suffer from reduced content validity.

Taken together, previous research provides strong evidence for the prediction that those who increased their drinking during the pandemic were drinking to cope, which may be moderated by impulsivity, and limited evidence that a reduction in affordability or availability played a role. Therefore, our work here was motivated by the need to evaluate risk factors for those who increased their drinking during the pandemic; whether they were drinking to cope and whether this relationship, if present, was moderated by impulsivity (using empirically validated measures).

As we move out of the pandemic, this work is of importance as it pertains to drinking in the home (vs. in public settings). For instance, prior to the pandemic, a significant proportion of alcohol was consumed at home (perhaps due to convenience, cost, safety, autonomy, and stress relief) (e.g., Callinan et al., 2016; Foster & Ferguson, 2012). Moreover, most long–term harms that occur because of alcohol use (e.g., liver disease and cancer) are linked to total alcohol consumption (Griswold et al., 2018). However, research typically focuses on public drinking (Callinan & MacLean, 2020). Thus, if a large amount of alcohol is typically consumed in the home going forward, further research which focuses on drinking in this setting is crucial in reducing the burden of alcohol, and data collected during the COVID–19 pandemic provides the perfect opportunity (Callinan & MacLean, 2020).

The potential for an alcohol–related public health crisis during and after the COVID–19 pandemic (Clay & Parker, 2020) has been heightened by an inability of those in need being able to access psychiatric service provision during lockdown (e.g., Columb et al., 2020; Da et al., 2020). However, in a review of meta–analyses, eHealth (e.g., web–based brief interventions) interventions, which can be implemented cheaply and remotely, have been shown to improve access to psychiatric

service and to be effective in improving anxiety, depression and alcohol–related problems (Bennett et al., 2020). Some of the most successful attempts at creating brief interventions to address heavy drinking have utilised a personal feedback intervention (PFI; e.g., Carey et al., 2007; Cronce & Larimer, 2011; Lewis et al., 2019; Patrick et al., 2014). Furthermore, research has shown that web–based / eHealth PFIs are effective at reducing drinking (Bewick et al., 2008; Doumas et al., 2009; Lewis et al., 2019). Therefore, web–based PFIs may reduce alcohol use during the COVID–19 pandemic. In brief, PFI typically involves providing a summary of self–report drinking behaviours and consequences, normative comparisons, and information about strategies to reduce drinking (Lewis et al., 2019).

Overall, the aim of the present study was to investigate how some of the theoretical mechanisms that underlie alcohol use may have operated during a period of social isolation brought on by the COVID–19 pandemic (Study 1 and Study 2). We also investigated the effectiveness of a PFI in reducing alcohol use and misuse among our sample (Study 2). We hope that this increased theoretical understanding of socially isolated home drinking, will have broader implications beyond the pandemic by, for instance, identifying those most at–risk of future alcohol–related long–term harm. Furthermore, the PFI, if effective, could be used during future lockdowns.

4.2. Study 1

Study 1 aimed to investigate how alcohol use changed during the first wave of the COVID–19 pandemic and whether drinking motives, impulsivity, or negative affect were associated with these changes. We pre–registered several hypotheses[12]: (1) alcohol use would increase during social isolation; (2) both coping and enhancement motives would be associated with increased alcohol use; (3) poor impulse control, stress, and boredom would be positively associated with an increase in alcohol use; and (4) the association between poor impulse control and alcohol use would be greater among those with higher negative affect (stress and boredom).

[12] The original preregistration listed ten hypotheses. Data testing hypotheses one to seven and hypothesis nine are reported in Study 1. These have been briefly summarised here. As there was no significant association between a change in stress and perceived stress reactivity (see Appendix C), our planned moderation analysis, detailed in hypothesis eight of the preregistration, was not conducted. As this was a two–part project, hypothesis ten was tested in Study 2.

4.2.1. Method

Design

This study used an online cross–sectional design. The dependent variable was change in alcohol use (the number of units consumed[13], the number of drinking days, the number of heavy drinking days[14], and the number of alcohol–related problems per week). The main independent variables were drinking motives, and pre– vs. intra– pandemic changes in stress and boredom. The moderator variable was impulse control. Several covariates controlled for age, gender, ethnicity, SES, the number of COVID–19 symptoms experienced, and whether the participant was isolated with children. Models including stress as a predictor also controlled for perceived stress reactivity.

Transparency and openness

We report how we determined our sample size, all data exclusions, all manipulations, and all measures in the study. Our final sample size was based on resource constraints (Lakens, 2022). In other words, due to limited financial and temporal resources, we used opportunity/snowball sampling to collect data from as many participants as possible within the study period. A simulation–based sensitivity power analysis (Lakens, 2022) showed that with $N = 337$ and $\alpha = 0.05$, our design had sufficient statistical power $(1 - \beta) = 80\%$ to detect an effect size of $B = 0.0015$ for our most complex model. Details of the sensitivity power analysis can be seen in the Appendix C. Data, preregistered hypotheses, materials, and code are posted on the Open Science Framework at https://osf.io/mnz34/. Data were analysed using Stata IC (version 16.1) and R (version 4.0.4) for Windows.

Recruitment

A survey designed to assess changes in, and factors related to, drinking behaviour during social isolation was created using Qualtrics (Provo, Utah). The survey was developed in English, and then translated into French, Spanish, Italian, Portuguese (European and Brazilian), and Hebrew by the native speaking authors. Some wording had to be changed slightly to retain the original meaning and to ensure consistency across countries. Participants were eligible if they were ≥ 18 years of age, had a reliable

[13] 1 unit = 8g of pure ethanol.
[14] 1 heavy day = > 8 units per day for men and > 6 units per day for women.

internet connection, and they were proficient in at least one of the languages listed above. Participants could complete the survey on either a computer, smartphone, or tablet. All responses were completed between 07 April 2020 and 03 May 2020. During this time, the survey was advertised by several news media outlets and throughout the co–authors' networks via email, word–of–mouth, and social media. All participants gave their informed consent and were not compensated.

Demographic information

Demographic data collected were age, gender, ethnicity, country of residence, education level, occupation, whether the respondent was a key worker, gross individual income over the last 12 months, subjective social status, marital status, the number of people in the same household as the respondent, number of offspring, who the respondent was isolated with, and whether the respondent was suffering from any COVID–19 associated symptoms. Country of residence was recoded to reflect sub–regions of the world based on the United Nations M49 Standard (United Nations, 2020). This allowed us to find a balance between the number of levels and the number of participants within each level (Hox et al., 2018). The gross individual income question was presented in local currency relative to British Pounds and then recoded to relative income using World Bank adjusted net national income per capita data (The World Bank, 2020), where:

$$\text{Relative Income} = \frac{\text{Income}}{\text{Income per Capita}} \tag{3}$$

An index of SES, combining relative income, education, occupation, and subjective social status (Diemer et al., 2013), was calculated using exploratory factor analysis (EFA) – see Appendix C. This allowed us to conserve statistical power during hypothesis testing by controlling for the variables entered into the final EFA using a single model parameter. Similar approaches to creating an index of SES have been published elsewhere (e.g., Scharoun-Lee et al., 2009; Yu et al., 2014).

Alcohol use and drinking behaviour

The AUDIT (see Chapter 2 for details) was used to assess prior alcohol use behaviour and alcohol dependency. Internal consistency of the AUDIT in the present study was good, Cronbach's $\alpha = 0.78$.

The Typical Atypical Drinking Diary (TADD) was used was used to retrospectively assess alcohol use (Patterson et al., 2019). When completing the TADD, participants fill in two weekly diaries: one for typical weeks and another for atypical weeks (i.e., either less than or greater than a typical week). Participants specified the type, strength, volume, and quantity of the beverages they consumed for each day of the seven–day week and then estimated how many weeks they drank this typical / atypical amount during the specified period. Participants were asked to estimate what they drank before (i.e., *"before the COVID–19 induced isolation")* and during (i.e., *"after the COVID–19 induced isolation"*) social isolation. This method allows for the calculation of units, drinking days, and heavy drinking days per week. Research indicates that the TADD is more accurate and time–efficient than other retrospective assessments of drinking, such as the Timeline Followback (Patterson et al., 2019).

Alcohol–related problems were assessed using the Alcohol Problems Questionnaire (APQ; Drummond, 1990). The APQ is a standalone scale that consists of 44 binary (yes/no) items designed to assess alcohol–related problems across four domains: commonly faced alcohol–related problems, problems related to romantic relationships, problems related to children, and problems related to work. Therefore, the maximum score on the APQ is 44, with a higher score reflecting a greater number of alcohol–related problems faced. Here, we added a *"Not Applicable"* option to the latter subscales to allow the questionnaire to be relevant to a larger proportion of the population than the original scale. For instance, an 18–year–old student may not have any children. We also changed the wording for questions about romantic relationships from *"spouse"* to *"spouse / partner"* for the same reason. The APQ has been shown to have good validity and test–retest reliability (Williams & Drummond, 1994). In the present study, the internal consistency was excellent, Cronbach's $\alpha = 0.94$.

Drinking motives

Drinking motives were assessed using the Revised Drinking Motives Questionnaire (DMQ–R; Cooper, 1994) The DMQ–R is a 20–item scale which proposes four motives for alcohol consumption:

conformity (e.g., *"so you won't feel left out"*); coping (e.g., *"drinking to forget your problems"*); enhancement (e.g., *"to have fun"*); and social (e.g., *"because it helps you enjoy a party"*). Here, participants responded to each item using a five–point Likert scale (1 = *Almost never/never*, 2 = *Some of the time*, 3 = *Half of the time*, 4 = *Most of the time*, 5 = *Almost always/always*). Each subscale contains five items. Thus, the maximum score per subscale is 25, with higher scores indicating greater endorsement of a motive. The DMQ–R has been shown to have good validity across cultures and in a variety of age groups (Fernandes-Jesus et al., 2016). Here, the internal consistency of the DMQ–R subscales ranged from acceptable to excellent, Cronbach's αs = 0.68 to 0.89.

Negative affect

Self–report stress levels were measured before (i.e., *"before the COVID–19 related isolation"*) and during (i.e., *"since the COVID–19 related isolation")* social isolation using the Short Stress Overload Scale (SOS–S; Amirkhan, 2018). The SOS–S is a ten–item scale designed to act as a brief diagnostic tool for stress and stress–related disorders and has been shown to have good psychometric properties. Here, participants responded to each item using a five–point Likert scale (1 = Not at all: 5 = A lot). Therefore, the maximum score on the SOS–S is 50, with higher scores reflecting greater levels of stress. In the present study, internal consistency was excellent, Cronbach's αs = 0.90 to 0.92.

Stress reactivity was assessed using the 23–item Perceived Stress Reactivity Scale (PSRS; Schlotz et al., 2011). The PSRS is a standalone scale with five subscales: prolonged reactivity, reactivity to work overload, reactivity to social conflict, reactivity to failure, and reactivity to social evaluation. Participants responded to each item using a three–point Likert–type scale that varied depending on the framing of each item (e.g., *"When tasks and duties build up to the extent that they are hard to manage..."*, 0 = *"...I am generally untroubled"*, 1 = *"...I usually feel a little uneasy"*, 2 = *"...I normally get quite nervous"*). Therefore, the maximum total score on the PSRS is 46, with higher scores indicating greater levels of stress reactivity. The psychometric properties of the PSRS have been established in several countries, with scores correlating with numerous stress related disorders (Schlotz et al., 2011). In the present study, the internal consistency was good, Cronbach's α = 0.88.

Boredom before and during social isolation was assessed using the Multidimensional State Boredom Scale (MSBS; Fahlman et al., 2013). The MSBS is a 29–item scale with good psychometric properties that can be used to quantify boredom by either using the total score or across five subscales: disengagement, high arousal, low arousal, inattention, and time perception. Here, participants responded to each statement using a seven–point Likert scale (1 = *Strongly disagree*, 2 = *Disagree*, 3 = *Somewhat disagree*, 4 = *Neutral*, 5 = *Somewhat agree*, 6 = *Agree*, 7 = *Strongly agree*). Thus, the maximum score was 203, where higher scores reflect greater levels of boredom. The internal consistency here was excellent with Cronbach's α ranging from 0.96 to 0.97.

Impulse control

The SUPPS–P (see Chapter 2 for details) was used to assess negative urgency, lack of premeditation, lack of perseverance, sensation seeking, and positive urgency. In the present study, internal consistency of each subscale ranged from acceptable to good, Cronbach's α = 0.67 to 0.82.

The Domain–Specific Risk–taking Scale (DOSPERT) was administered to assess risk–taking (Blais & Weber, 2006). The DOSPERT is a 30–item scale designed to assess five sub–domains risk–taking: ethical, financial, health, recreational, and social. Here, participants rate how likely it is that they would engage with each activity or behaviour using a seven–point Likert scale (1 = *Extremely unlikely*, 2 = *Moderately unlikely*, 3 = *Somewhat unlikely*, 4 = *Not sure*, 5 = *Somewhat likely*, 6 = *Moderately likely*, 7 = *Extremely likely*). Scores can be summed across all items or by subscale. Each subscale contains six–items. Therefore, the maximum score overall is 210, with higher scores indicating a greater propensity to take risks. The DOSPERT has been shown to be a reliable and valuable assessment of 'real world' risk–taking via questionnaire (e.g., Highhouse et al., 2017). Here, the internal consistency of the DOSPERT was good, Cronbach's α = 0.82.

Procedure

After informed consent was confirmed, participants reported their demographic information before completing the remaining scales in counterbalanced order to eliminate order effects. Scales that measured both pre– and intra–isolation data (e.g., the TADD) were presented as one block, whereby the scale which sought pre–isolation responses were presented first.

Sample

Overall, 1,148 responses were recorded. Of these, 811 were excluded to ensure data integrity: 39.55% had > 40% missing data[15]; 21.43% reported living in sub–regions with an inadequate number of responses[16]; 7.40% were classified as multivariate outliers based upon a Mahalanobis distance that is significant at $p < .001$ (Tabachnick & Fidell, 2014; Verardi & Dehon, 2010) and 0.17% were considered clear univariate outliers (see Figure C2); 0.87% reported experiencing no social isolation; 0.52% were test data; 0.44% had gender recorded as transgender or 'prefer not to say' and 0.09% had ethnicity recorded as 'prefer not to say'[17]; and 0.17% were duplicate responses. This left 337 cases for analysis. Sociodemographic characteristics of the sample are shown in Table 4.1.

Analysis

Missing data was dealt with using multiple imputation (MI; Enders, 2010; Woods et al., 2023). White et al. (2011, p. 388) recommended *that "m should be at least equal to the percentage of incomplete cases"*. Here, the overall percentage of cases with incomplete data on analysis variables was 37.69%. Therefore, we used the *mi impute chained* command in Stata to generate 40 imputed datasets, using predictive mean matching, with **d** = 5 (Schenker & Taylor, 1996). Graphical diagnostics (see Figure C4) suggested that the datasets should be separated by at least 125 iterations of the imputation algorithm, thus we conservatively saved each dataset after the 150th iteration. The imputation model included all variables used in subsequent analyses together with the hypothesised interaction terms and three auxiliary variables that were believed to be correlated with missingness (percent progress in survey, date of response, AUDIT score). Interaction terms were imputed and estimated following Enders (2014).

Change scores were calculated for units, drinking days, heavy drinking days, alcohol–related problems, stress, and boredom, using the mi passive command. Descriptive statistics were calculated for each of the key study variables. Bivariate relationships were explored using Pearson correlations.

[15] Royston (2004) recommends that caution should be taken when implementing multiple imputation when the proportion of missing data exceeds 50%. Therefore, acting conservatively, we used 40% as our cut off.
[16] When utilising multilevel analyses, the minimum sample size at each level of a random effect (e.g., a sub-region) should be ≥ 10 (Hox et al., 2018).
[17] Analysis of such low numbers of participants would lead to low power and unstable parameter estimates.

Linear mixed–effects models (LMMs) were used to test our hypotheses. We included sub–region as a random effect to improve inference and generalisability (Barr et al., 2013) We first assessed change in alcohol use, stress, and boredom by entering change scores and covariates into models as fixed effects and interpreting the intercept (analogous to a one sample t–test comparing the change score to zero). Next, we regressed change in alcohol use scores on our predictors of interest and covariates. Finally, we entered our hypothesised interactions into the models.

All continuous predictor variables were grand mean centred to aid interpretation and reduce potential collinearity. Models were separated by construct to conserve statistical power and to avoid erroneously conditioning the model estimates (McMullin et al., 2020). We implemented Benjamini and Hochberg's (1995) method of false discovery rate (FDR) control for pre–registered confirmatory analyses to reduce the probability of making a type I error due to multiple testing (Glickman et al., 2014). Significant interactions were probed using the Johnson–Neyman (JN) technique (Johnson & Neyman, 1936) as suggested by (Hayes, 2022).

Covariates included in all models were: age (e.g., Leigh & Stacy, 2004) gender (e.g., A. White et al., 2015), ethnicity (e.g., Twigg & Moon, 2013), SES (e.g., Probst et al., 2020), the number of COVID–19 symptoms experienced (e.g., Chaaban et al., 2021), and whether the participant was isolated with children (e.g., MacMillan et al., 2021). Models including stress as a predictor also controlled for perceived stress reactivity (e.g., Clay & Parker, 2018). As the sample lacked ethnic diversity, a dichotomous White/non–White variable was used. As the margins command is incompatible with imputed data, the first complete dataset was used to probe and visualise significant interactions. For brevity, non–significant LMM results are reported in the Appendix C. Results were considered significant when $p < 0.05$ or when the 95% CI did not contain zero.

Table 4.1 Sociodemographic characteristics of the sample.

Variable	Total (SD)	Female (SD)	Male (SD)
N	337	243	94
Age	34.69 (12.84)	32.96 (11.70)	39.18 (14.53)
Ethnicity			
White	95.25%	94.24%	97.87%
Black	0.30%	0.41%	0.00%
Asian	2.08%	2.06%	2.13%
Mixed	2.08%	2.88%	0.00%
Other	0.30%	0.41%	0.00%
Sub–region			
N. Europe	39.17%	41.56%	32.98%
E. Europe	5.04%	4.12%	7.45%
S. Europe	21.07%	19.34%	25.53%
W. Europe	13.06%	13.58%	11.70%
N. America	15.73%	16.46%	13.83%
Oceania	5.93%	4.94%	8.51%
Education			
GCSE/GED	6.23%	5.76%	7.45%
A–levels/High School Diploma	18.69%	16.87%	23.40%
Undergraduate Degree	22.85%	22.63%	23.40%
Graduate Degree	31.45%	32.51%	28.72%
Doctoral Degree or Higher	20.77%	22.22%	17.02%
Occupation			
Full–time students	23.44%	25.51%	18.09%
Never worked / long–term unemployment	13.35%	9.88%	22.34%
Consultant	2.37%	2.06%	3.19%
Skilled labourer	4.15%	2.88%	7.45%
Trained professional	22.85%	23.87%	20.21%
Support staff	4.45%	4.53%	4.26%
Administrative staff	6.53%	8.64%	1.06%
Junior management	10.98%	11.11%	10.64%
Middle management	8.01%	8.64%	6.38%

Upper management	3.86%	2.88%	6.38%
Key Worker = Yes	21.07%	17.70%	29.79%
Income			
Under £2,500	14.24%	15.23%	11.70%
£2,500 to £4,999	4.45%	4.94%	3.19%
£5,000 to £9,999	7.42%	5.76%	11.70%
£10,000 to £14,999	10.68%	11.93%	7.45%
£15,000 to £19,999	10.09%	11.11%	7.45%
£20,000 to £24,999	7.72%	9.05%	4.26%
£25,000 to £29,999	8.31%	8.23%	8.51%
£30,000 to £34,999	6.82%	8.64%	2.13%
£35,000 to £39,999	4.15%	3.29%	6.38%
£40,000 to £44,999	4.75%	4.94%	4.26%
£45,000 to £49,999	3.26%	2.88%	4.26%
£50,000 or more	18.10%	13.99%	28.72%
Subjective Social Status			
Working Class	15.13%	16.05%	12.77%
Lower–middle Class	39.76%	40.33%	38.30%
Upper–middle class	43.03%	41.15%	47.87%
Upper Class	2.08%	2.47%	1.06%
Marital Status			
Single/Separated/Widowed/Divorced	56.08%	60.91%	43.62%
Married/Domestic Partnership	43.92%	39.09%	56.38%
Experienced COVID–19 Symptoms	15.43%	14.40%	18.09%
No. People in Same Household	2.65 (1.18)	2.69 (1.21)	2.55 (1.08)
No. Offspring	0.55 (0.99)	0.46 (0.94)	0.78 (1.08)
Isolated With [a]			
Alone	11.57%	10.70%	13.83%
With children	67.56%	67.36%	68.09%
With romantic partner	62.50%	61.98%	63.83%
With parents	18.15%	20.66%	11.70%
With siblings	2.98%	4.13%	0.00%
With housemates	8.33%	7.85%	9.57%

With friends	1.19%	1.65%	0.00%
With extended family	20.83%	16.94%	30.85%

Note. Data are presented as mean (SD) for continuous measures and % for categorical measures. Symptoms included: (1) a high temperature, (2) a new, continuous cough, (3) a continuous headache, (4) a loss of taste and/or smell, (5) muscle aches, (6) a sore throat. Countries in the sample included: Australia (*n* = 17), Austria (*n* = 3), Bulgaria (*n* = 1), Canada (*n* = 3), Denmark (*n* = 2), Finland (*n* = 1), France (*n* = 18), Germany (*n* = 21), Hungary (*n* = 14), Ireland (*n* = 1), Italy (*n* = 63), Luxembourg (*n* = 1), New Zealand (*n* = 3), Portugal (*n* =4), Romania (*n* = 1), Russia (*n* = 1), Serbia (*n* = 1), Spain (*n* = 3), Switzerland (*n* = 1), United Kingdom (*n* = 128), United States (*n* = 50).

[a] *n* = 336

4.2.2. Results

Table 4.2 displays the descriptive statistics for the main study variables in terms of alcohol use and drinking behaviour, drinking motives, stress, boredom, and impulse control. See Table C2 for correlations between variables.

Changes in alcohol use, stress, and boredom

Figure 4.1 shows changes in alcohol use, stress, and boredom. A sizeable number of respondents reported increased alcohol use (units = 23.52%, drinking days = 20.73%, heavy days = 7.06%), alcohol–related problems (9.67%), and stress (36.63%). Meanwhile, the majority of respondents reported increased boredom (67.42%). Results from the unadjusted models, which tested whether change occurred on average, suggested that alcohol units (B = -1.53, FDR–adjusted p = .004) and alcohol–related problems (B = -1.47, FDR–adjusted p < .001) decreased. Meanwhile, boredom (B = 18.16, FDR–adjusted p < .001) increased. In the adjusted models, there was evidence to suggest that alcohol–related problems (B = -1.43 FDR–adjusted p < .001) decreased while boredom increased (B = 21.22, FDR–adjusted p < 001). No other significant changes were found.

Associations between drinking motives and alcohol use

Social motives were associated with a decrease in alcohol–related problems (B = -0.09, FDR–adjusted p = .005). No other significant relationships were found

Table 4.2 Descriptive statistics (*M* and *SD*) for main study variables (*N* = 337).

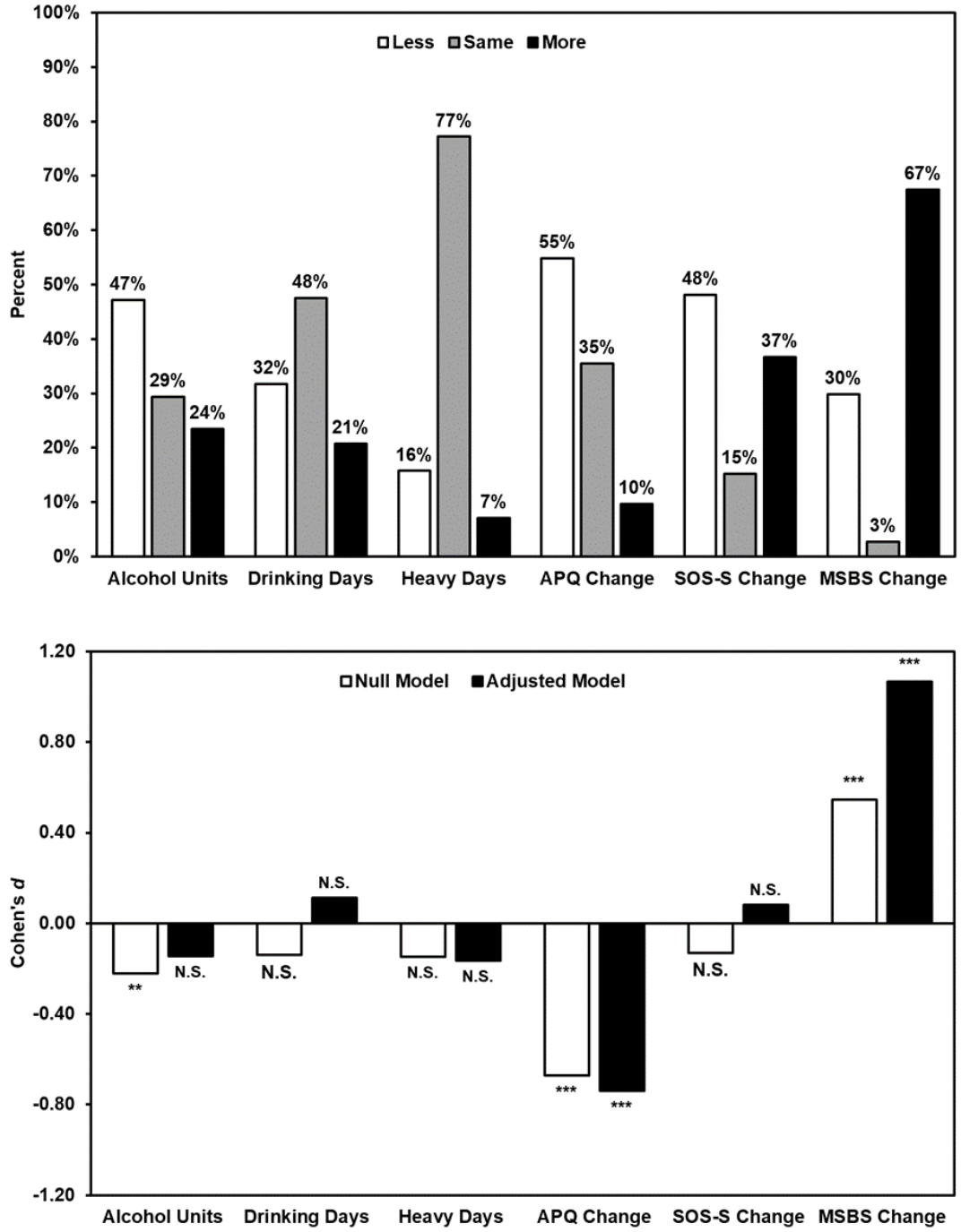
Variable	Total (SD)	Female (SD)	Male (SD)	% Miss.
AUDIT	6.52 (4.35)	6.10 (3.89)	7.62 (5.23)	12.76
Alcohol Units Change	-1.57 (6.89)	-0.87 (6.84)	-3.38 (6.72)	12.46
Drinking Days Change	-0.21 (1.50)	-0.05 (1.41)	-0.62 (1.63)	12.46
Heavy Days Change	-0.12 (0.61)	-0.13 (0.66)	-0.10 (0.45)	12.46
APQ Change	-1.39 (2.19)	-1.46 (2.21)	-1.21 (2.14)	8.61
DMQ–R Social	13.67 (5.25)	13.59 (5.23)	13.86 (5.31)	9.79
DMQ–R Coping	8.56 (3.63)	8.60 (3.59)	8.46 (3.75)	9.79
DMQ–R Enhancement	11.58 (4.86)	11.57 (5.00)	11.61 (4.49)	9.79
DMQ–R Conform	6.69 (2.16)	6.68 (2.12)	6.71 (2.26)	9.79
SOS–S Change	-0.83 (5.91)	-0.89 (6.35)	-0.67 (4.62)	11.28
PSRS Total	22.19 (7.98)	23.66 (7.74)	18.4 (7.33)	11.28
MSBS Change	18.51 (33.29)	19.71 (35.75)	15.41 (25.79)	9.79
SUPPS–P Negative Urgency	8.58 (2.59)	8.67 (2.59)	8.34 (2.59)	12.17
SUPPS–P Premeditation	6.80 (2.01)	6.83 (2.04)	6.72 (1.94)	12.17
SUPPS–P Perseverance	7.22 (1.95)	7.16 (1.93)	7.37 (1.98)	12.17
SUPPS–P Sensation Seeking	9.39 (2.76)	9.13 (2.68)	10.06 (2.87)	12.17
SUPPS–P Positive Urgency	6.76 (2.25)	6.70 (2.29)	6.91 (2.15)	12.17
DOSPERT Total	87.11 (20.14)	85.83 (19.28)	90.41 (21.95)	11.87

Note. Summary statistics calculated using imputed data (*m* = 40). 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; DMQ–R = Revised Drinking Motives Questionnaire; SOS–S = Short Stress Overload Scale; PSRS = Perceived Stress Reactivity Scale; MSBS = Multidimensional State Boredom Scale; SUPPS–P = The Shortened Urgency, Premeditation (lack of), Perseverance (lack of), Sensation Seeking, Positive Urgency, Impulsive Behaviour Scale; DOSPERT = Domain–Specific Risk–taking Scale.

Associations between impulse control, stress, boredom, and alcohol use

Risk–taking (DOSPERT score) was associated with a decrease in alcohol–related problems (B = -0.02, FDR–adjusted p = .008). No other significant associations were found.

Figure 4.1 Changes in alcohol use, alcohol-related problems, stress, and boredom during social isolation (N = 337).



Note. Both prevalence estimates (top) and effect sizes (bottom) were calculated using imputed data (m = 40). Adjusted models controlled for age, gender, ethnicity, socioeconomic status, the number of symptoms experienced, and whether the respondent was isolating with children. 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; SOS-S = Short Stress Overload Scale; MSBS = Multidimensional State Boredom Scale.

* FDR-adjusted p < 0.05, ** FDR-adjusted p < 0.01, *** FDR-adjusted p < 0.001

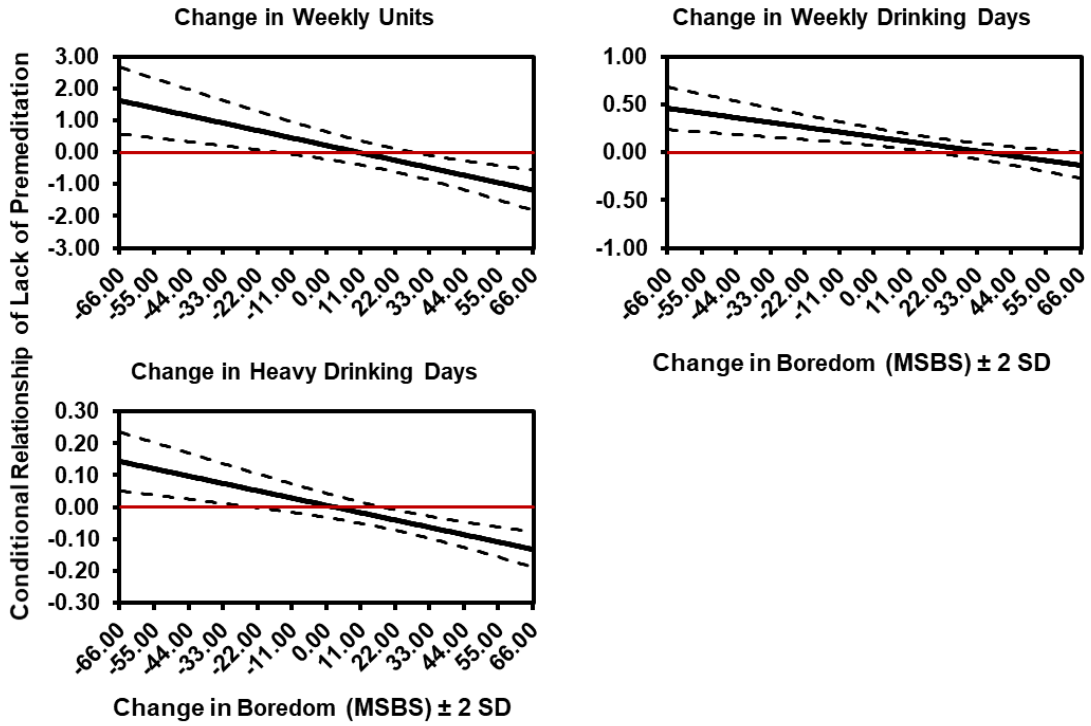
Moderation analyses suggested that boredom modified the relationship between lack of premeditation and the number of units consumed per week (B = -0.02, FDR–adjusted p = .034), the number of weekly drinking days (B = -0.004, FDR–adjusted p =.027), and the number of heavy drinking days (B = -0.002, FDR–adjusted p = .048). No other significant interactions were observed. JN plots (see Figure 4.2) revealed that those who were more impulsive and less bored tended to report increased alcohol use, and vice–versa. Specifically, a decrease of ≥ 16 MSBS points was associated with an increase in the number of units consumed. Whereas an increase of ≥ 28 points was associated with a decrease in the number of units consumed. Similarly, decreased MSBS scores were associated with an increased number of drinking days. Meanwhile, an increase of < 19 MSBS points was associated with a decrease in drinking days. Finally, a decrease of ≥ 16 MSBS points was associated with an increase in the number of heavy drinking days. Whereas an increase of ≥ 18 MSBS points was associated with an increase in the number of heavy drinking days.

4.2.3. Discussion

Study 1 aimed to better understand how a period of social isolation, brought about by the recent COVID-19 pandemic, affected alcohol use. By assessing associations between changes in drinking behaviour, drinking motives, impulse control, stress, and boredom, this data provides a nuanced overview of how some of the theoretical mechanisms which underlie alcohol use and misuse may have operated during this time.

We found that approximately one in four respondents reported drinking more and around one in ten reported experiencing an increased number of alcohol-related problems. Similarly, most respondents reported feeling more bored. However, stress levels either stayed the same or decreased for most and, despite our prediction, stress was not significant in any model. Our results also suggest that those who were high in risk-taking (DOSPERT total score) and social drinking motives tended to face fewer alcohol-related problems during social isolation. Moreover, boredom was found to be a critical

Figure 4.2 Johnson–Neyman plots illustrating significant impulse control x boredom interactions (N = 337).



Note. Models were fitted using imputed data (*m* = 40). Models were adjusted for age, gender, ethnicity, socioeconomic status, the number of symptoms experienced and whether the respondent was isolating with children. The first imputed dataset was used to visualise statistically significant interactions. 1 unit = 8g pure ethanol; 1 heavy drinking day = consuming > 8 units per day for men or > 6 units per day for women. Dashed lines represent the 95% CI.

moderator, whereby those who were less impulsive (in terms of lack of premeditation), who also reported feeling more bored, were more likely to increase alcohol use during the isolation and vice-versa. Therefore, during a period of social isolation, some theoretical mechanisms which underlie alcohol use and misuse may not hold. In study 2, we built on this to investigate the same questions but on a longitudinal basis and by also testing a potential solution for reducing future alcohol–related harm: a PFI.

4.3. Study 2

Study 2 investigated the same relationships as Study 1 but on a longitudinal basis among a subset of respondents (*N* = 60) who were followed up until September 2020. As web–based PFIs are relatively cheap to implement and have been shown to be an effective alcohol use intervention (e.g., Bewick et

al., 2008; Doumas et al., 2009; Lewis et al., 2019), the efficacy of a web–based PFI was also investigated in Study 2. Therefore, along with testing the same hypotheses from Study 1 (see section 4.2), we also tested the preregistered hypothesis that those receiving the PFI would report lower alcohol use.

4.3.1. Method

Design

This study used an online longitudinal design. Data from 60 participants across 14 timepoints (07 April 2020 to 08 September 2020) were analysed. The dependent variable was alcohol use (the number of units consumed, the number of drinking days, the number of heavy drinking days, and the number of alcohol–related problems per week). The main independent variable was whether the participant was randomly assigned to the PFI or control condition. Moderator variables included impulsivity, stress, boredom, and drinking motives. Several covariates controlled for age, gender, ethnicity, SES[18], the number of COVID–19 symptoms experienced, whether the participant was experiencing isolation at a given timepoint, and whether the participant was isolated with children.

Transparency and openness

We report how we determined our sample size, all data exclusions, all manipulations, and all measures in the study. Due to financial and temporal restrictions, we use opportunity / snowball sampling to collect data from as many participants as possible within the study period (Lakens, 2022). Preregistered hypotheses and materials are posted on the Open Science Framework at https://osf.io/mnz34/. Data and code will be posted on the Open Science Framework at a later date following publication. Data were analysed using Stata IC (version 16.1) and R (version 4.0.4) for Windows.

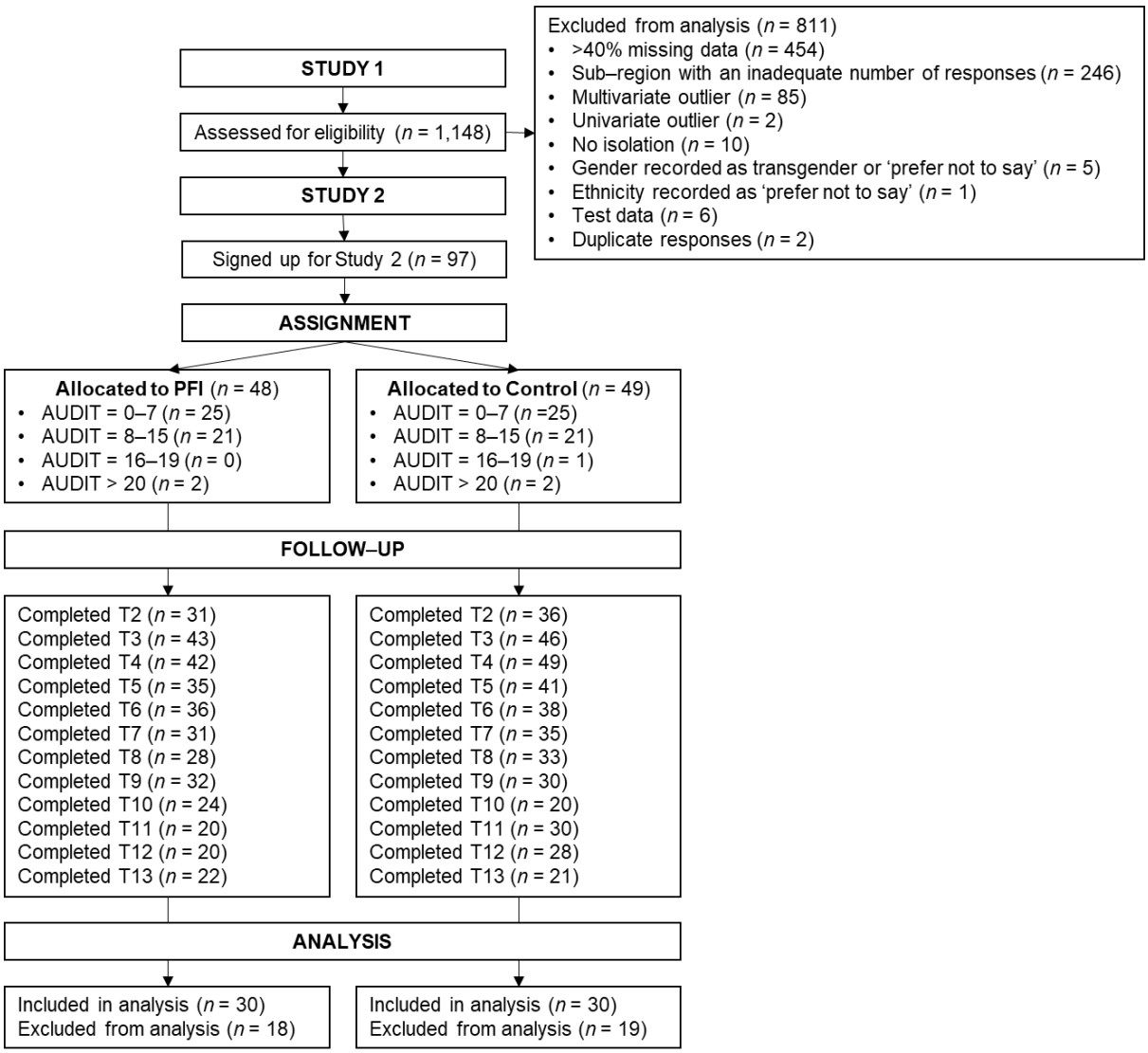
Participants and procedure

Participant flow throughout the study is presented in Figure 4.3. At the end of Study 1, participants were asked whether they would like to participate in Study 2 using the following question: *"Thank you for completing our survey up to this point. We are interested in tracking your alcohol use throughout the*

[18] Study 1 describes how the SES index was created.

isolation period. To do so, we will send you links to new surveys each week. These will take approximately 10-15 minutes to complete. Are you happy to continue participating in stage 2?"

Figure 4.3 Participant flow through the study process.



Following re–consent, participants were randomised to an intervention or control group. Subsequently, weekly surveys were sent out to participants between 12 April 2020 and 27 June 2020. Each survey asked participants about their COVID–19 diagnosis / status and symptoms, their level of social isolation, as well as their weekly stress (using the SOS–S) and boredom (using the MSBS) levels, and their weekly alcohol use. For each weekly survey, the daily drinking diary (see Appendix C) method

was used to capture the number of weekly units, drinking days, and heavy drinking days (Gmel & Rehm, 2004). Previous work indicates that daily drinking diary methods offer a reliable and valid approach to measuring alcohol consumption (e.g., Del Boca & Darkes, 2003; Poikolainen et al., 2002). Meanwhile, the APQ was used to measure alcohol–related problems. For each survey, each questionnaire was presented in counterbalanced order to reduce any order effects.

The date each survey was sent out to the participants, the response cut-off date, the drinking assessment used, and the response rate (RR) for each survey is shown in Table 4.3. As the number of responses to each weekly survey began to wane in June 2020, we decided to reduce participant burden by sending out two further monthly (instead of weekly) surveys in August 2020 and September 2020. As the frequency of surveys was reduced, it was hoped that the RR for subsequent surveys would stabilise or improve due to the decreased work required of the participants. The monthly surveys reverted to using the TADD alcohol use as described in Study 1.

Table 4.3 Survey dates, drinking assessments used and response rates for each survey.

T	Study	Date Sent	Cut-Off Date	Drinking Assessment	RR
0	1	-	-	TADD	-
1	1	-	-	TADD	-
2	2	19 April 2020	25 April 2020	Daily Diary	69.07%
3	2	26 April 2020	02 May 2020	Daily Diary	91.75%
4	2	03 May 2020	09 May 2020	Daily Diary	93.81%
5	2	10 May 2020	16 May 2020	Daily Diary	68.04%
6	2	17 May 2020	23 May 2020	Daily Diary	76.29%
7	2	24 May 2020	30 May 2020	Daily Diary	68.04%
8	2	31 May 2020	06 June 2020	Daily Diary	62.89%
9	2	07 June 2020	13 June 2020	Daily Diary	63.92%
10	2	14 June 2020	20 June 2020	Daily Diary	45.36%
11	2	21 June 2020	27 June 2020	Daily Diary	51.55%
12	2	01 August 2020	08 August 2020	TADD	49.48%
13	2	01 September 2020	08 September 2020	TADD	44.33%

Note. T = timepoint; TADD = Typical Atypical Drinking Diary; RR = response rate.

Randomisation

Participants were randomised into one of two groups which received either a PFI or general feedback (i.e., control). Random allocation to each group was stratified by AUDIT category, whereby 50% of each AUDIT group (i.e., 0–7 = low-risk drinking, 8–15 = increasing risk of harm, 16–19 = higher risk drinking, scores > 20 = alcohol dependence) were allocated to each group. This prevented a situation where (for example) only low risk drinkers were allocated to the intervention group.

Intervention

As shown in Appendix C, the PFI included information pertaining to the participant's self–reported alcohol use from the previous survey and compared their consumption to a typical low–volume drinker. To make the PFI "feel" more personal, the participant's age and place of residence data were used as part of the intervention message (e.g., if the participant was 27 years of age, the age range shown would be 25–34). The intervention also informed participants how their drinking compared to the UK LRDG (i.e., 14 units per week), reminded them of the health implications of long–term excessive drinking, and provided a link to further information on available support for reducing their alcohol consumption should they wish to do so. Similar interventions have been shown to be effective (Carey et al., 2007; Cronce & Larimer, 2011; Lewis et al., 2019; Patrick et al., 2014). Finally, the PFI message thanked participants for their participation and reminded them that another survey would be sent soon. Meanwhile, those allocated to the control group received the "thank you" and "reminder" messages only.

Sample

A total of 97 participants signed up for Study 2. Of these, 37 were excluded to ensure data integrity: 32.99% had > 40% missing data; 2.06% reported living in sub–regions with an inadequate number of responses; 2.06% were classified as multivariate outliers based upon a Mahalanobis distance that is significant at $p < .001$ (Tabachnick & Fidell, 2014; Verardi & Dehon, 2010); 1.03% had gender recorded as transgender or 'prefer not to say'. This left 60 cases for analysis. Sociodemographic characteristics are shown in Table 4.4.

Table 4.4 Sociodemographic characteristics of the sample.

Variable	Total (SD)	Control (SD)	Intervention (SD)
N	60	30	30
Age	31.40 (10.40)	31.97 (11.16)	30.83 (9.74)
Gender = Female	48	25	23
Ethnicity = White	95.00%	93.33%	96.67%
Sub-region			
N. Europe	53.33%	50.00%	56.67%
E. Europe	3.33%	3.33%	3.33%
S. Europe	25.00%	30.00%	20.00%
W. Europe	8.33%	0.00%	16.67%
N. America	5.00%	6.67%	3.33%
Oceania	5.00%	10.00%	0.00%
Education			
GCSE/GED	1.67%	3.33%	0.00%
A-levels/High School Diploma	13.33%	13.33%	13.33%
Undergraduate Degree	28.33%	23.33%	33.33%
Graduate Degree	45.00%	43.33%	46.67%
Doctoral Degree or Higher	11.67%	16.67%	6.67%
Occupation			
Full-time students	40.00%	50.00%	30.00%
Never worked / long-term unemployment	5.00%	0.00%	10.00%
Skilled labourer	1.67%	3.33%	0.00%
Trained professional	18.33%	10.00%	26.67%
Support staff	8.33%	13.33%	3.33%
Administrative staff	5.00%	6.67%	3.33%
Junior management	16.67%	16.67%	16.67%
Middle management	5.00%	0.00%	10.00%

Key Worker = Yes	20.00%	16.67%	23.33%
Income			
Under £2,500	13.33%	13.33%	13.33%
£2,500 to £4,999	5.00%	6.67%	3.33%
£5,000 to £9,999	5.00%	3.33%	6.67%
£10,000 to £14,999	11.67%	20.00%	3.33%
£15,000 to £19,999	20.00%	23.33%	16.67%
£20,000 to £24,999	11.67%	10.00%	13.33%
£25,000 to £29,999	8.33%	6.67%	10.00%
£30,000 to £34,999	10.00%	6.67%	13.33%
£35,000 to £39,999	1.67%	0.00%	3.33%
£40,000 to £44,999	1.67%	0.00%	3.33%
£45,000 to £49,999	5.00%	3.33%	6.67%
£50,000 or more	6.67%	6.67%	6.67%
Subjective Social Status			
Working Class	15.00%	20.00%	10.00%
Lower-middle Class	55.00%	50.00%	60.00%
Upper-middle class	30.00%	30.00%	30.00%
Upper Class	0.00%	0.00%	0.00%
Marital Status			
Single/Separated/Widowed/Divorced	66.67%	70.00%	63.33%
Married/Domestic Partnership	33.33%	30.00%	36.67%
Timepoints Experienced COVID-19 Symptoms	3.77 (2.94)	3.47 (3.04)	4.07 (2.85)
No. People in Same Household	2.75 (1.26)	2.77 (1.18)	2.73 (1.34)
No. Offspring	0.40 (0.95)	0.33 (0.87)	0.47 (1.03)
Isolated With			
Alone	12.35%	12.99%	11.68%
With children	19.77%	15.54%	24.25%

With romantic partner	46.95%	39.27%	55.09%
With parents	5.76%	6.78%	4.49%
With siblings	9.88%	11.30%	8.38%
With housemates	10.32%	12.71%	7.78%
With friends	0.44%	0.85%	0.00%
With extended family	2.91%	2.82%	2.99%

Note. Data are presented as mean (SD) for continuous measures and % for categorical measures. Symptoms included: (1) a high temperature, (2) a new, continuous cough, (3) a continuous headache, (4) a loss of taste and/or smell, (5) muscle aches, (6) a sore throat. Countries in the sample included: Australia (*n* = 3), France (*n* = 3), Germany (*n* = 2), Hungary (*n* = 2), Italy (*n* = 15), United Kingdom (*n* = 32), and United States (*n* = 3).

Analysis

Descriptive statistics (means, standard deviations, and the proportion of missing data) were calculated for key study variables. The proportion of missing data is shown in Appendix C. As this study employed a longitudinal design with data collected over 14 timepoints, multiple imputation of missing values was not necessary before performing mixed–model analysis (Twisk et al., 2013). Generalised linear mixed models (GLMMs) with a random intercept for participant were used to test our hypotheses. More complex models (e.g., random slopes, polynomials) were tested. However, adding parameters to the models did not significantly improve model fit and oftentimes led to convergence issues (likely due to increased model complexity). The number of weekly units consumed was semicontinuous (i.e., a skewed continuous outcome with many zeros). Therefore, a two–part model was used to analyse: (a) the process underlying whether a participant had a zero vs. non–zero value for the outcome (i.e., whether the participant consumed alcohol at a given timepoint); and (b) the process that governs level of the outcome (i.e., the number of units consumed) if the participant had experienced any amount of it (Boulton & Williford, 2018). In other words, a logit model was used to model the likelihood of a non–zero and estimate odds ratios (ORs) and a LMM was used to model the non–zero data. The non–zero data were also log transformed to improve the distribution of the data (Boulton & Williford, 2018). For count outcomes (i.e., the number of drinking days, the number of heavy drinking days, and the number of alcohol–related problems), Poisson models were used and incidence rate ratios (IRRs) were estimated. All continuous predictors were grand mean centred to aid interpretation and reduce potential collinearity. Models were separated by construct to conserve statistical power and to avoid erroneously conditioning the model estimates (McMullin et al., 2020). Benjamini and Hochberg's (1995) method of false discovery rate (FDR) control was used to reduce the type I error rate when testing hypotheses. The same set of covariates from Study 1 were used here with the addition of whether the participant was experiencing isolation (as lockdown rules changed over the course of the study). Significant continuous by continuous interactions were probed using the JN technique (Johnson & Neyman, 1936) as suggested by (Hayes, 2022). Results were considered significant when $p < 0.05$ or when the 95% CI did not contain zero.

4.3.2. Results

Table 4.5 displays the descriptive statistics for the main study variables in terms of alcohol use, drinking motives, impulsivity, stress and boredom. A series of Kruskal-Wallis (for continuous variables) and chi-square (for categorical data) tests were used to test for any between–group differences. There were statistically significant differences between the control and intervention groups in terms of the percentage of non–zero values for the number of units of alcohol consumed, $\chi^2(1) = 34.40$, $p < .001$, the number of drinking days, $\chi^2(1) = 30.67$, $p < .001$, conformity drinking motives $\chi^2(1) = 6.88$, $p = .009$, MSBS total score, $\chi^2(1) = 7.12$, $p = .008$, lack of perseverance, $\chi^2(1) = 8.16$, $p = .004$, sensation seeking, $\chi^2(1) = 36.95$, $p < .001$, positive urgency, $\chi^2(1) = 6.46$, $p = .011$, and risk-taking (DOSPERT), $\chi^2(1) = 22.72$, $p < .001$.

Intervention effectiveness

Models containing time x group interactions and covariates were used to test whether the PFI was effective over the course of the study. This interaction was significant for alcohol–related problems (IRR = 0.95, FDR–adjusted $p = .023$). As shown in Figure 4.4, the number of alcohol–related problems (APQ) experienced by participants reduced at a faster rate in the intervention group compared to the control group.

Associations between drinking motives and alcohol use

Coping motives were positively associated with all alcohol use outcomes: the likelihood of a non–zero value for weekly units (OR = 1.67, FDR–adjusted $p = .019$), the number of units consumed per week (B = 0.18, FDR–adjusted $p < .001$), the number of weekly drinking days (IRR = 1.19, FDR–adjusted $p = 0.020$), the number of weekly heavy drinking days (IRR = 1.42, FDR–adjusted $p < .001$), and the number of alcohol–related problems (IRR = 1.29, FDR–adjusted $p = .008$). Enhancement motives were positively associated with alcohol–related problems (IRR = 1.13, FDR–adjusted $p = .008$) and heavy drinking days (IRR = 1.13, FDR–adjusted $p = .019$). Social motives were positively associated with alcohol–related problems (IRR = 1.14, FDR–adjusted $p < .001$). No other significant relationships were found.

Table 4.5 Descriptive statistics (M and SD) for main study variables (*N* = 60).

Variable	Total (SD)	Control (SD)	Intervention (SD)
N	60	30	30
AUDIT	6.58 (4.20)	6.67 (4.77)	6.5 (3.55)
% Non-zero [a]	**76.29%**	**66.95%**	**86.10%**
Alcohol Units [b]	14.57 (13.40)	15.79 (15.43)	13.58 (11.42)
Drinking Days	**2.19 (1.98)**	**1.84 (2.00)**	**2.55 (1.90)**
Heavy Days	0.52 (0.96)	0.55 (1.01)	0.49 (0.89)
APQ	1.05 (2.11)	1.08 (2.16)	1.01 (2.07)
DMQ–R Social	15.25 (5.59)	15.3 (5.91)	15.2 (5.25)
DMQ–R Coping	9.07 (4.25)	9.07 (4.29)	9.07 (4.21)
DMQ–R Enhancement	12.27 (5.03)	12.43 (5.26)	12.1 (4.79)
DMQ–R Conform	**6.88 (2.29)**	**6.70 (2.20)**	**7.07 (2.37)**
SOS–S	21 (10.21)	21.43 (10.87)	20.54 (9.44)
PSRS Total	24.67 (8.59)	25.37 (8.71)	23.97 (8.42)
MSBS	**90.41 (38.78)**	**94.16 (39.14)**	**86.44 (38.06)**
SUPPS–P Negative Urgency	8.80 (3.18)	8.90 (3.20)	8.7 (3.17)
SUPPS–P Premeditation	6.68 (1.87)	6.67 (1.66)	6.7 (2.05)
SUPPS–P Perseverance	**7.02 (2.13)**	**7.33 (2.44)**	**6.70 (1.70)**
SUPPS–P Sensation Seeking	**9.47 (2.61)**	**8.93 (2.21)**	**10.00 (2.87)**
SUPPS–P Positive Urgency	**7.20 (2.35)**	**7.03 (2.30)**	**7.37 (2.38)**
DOSPERT Total	**86.57 (19.42)**	**83.37 (18.58)**	**89.77 (19.75)**

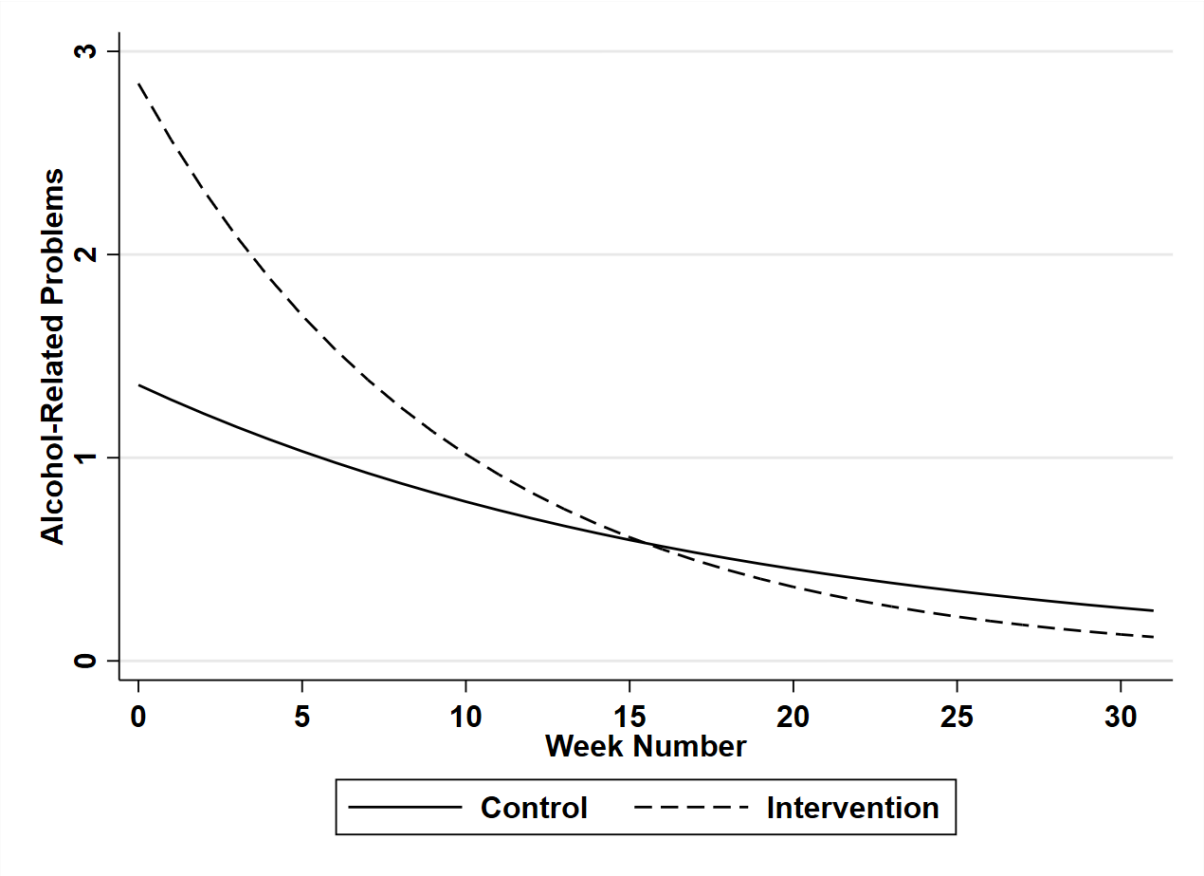
Note. 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; DMQ–R = Revised Drinking Motives Questionnaire; SOS–S = Short Stress Overload Scale; PSRS = Perceived Stress Reactivity Scale; MSBS = Multidimensional State Boredom Scale; SUPPS–P = The Shortened Urgency, Premeditation (lack of), Perseverance (lack of), Sensation Seeking, Positive Urgency, Impulsive Behaviour Scale; DOSPERT = Domain–Specific Risk–taking Scale.

Significant group differences ($p < .05$) are in boldface.

[a] Reflects the percentage of records recorded as a non–zero value.

[b] The mean among of non–zero values.

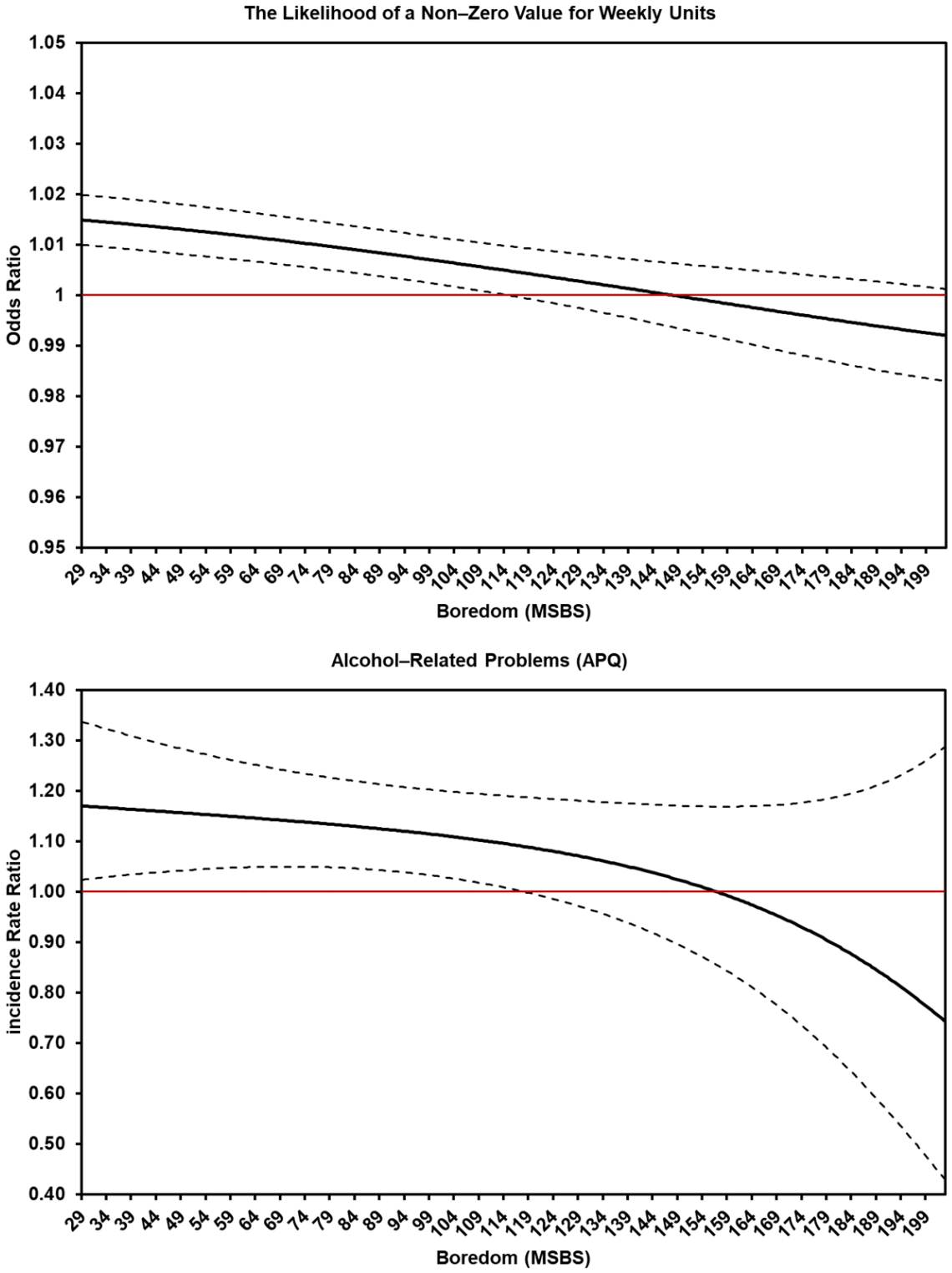
Figure 4.4 The effect of the personal feedback intervention on alcohol–related problems over time.



Associations between impulse control, stress, boredom, and alcohol use

Risk–taking (DOSPERT score), IRR = 1.03, FDR–adjusted *p* = .006, and (lack of) premeditation, IRR = 1.45, FDR–adjusted *p* = .023, were positively associated with heavy drinking days. Similarly, sensation seeking was positively associated with alcohol–related problems, IRR = 1.24, FDR–adjusted *p* = .019. No other significant associations were found. Moderation analyses suggested that boredom (MSBS score) moderated the association between risk–taking and the likelihood of a non–zero value for weekly units (OR = 0.99, FDR–adjusted *p* = .020) and the relationship between sensation seeking and alcohol–related problems (IRR = 0.99, FDR–adjusted p = .019). No other significant interactions were observed. JN plots (see Figure 4.5) revealed that *those who were more impulsive and less bored were more likely to report increased alcohol use*. Specifically, when MSBS scores were < 115, risk–taking was significantly positively associated with the likelihood of a non–zero value for weekly units. Similarly, when MSBS scores were < 118, the relationship between sensation seeking and alcohol–related problems was positive and significant.

Figure 4.5 Johnson–Neyman plots illustrating significant impulse control x boredom interactions.



Note. Models were adjusted for age, gender, ethnicity, socioeconomic status, the number of symptoms experienced, whether the respondent was experiencing isolation, group allocation, study timepoint, and whether the respondent was isolating with children. 1 unit = 8g pure ethanol; APQ = Alcohol Problems Questionnaire; MSBS = Multidimensional State Boredom Scale. Dashed lines represent the 95% CI.

4.3.3. Discussion

Study 2 aimed to investigate the effectiveness of a PFI intervention on alcohol use behaviour during the COVID–19 pandemic. This study also aimed to assess relationships between drinking motives, impulse control, stress, boredom, and alcohol use over a longitudinal basis, during the pandemic. The PFI was effective at reducing the number of alcohol–related problems experienced by participants over the course of the study but did not significantly reduce other alcohol use variables. Drinking to cope was positively associated with all alcohol use outcomes; enhancement motives were positively associated with alcohol–related problems and heavy drinking days; and social motives were positively associated with alcohol–related problems. In terms of impulse control, both risk–taking and (lack of) premeditation were positively associated with heavy drinking days; and a positive relationship between sensation seeking and alcohol–related problems was identified. Like Study 1, boredom was a significant moderator, whereby those who were more impulsive (in terms of risk–taking and sensation seeking) and less bored tended to report increased alcohol use behaviour and alcohol–related problems. Thus, this analysis also established how some theoretical mechanisms of alcohol misuse operated over the course of the pandemic among our sample.

4.4. General Discussion

In Study 1, we found that approximately one in four respondents reported drinking more and around one in ten reported experiencing an increased number of alcohol-related problems. These findings correspond to similar work conducted during the COVID-19 pandemic (Clay et al., 2021; Garnett et al., 2021; Jacob et al., 2021; Kilian et al., 2021; Koopmann et al., 2020; Neill et al., 2020; Schmits & Glowacz, 2021; Tran et al., 2020). Similarly, as in previous work, most respondents reported feeling more bored during lockdown (Garnett et al., 2021; Latif & Karaman, 2021; Martarelli & Wolff, 2020). Stress levels, however, either stayed the same or decreased for most and, despite our prediction, stress was not significant in any model in either study. These findings are at odds with previous literature that has found the pandemic has been associated with increased mental distress (Garnett et al., 2021; Jacob et al., 2021; Koopmann et al., 2020; Neill et al., 2020; Tran et al., 2020).

One explanation for this discrepancy may be that the physiological and psychological effects of acute vs. chronic stress differ (see Crosswell & Lockwood, 2020; Epel et al., 2018; Stephens & Wand, 2012 and section 1.3.2). Thus, it is plausible that the effect of stress on drinking differs as a function of the timescale and severity. To the best of our knowledge, this has not been investigated specifically and is therefore an important area for future work. Alternatively, it may be due to differences in measures used; several studies cited above utilised measures that are typically used to diagnose manifestations of poor mental health (e.g., depression, anxiety) in clinical settings, while we used a measure of perceived stress. Similar to what was observed here, other non-clinical studies carried out during the pandemic, using momentary assessments of positive and negative affect, suggested that pre–consumption affect was not associated with increased drinking during the pandemic (Tovmasyan et al., 2023). Finally, the discrepancy may relate to the nature of our sample, which was predominantly highly educated Westerners.

On the other hand, drinking to cope (i.e., drinking to reduce negative affect) was positively related to all alcohol use outcomes in Study 2. This is particularly concerning given that a substantial number of people also reported "drinking to cope" with pandemic–related distress in previous studies (e.g., Gadermann et al., 2021; Rodriguez et al., 2020; Tucker et al., 2022; Wardell et al., 2020), and that meta–analysis shows that solitary drinking (e.g., during social isolation) and drinking to cope with negative affect are both precursors for future alcohol–related problems (Skrzynski & Creswell, 2020). Similarly, enhancement (i.e., drinking to increase positive affect) and social drinking motives (i.e., drinking to improve social situations) also predicted increased heavy drinking and alcohol–related problems in Study 2.

In Study 1, however, social motives were associated with decreased alcohol–related problems. Nevertheless, this heterogeneity in results is also reflected in the literature. For example, several studies which examined the role of drinking motives on drinking behaviour during the pandemic found significant positive associations between coping motives and alcohol use behaviour (e.g., Bollen et al., 2021; Carbia et al., 2022; Irizar et al., 2021; Monk et al., 2023; Prestigiacomo et al., 2021; Tucker et al., 2022). Others, on the other hand, also identified enhancement (e.g., Bollen et al., 2021; Monk et al.,

2023; Prestigiacomo et al., 2021), social (e.g., Bollen et al., 2021; Carbia et al., 2022; Tucker et al., 2022) and conformity (Monk et al., 2023) motives as significant predictors of drinking behaviour. Therefore, taken together, it is clear that drinking to cope is an important factor in predicting who is most at risk of alcohol–related harm post–pandemic. Moreover, further research aiming to understand what is driving the diversity in drinking motive results between–studies would be useful as it may be that an important third variable is identified.

Those who were high in risk–taking (DOSPERT total score) tended to face fewer alcohol–related problems in Study 1, despite impulse control being an established risk–factor for addictive behaviours (for reviews, see: Dalley & Ersche, 2019; Lee et al., 2019). However, in support of our hypotheses, results from Study 2 suggest that risk–taking, lack of premeditation, and sensation seeking were associated with alcohol use outcomes. These results are in line with previous literature. For instance, in meta–analysis, Coskunpinar et al. (2013) showed that all UPPS–P traits are related to alcohol use. Similarly, risk–taking (Canning et al., 2021; Courtney et al., 2012), and specifically the DOSPERT (Brailovskaia et al., 2018; Farnham et al., 2018), is consistently associated with alcohol–related outcomes. In light of the literature, it may be that the risk–taking results shown in Study 1 were a result of sampling error and / or a statistical artefact. Alternatively, Tsai & Zen (2021) found that risk–taking increased as a function of pandemic severity (i.e., those who were more severely affected by the pandemic were more likely to engage in risky activities than thank those who were less severely affected). Thus, another explanation may be that perceived pandemic severity was greater in Study 2 due to the ongoing social isolation experienced by those who took part (i.e., the study was longitudinal, not cross–sectional). Therefore, in general, poor impulse control is related to alcohol use and misuse.

Boredom was a critical moderator in both studies. In Study 1, those who were less impulsive (in terms of lack of premeditation), who also reported feeling more bored, were more likely to increase alcohol use during the isolation and vice-versa. A similar pattern was also observed in Study 2, whereby those who were more impulsive (in terms of risk–taking and sensation seeking) and less bored tended to report increased alcohol use behaviour and alcohol–related problems. Previous research has identified boredom as a risk-factor for health risk behaviours, such as substance misuse (e.g., Wegner

& Flisher, 2009). However, we found that although most participants reported increased boredom, the majority also reported a decrease in alcohol use (see Study 1). A reason for the decreased alcohol use in those that were showing higher rates of boredom may relate to the lack of interest in alcohol outside of the typical situations. For instance, drinking is typically a social activity (e.g., Niland et al., 2013), and we found that social motives were the most endorsed drinking motive among our sample; indeed, those with higher social drinking motives reported fewer alcohol-related problems in Study 1. Thus, this suggested that, on average, our sample were motivated to drink when in social situations; something clearly impacted significantly by the social isolation.

Reward expectancy (i.e., the anticipated reward associated with alcohol consumption) is determined by drinking motives, with those who tend to 'drink to cope' showing the highest anticipated reward expectancy (Birch et al., 2004; Grant & Stewart, 2007). In our samples, coping was one of the least endorsed motives, suggesting that our sample was low in this trait. In this sense, the expected positive reinforcement associated with drinking (i.e., alleviation of the boredom) would not be a strong motivator to drink in our sample. Further research is needed to disentangle the relationship between drinking motives, reward expectancy, boredom and alcohol consumption.

Boredom is associated with a negative affective state, which can be high– or low– arousal (Fahlman et al., 2013). In either case, boredom is associated with anhedonia, thus theoretically decreasing the pleasure associated with usually rewarding activities (Watson et al., 2020). Although typically boredom–induced anhedonia is not associated with substance misuse (Nikčević et al., 2017), boredom is a complex and multifaceted phenomenon (Raffaelli et al., 2018). Therefore, as people were exposed to an unprecedented period of social isolation, and subsequently high levels of boredom were reported here and in other studies (e.g., Droit-Volet et al., 2020), it may be that the phenomenon experienced during the pandemic is dissimilar (in terms of intensity and duration) than previous work (e.g., laboratory–based studies) or during previous times. Taken together, these factors may offer a potential explanation for our findings.

To summarise so far, drinking to cope with negative affect, poor impulse control, and increased boredom were important in understanding pandemic–related increases in alcohol use behaviour among

a sizable minority of participants who showed increased alcohol use. However, we also tested the efficacy of a web–based PFI for reducing alcohol use and misuse, finding that the intervention successfully reduced the number of alcohol–related problems experienced by respondents over time, but not other outcomes. Previously, similar web–based interventions have been typically shown to reduce hazardous drinking among heavy drinkers (Bewick et al., 2008; Doumas et al., 2009; Lewis et al., 2019). Thus, as alcohol dependence severity is strongly associated with APQ total score (i.e., the number of alcohol–related problems experienced; r = .63) (Drummond, 1990), our findings are in agreement with the literature. Therefore, as highlighted in a review by Deutsch-Link et al. (2022), access to psychiatric services was reduced mid–pandemic and there are significant challenges in managing alcohol treatment post–pandemic (e.g., increased alcohol–associated liver disease, alcohol–related deaths, and waiting times for treatment), thus PFI may provide a relatively cheap and effective method of reducing alcohol–harm among at–risk groups: for example, among those who tend to drink to cope with negative affect, or individuals who lack impulse control.

4.4.1. Limitations

We acknowledge several study limitations. First, there were relatively high levels of attrition. This may have been driven by high participant burden, such as the length of the survey in Study 1 (as several relatively long and detailed psychometric instruments were employed) or the frequency of survey request in Study 2. However, a limitation of previous work in this area is that brief single–item measures, that may be limited by reduced content validity, were used (Clay et al., 2021). Thus, the present work overcomes this limitation, providing nuance at the expense of sample size. Nevertheless, the bias introduced by missing data was minimised by employing sophisticated statistical techniques, such as multiple imputation (Woods et al., 2023) or longitudinal mixed–model analysis (Twisk et al., 2013). Second, respondents tended to be White, highly educated, and relatively wealthy. Ultimately, this may limit the generalisability of our findings to those with similar sociodemographic characteristics. Similarly, the COVID-19 pandemic has been an unprecedented time, thus pandemic-related findings may only hold true inside this timeframe. Third, self-report measures are prone to measurement error. For instance, there is no way to independently verify self-report drinking and people

typically under-estimate their alcohol consumption on questionnaires (Northcote & Livingston, 2011). Fourth, "true" baselines for drinking behaviour, stress, and boredom were unavailable and retrospective measures were employed as a proxy. Therefore, causal inference is precluded. Fifth, accurately estimating determinants of change is notoriously difficult and these considerations informed our analysis. Therefore, we purposefully tried to avoid spurious findings by not including baseline measures in our cross–section analyses (Glymour et al., 2005). Finally, there are other potential confounding factors that were not accounted for here, such as mood disorders (Charles et al., 2021), as these data were not available.

4.4.2. Conclusion

We aimed to understand how a period of long-term social isolation affected alcohol use, particularly focussing on drinking motives, negative affect (i.e., stress and boredom), and impulse control. Our rationale was not just to characterise patterns observed during COVID–19, but to use the government–enforced lockdowns to model theoretical mechanisms by which alcohol consumption in the home could be affected by periods of enforced social isolation. We found that approximately one quarter of respondents reported drinking more and around one tenth reported facing an increased number of alcohol related problems. Coupled with recent national statistics which suggest that alcohol–related deaths in the UK reached an all-time high in 2020 (14 deaths per 100,000 people) (Office for National Statistics, 2021), it is clear an 'at risk' group of individuals, who deserve immediate attention, may also require the allocation of future resources to mitigate harm. Such at–risk individuals include those who drink to cope with negative affect and those who are more impulsive (in terms of risk–taking, lack of premeditation, and sensation seeking). These individuals may benefit from PFI while awaiting more extensive treatment. Moreover, these relationships between impulse control and drinking behaviour were generally strengthened by reduced pandemic–related boredom. Therefore, further research in understanding the complex interplay between impulsivity and boredom is required. Finally, these findings have important implications when considering mechanisms of alcohol misuse; researchers should potentially consider evaluating people's social interactions and isolation status during future work and interventions.

Chapter 5. The Role of Impulse Control in the Mediation Association Between Emotional Dysregulation, Cumulative Lifetime Stressor Exposure, and Lifetime Alcohol Use

The research reported in this chapter has been published as:

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Chapter Foreword

The main hypothesis of this thesis was extended here, in the final empirical chapter of this dissertation, to include emotional dysregulation. Specifically, it was hypothesised that the association between cumulative lifetime stressor exposure and lifetime alcohol use would be mediated by emotional dysregulation, and that increased impulsivity would strengthen these relationships. This chapter contributes to the overall aims of this thesis by investigating the role of several self–report and behavioural measures of impulsivity on lifetime alcohol consumption in the context of cumulative lifetime stress.

Abstract

Alcohol misuse is a global health issue. Although stress, trait impulsivity, and emotional dysregulation are independent predictors of alcohol use and misuse, relatively little is known about the potential mechanisms that link these risk–factors together. To address this issue, we tested a theory–driven model, which posits that emotional dysregulation mediates the association between cumulative lifetime stressor exposure and lifetime alcohol use. We also hypothesised that heightened impulsivity would strengthen these relations. Data from 296 participants (150 women) aged 18–68 (M = 39.60, SD = 12.11) were collected using Prolific. Participants completed the Stress and Adversity Inventory for Adults; Difficulties in Emotional Regulation Scale Short Form; Lifetime Drinking History Questionnaire; and a battery of self–report and behavioural measures of impulsivity. We used conditional process analysis to test our pre–registered hypotheses, and controlled for age, sex, and socioeconomic status in all models. As hypothesised, emotional dysregulation fully mediated the relation between cumulative lifetime stressor exposure and lifetime alcohol use. We also found that several facets of impulsivity moderated these associations. For example, as levels of negative urgency increased, the associations between cumulative lifetime stressor exposure and emotional dysregulation, emotional dysregulation and lifetime alcohol use, and lifetime stress exposure and lifetime alcohol use, via emotional dysregulation, strengthened. These findings have important implications. They integrate several prominent risk-factors for alcohol misuse into a single model, extending prior research and generating interesting and novel lines of enquiry. They also highlight the clinical utility for lifetime stress exposure screening and identify potential targets for personalised treatment interventions.

Ethnics Approval Reference: SHFEC 2021–022A (Appendix D)

5.1. Introduction

Alcohol misuse (i.e., hazardous drinking) is a global health concern (World Health Organization, 2022). It is associated with severe socioeconomic problems, and chronic alcohol misuse is a leading cause of morbidity and mortality (World Health Organization, 2018). A recent meta-analysis of over 1.6 million people suggested that approximately one-in-five patients who enter the UK health system misuse alcohol, and one in ten are dependent (Roberts et al., 2019). Despite this, AUD treatment rates are low (Mekonen et al., 2021) and current interventions are only modestly effective (Ray et al., 2019). Personalised treatment (i.e., identifying the best treatment matches for individual patients) appears to be a goal that the alcohol research community has been working towards in an effort to advance treatment efficacy (Litten et al., 2015). An important first step towards achieving this goal is to better understand how previously identified risk-factors for alcohol misuse interconnect.

Stress is a strong risk factor for both alcohol misuse (Jose et al., 2000; Ruisoto & Contador, 2019) and emotional dysregulation (Compton et al., 2013; Paulus et al., 2021). Emotional dysregulation is defined as the inability to identify, understand, accept and appropriately react to unwelcome emotional states (Kaufman et al., 2016). Extensive theoretical and empirical work affirms the that link between stress, emotional dysregulation and the risk for alcohol misuse are the result of chronic HPA axis activation (Koob & Kreek, 2007; Koob & Schulkin, 2019; Milivojevic & Sinha, 2018). Repetitive activation of the HPA axis, caused by cumulative lifetime stressor exposure, results in neurophysiological changes to areas associated with emotional processing, stress reactivity, and reward regulation (Casement et al., 2015; Kim et al., 2013). Ultimately, these neurophysiological changes can degrade individuals' ability to regulate their emotions, putting them at increased risk of 'self-medicating' (i.e., compensating) through alcohol misuse.

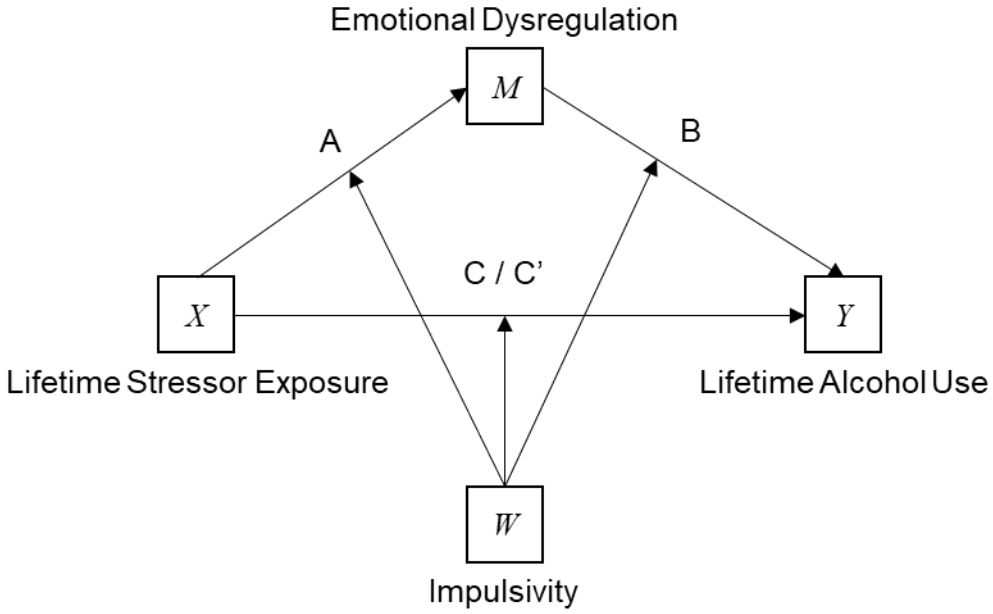
Alcohol is a powerful trigger for activation of stress systems, including the HPA axis (Armario, 2010; Milivojevic & Sinha, 2018) and an important underlying loop exists in which individuals are left unable to regulate their emotions due to stressful life experiences. In turn, increased emotional dysregulation means that individuals may choose to misuse alcohol, attempting to self-regulate (Wemm et al., 2022). Finally, following periods of prolonged alcohol misuse (e.g., binge drinking),

neuroadaptations and the experience of more stressful life events (e.g., failing to fulfil commitments and interpersonal issues) may occur, exacerbating the emotional dysregulation and subsequent inability to cope (Carbia et al., 2021). Additional evidence for this is found in the clinical literature, where alcohol-dependent (versus non-dependent social drinkers) commonly present with such neuroadaptations (Sinha, 2012)

The association between impulsivity and addition (e.g., AUD) is also well established (Belin et al., 2008; Dalley & Ersche, 2019; Karlsson Linnér et al., 2021; Kreek et al., 2005; Lee et al., 2019; Voon et al., 2020). Impulsivity is defined as a multidimensional personality trait whereby individuals have the propensity to act without forethought to internal or external stimuli with little to no regard for possible negative consequences related to these actions (Strickland & Johnson, 2020). Several clinical diagnoses in the *Diagnostic and Statistical Manual for Mental Disorders, 5th Edition* (American Psychiatric Association, 2013) include impulsivity as a core diagnostic criterion. Research on many of these diagnoses, such as personality disorder (Garofalo et al., 2018), attention deficit hyperactivity disorder (ADHD) (Retz et al., 2012), and AUD (Herman & Duka, 2019), suggests that although related, both emotional dysregulation and impulsivity independently contribute to these conditions.

Prior research from our lab has focussed on how impulsivity may act as a moderator of craving and drinking in times of acute (Clay et al., 2018; Clay & Parker, 2018) and chronic stress (Clay et al., 2021, 2022). Meanwhile, others have focussed on how AUD influences impulsivity via emotional dysregulation (Jakubczyk et al., 2018) or whether the interaction between cumulative lifetime stressor exposure and impulsivity predicts hazardous drinking (Fox et al., 2010). However, no studies have integrated cumulative lifetime stressor exposure, impulsivity and emotional dysregulation into a single model. To address this issue, we consolidated the theories outlined above into a single model (see Figure 5.1), which predicts who is most likely to consume increased amounts of alcohol over the life course. Establishing how such clearly defined risk-factors for alcohol use fit together into a unified theory will help to better determine personalised interventions which focus on impeding the onset and progression of alcohol misuse and related harms.

Figure 5.1 A conceptual diagram illustrating the hypothesised associations between cumulative lifetime stressor exposure (X), emotional dysregulation (M), lifetime alcohol use (Y), and impulsivity (W).



Towards the aim of predicting lifetime alcohol use, we tested several pre–registered hypotheses using conditional process analysis (i.e., moderated mediation analysis). Conditional process analysis is a statistical technique that allows researchers to answer questions, such as *"through which mechanism does an effect operate?"* (i.e., mediation) and *"when/for whom does an effect exist?"* (i.e., moderation) (Hayes, 2022; Hayes & Rockwood, 2020). Such analyses assume a cause-and-effect sequence. For example, the effect of *X* on *Y* is caused by an increase through *M*. Certain causal inference is precluded in cross-sectional research as a time component or random assignment is required (Imai et al., 2010; VanderWeele, 2016). However, variables may be correlated due to causal relation (Hayes & Rockwood, 2020). Therefore, in the presence of a robust theoretical framework, strong statistical evidence, and clear arguments for causality, testing for statistical mediation using cross-sectional data is a valid approach (Hayes, 2022; Hayes & Rockwood, 2020).

Our primary hypothesis was that the positive relation between cumulative lifetime stressor exposure and lifetime alcohol use would operate through emotional dysregulation (mediation effect). For mediation to occur in this manner, we also hypothesised that cumulative lifetime stressor exposure would positively predict emotional dysregulation and lifetime alcohol use, and that emotional

dysregulation would positively predict lifetime alcohol use. We further hypothesised that impulsivity would moderate these effects, whereby each effect would be strengthened as impulsivity increased.

5.2. Method

5.2.1. Design

This study used an online cross-sectional design. The independent variable was cumulative lifetime stressor exposure; the mediator variable was emotional dysregulation; the moderator variable was impulsivity; and the dependent variable was lifetime alcohol use.

5.2.2. Transparency and openness

We report how we determined our sample size, all data exclusions, all manipulations, and all measures in the study. A simulation-based sensitivity (Monte Carlo) power analysis (Lakens, 2022) revealed that a minimum of 110 participants were required to achieve sufficient statistical power, $(1 - \beta) = 80\%$, to test our primary hypothesis (i.e., the mediation effect); see the Appendix D for more information. Our final sample size was based on resource constraints (Lakens, 2022). In other words, we collected data from as many participants as we could afford to allow us to address our secondary hypotheses (i.e., the moderation effects). Data and pre-registered hypotheses, and code are posted on the Open Science Framework at https://osf.io/we64c. Data were analysed using Stata (version 16.1), R (version 4.2.1), and PROCESS for R (version 4.1)

5.2.3. Sample

Participants were 301 adults (152 females, 149 males) ranging in age from 18-68 years old (M = 39.56, SD = 12.09), who were recruited using Prolific Academic (https://www.prolific.co) and reimbursed at a rate of £5/hour. Participants were required to be aged 18 or older; a UK resident (as the sample size required to test our hypotheses across contexts would be substantially larger); fluent in English; and have a stable internet connection. To bolster the generalisability of the sample, as being an abstainer or heavy drinker is related to higher attrition rates (Torvik et al., 2012), recruitment was stratified by the self-reported units of alcohol consumed per week: 1-4 (25.58%), 5-9 (25.25%), 10-13 (24.58%), and 14+ (24.58%).

5.2.4. Demographic information

Demographic data collected were age, biological sex, relationship status, employment status, student status, highest level of education achieved, past year household income (GBP), and subjective social status using the socioeconomic ladder method (Operario et al., 2004).

5.2.5. Cumulative lifetime stressor exposure

The Stress and Adversity Inventory for Adults (STRAIN) was used to ass cumulative lifetime stressor exposure (Slavich & Shields, 2018). The STRAIN is an online interview that assesses stressful experiences across 55 unique acute and chronic stressors. It uses branching logic to ask follow up questions when a stressor is endorsed (see https://www.strainsetup.com), thus enabling the assessment of both objective (i.e., stressor count) and subjective (i.e., stressor severity) features of major life stressors. The STRAIN has excellent concurrent, discriminant, and incremental validity (Cazassa et al., 2020; Slavich & Shields, 2018; Sturmbauer et al., 2019) and is considered as a 'gold standard' assessment (Crosswell & Lockwood, 2020). The stressor severity index captures both the number of stressor exposures that a participant experienced over their entire lifespan and the self-reported severity of each of those stressors.

5.2.6. Emotional dysregulation

We used the 18-item Difficulties in Emotional Regulation Scale Short Form (DERS-SF) to measure emotional dysregulation (Kaufman et al., 2016). The DERS-SF is a standalone scale with six subscales: strategies, non-acceptance, impulse, goals, awareness, and clarity. Participants respond to each item using a five-point Likert scale (1 = Almost Never; 5 = Almost Always). Therefore, the maximum total score is 90, with higher scores reflecting greater ED. The short form scale retains the excellent psychometric properties of the original scale with half the number of items (Kaufman et al., 2016). The internal consistency was excellent, Cronbach's $\alpha = 0.92$.

5.2.7. Alcohol use

The AUDIT (see Chapter 2 for details) was used to assess prior alcohol use behaviour and alcohol dependency. Internal consistency of the AUDIT in the present study was excellent, Cronbach's $\alpha = 0.85$.

The Lifetime Drinking History Questionnaire (LDH–q) was used to establish participant's lifetime alcohol use (Friesema et al., 2004). The LDH-q is a validated and reliable tool that captures data about patterns of alcohol use from the onset of regular drinking (defined drinking at least once every three months) across the lifespan (Friesema et al., 2004). Five drinking periods were defined: youth (aged 12 to 18 years), young adult (aged 19 to 27 years), adult (aged 28 to 45 years), middle age (aged 46 to 60 years), and elderly (aged ≥ 61 years) (Friesema et al., 2004; Lemmens et al., 1997). In each drinking period, participants were asked to record their usual quantity (average units consumed per occasion) and frequency (the number of days per month that the participant drank at this usual level) of drinking. Participants also reported the type of beverage(s) (beer, wine, or sprits) that they consumed, the time of day (morning, afternoon, or evening) that they were drinking, the context (drinking alone or with others) in which they were drinking, and their binge drinking frequency. Using the frequency and quantity data, we calculated the average (expressed as units per week) and total consumption for each phase and across the lifespan.

5.2.8. Impulsivity

The S–UPPSP, BART, TADD, and SST (see Chapter 2 for details) were used to assess self–report and performance–based (i.e., behavioural) impulsivity. The internal consistency of each S–UPPSP in this study ranged from acceptable to good, Cronbach's $\alpha = 0.72$ to 0.84. In the present study, the SST task parameters indicated that this data was unreliable according to Verbruggen et al. (2019). Therefore, the task was omitted from analyses. Towards transparency and openness, we report the task parameters and rationale for omitting this measure from the analyses in Appendix D. A Spearman's rank correlation indicated a strong relationship between AUC and k, $rs = .87$, $p < .001$. Therefore, AUC was used in the analysis, where AUC was reverse scored (1 – score) to reflect greater delay discounting.

5.2.9. Procedure

After informed consent was obtained, participants reported their demographic information and then completed the AUDIT. Participants then completed the BART, SST, and TADD in counterbalanced order. Computer tasks were programmed using PsychoPy (Peirce et al., 2019, 2022) and hosted on Pavlovia (https://pavlovia.org/). Next, participants completed the LDH-q, S-UPPSP, and DERS-SF in

counterbalanced order using Qualtrics (Provo, Utah). Finally, participants completed the STRAIN, followed by a thank you/debrief message. To ensure data quality, two attention checks (e.g., "It is important that you pay attention to this study. Please select "Disagree some") were embedded in the AUDIT and S-UPPSP. Four participants failed the attention checks and were removed from analyses.

5.2.10. Analysis

Descriptive statistics (means, standard deviations, and the proportion of missing data) were calculated and bivariate associations were explored for key study variables. The proportion of missing data by variable is shown in Table D2. Due to the small proportion of missing data, deletion methods are unlikely to bias the results (Schafer, 1999).

The original preregistration stated that structural equation modelling would be used to test our hypotheses. However, as conditional process analyses (i.e., PROCESS models) are easier to implement and the results are largely identical, thus the choice between the two are inconsequential (Hayes et al., 2017), we chose to use PROCESS to analyse our data. Our primary hypothesis (mediation) was tested using PROCESS model 4. Our secondary hypotheses (moderation) were tested using PROCESS model 59. Bias-corrected bootstrapped (n = 10,000) 95% confidence intervals (CIs) were used to test for statistical significance in PROCESS models. Pairwise deletion and listwise deletion was used for correlations and regressions (i.e., PROCESS models), respectively.

Pre–registered covariates included: age (Leigh & Stacy, 2004), sex (White et al., 2015), and socioeconomic status (SES) (Probst et al., 2020). In our preregistration, we expected that variables related to SES would load together during factor analysis, enabling us to create an index of SES. However, this was not observed (see Appendix D). Instead, we recoded education (GCSE & below, A-levels & equivalent, and Undergraduate & higher), employment (unemployed, student, employed), household income (low < £54,406 , medium = £54,406, high > £54,406[19]), and subjective social status (low < 5, medium = 5, high > 5) into larger groups, to conserve statistical power, and included them in our models as separate variables along with age and sex. Similarly, our impulsivity variables did not

[19] The median household income in the UK for the financial year ending in 2021 was £54,406 (Office for National Statistics, 2022).

load together in a factor analysis (see Appendix C); therefore, the models were separated by construct to conserve statistical power and to avoid erroneously conditioning our estimates (Clay et al., 2022; McMullin et al., 2020).

Interactions were probed using the Johnson-Neyman technique (Johnson & Neyman, 1936). Prior to analysis, both univariate and multivariate outliers were screened following Tabachnick and Fidell (2014). Univariate outliers were assessed using z-scores, where a z-score >3.29 and < -3.29 ($p < .001$, two-tailed test) was considered a univariate outlier (one participant was excluded). The assessment of multivariate outliers was based on a Mahalanobis distance that is significant at the $p < .001$ level, assuming that the test statistic follows a chi-square distribution (Verardi & Dehon, 2010). Results were considered significant when $p < 0.05$ or when the 95% CI did not contain zero.

5.3. Results

Table 5.1 presents the sociodemographic characteristics of the sample. Table 5.2 displays the descriptive statistics (means and standard deviations) for the main study variables in terms of cumulative lifetime stress, emotional dysregulation, alcohol use behaviour, and impulsivity. Further descriptive statistics for alcohol use behaviour variables can be seen in Figure C3.

Table 5.1 Sociodemographic characteristics of the sample.

Variable	**Total (*SD*)**	**Female (*SD*)**	**Male (*SD*)**
N	296	150	146
Age	39.60 (12.11)	39.44 (12.08)	39.76 (12.18)
Average units per week			
1-4	25.68%	26.00%	25.34%
5-9	25.00%	24.00%	26.03%
10-13	24.32%	24.67%	23.97%
14+	25.00%	25.33%	24.66%
Relationship Status			
Divorced	3.38%	6.00%	0.68%
Engaged	4.73%	6.00%	3.42%
Civil partnership	1.01%	2.00%	0.00%
In a relationship	28.72%	31.33%	26.03%

Married	34.46%	29.33%	39.73%
Never married	0.68%	0.00%	1.37%
Separated	1.69%	2.00%	1.37%
Single	20.95%	19.33%	22.60%
Widowed	1.01%	1.33%	0.68%
Employment			
Unemployed	4.73%	6.00%	3.42%
Student	16.55%	16.00%	17.12%
Employed	75.34%	76.00%	74.66%
Education			
No qualifications	0.34%	0.67%	0.00%
GCSE	6.76%	6.00%	7.53%
A-levels	13.85%	15.33%	12.33%
Technical college	14.19%	12.00%	16.44%
Undergraduate degree	37.84%	37.33%	38.36%
Graduate degree	20.95%	21.33%	20.55%
Doctorate degree	4.73%	5.33%	4.11%
Household Income			
< £10,000	3.38%	4.00%	2.74%
£10,000 - £15,999	3.72%	4.67%	2.74%
£16,000 - £19,999	6.76%	8.00%	5.48%
£20,000 - £29,999	16.22%	17.33%	15.07%
£30,000 - £39,999	18.24%	20.67%	15.75%
£40,000 - £49,999	16.55%	15.33%	17.81%
£50,000 - £59,999	10.14%	7.33%	13.01%
£60,000 - £69,999	8.45%	5.33%	11.64%
£70,000 - £79,999	4.73%	3.33%	6.16%
£80,000 - £89,999	3.38%	2.67%	4.11%
£90,000 - £99,999	3.04%	4.67%	1.37%
£100,000 - £149,999	4.39%	5.33%	3.42%
> £150,000	1.01%	1.33%	0.68%
Subjective Social Status (Socioeconomic Ladder)			
1	0.34%	0.00%	0.68%

2	1.01%	0.67%	1.37%
3	5.74%	6.00%	5.48%
4	16.22%	18.00%	14.38%
5	20.27%	22.00%	18.49%
6	25.68%	24.67%	26.71%
7	22.97%	22.67%	23.29%
8	6.42%	6.00%	6.85%
9	1.35%	0.00%	2.74%
10	0.00%	0.00%	0.00%

Table 5.2 Descriptive statistics (M and SD) for main study variables.

Variable	**Total (*SD*)**	**Female (*SD*)**	**Male (*SD*)**
Lifetime Stressor Count (STRAIN)	18.24 (12.24)	20.77 (12.92)	15.64 (10.94)
Lifetime Stressor Severity (STRAIN)	45.10 (30.94)	52.58 (33.22)	37.41 (26.38)
DERS-SF Total	42.80 (13.15)	44.12 (13.20)	41.45 (13.01)
AUDIT	11.87 (6.89)	12.39 (7.24)	11.34 (6.50)
Weekly Consumption (UK Units)	31.56 (33.69)	29.61 (33.84)	33.57 (33.52)
SUPPS-P Negative Urgency	9.50 (3.08)	10.01 (3.03)	8.97 (3.05)
SUPPS-P Premeditation	7.37 (2.06)	7.49 (2.11)	7.25 (2.02)
SUPPS-P Perseverance	7.06 (2.19)	7.44 (2.18)	6.66 (2.13)
SUPPS-P Sensation Seeking	9.73 (2.87)	9.15 (2.93)	10.32 (2.68)
SUPPS-P Positive Urgency	7.54 (2.74)	7.58 (2.70)	7.51 (2.78)
BART	29.12 (11.90)	29.34 (11.96)	28.91 (11.87)
1 - AUC	0.77 (0.12)	0.78 (0.12)	0.77 (0.12)

Note. STRAIN = The Stress and Adversity Inventory for Adults; DERS-SF = The Difficulties in Emotional Regulation Scale Short Form; AUDIT = The Alcohol Use Disorders Identification Test; 1 unit = 8g pure ethanol; SUPPS-P = The Shortened Urgency, Premeditation (lack of), Perseverance (lack of), Sensation Seeking, Positive Urgency, Impulsive Behaviour Scale; BART = average number of space bar presses for unburst balloons during the Balloon Analogue Risk Task; 1 – AUC = 1 minus the area under the curve score (so that greater scores reflect greater delay discounting) for the Titrating Alternatives Delay Discounting Task.

5.3.1. Bivariate analysis

As shown in **Table 5.3**, AUDIT and lifetime alcohol use were intercorrelated (rs = .69, p < .001), and were also positively correlated with emotional dysregulation (DERS-SF; rs = .24 to .41, all ps < .001),

cumulative lifetime stress (STRAIN stressor severity; *r*s = .26 to .34, all *p*s < .001), and all measures of self-report impulsivity (S-UPPSP; *r*s = .15 to .38, all *p*s < .011), except sensation seeking and perseverance, which were not correlated with lifetime alcohol use (*p*s > .05). There was also a significant positive correlation between delay discounting (1 – AUC) and lifetime alcohol use (*r*s = .13, *p* = .025). All measures of self-reported impulsivity were intercorrelated (*r*s = .13 to .68, all *p*s < .05), except for the relations between premeditation and sensation seeking (*p* = .303) and perseverance and positive urgency (*p* = .134). Surprisingly, a negative correlation between delay discounting and risk-taking (BART) was observed (*r*s = -.12, *p* = .0375).

5.3.2. Emotional dysregulation mediates the relationship between cumulative lifetime stressor exposure and lifetime alcohol use

The results of the mediation analysis are summarised in Table 5.4. After adjusting for covariates, cumulative lifetime stressor exposure positively predicted emotional dysregulation (B = 0.15, β = 0.34, 95% CI = 32.88 to 56.09) and emotional dysregulation positively predicted lifetime alcohol use (B = 0.47, β = 0.19, 95% CI = 0.10 to 0.85). Significant indirect (B = 0.07, β = 0.06, 95% CI = 0.01 to 0.14) and total (B = 0.20, β = 0.18, 95% CI = 0.06 to 0.33) effects were observed, while the direct effect was not significant (B = 0.13, β = 0.12, 95% CI = -0.02 to 0.12). Taken together, these results suggest full statistical mediation of the association between cumulative lifetime stress exposure and lifetime alcohol use through cumulative lifetime stress exposure.

5.3.3. Negative urgency is a critical moderator of the cumulative lifetime stressor exposure, emotional dysregulation, lifetime alcohol use pathway

Tables summarising the output for the conditional process analyses are reported in the Appendix D. Moderation analysis suggested that negative urgency modified the association between cumulative lifetime stressor exposure and emotional dysregulation (B = 0.02, 95% CI = 0.01 to 0.03) and the association between emotional dysregulation and lifetime alcohol use (B = 0.13, 95% CI = 0.01 to 0.26). Lack of perseverance also modified the relation between emotional dysregulation and alcohol use (B = 0.21, 95% CI = 0.02 to 0.37), whereas positive urgency modified the association between cumulative lifetime stressor exposure and alcohol use (B = -0.05, 95% CI = -0.10 to -0.001).

Table 5.3 Inter-correlations (Spearman's rank values) of key study variables.

	1	2	3	4	5	6	7	8	9	10	11
1. Stressor Count (STRAIN)	-										
2. Stressor Severity (STRAIN)	.92**	-									
3. DERS-SF	.24**	.28**	-								
4. AUDIT	.32**	.34**	.41**	-							
5. Weekly Consumption (Units)	.22**	.26**	.24**	.69**	-						
6. SUPPS-P Negative Urgency	.21**	.25**	.63**	.38**	.24**	-					
7. SUPPS-P Perseverance	.08	.06	.21**	.16**	.00	.13*	-				
8. SUPPS-P Premeditation	.19**	.22**	.33**	.30**	.15*	.34**	.44**	-			
9. SUPPS-P Sensation Seeking	-.05	-.09	.09	.06	.03	.18**	-.17**	.06	-		
10. SUPPS-P Positive Urgency	.12*	.14*	.50**	.41**	.28**	.68**	.09	.36**	.39**	-	
11. BART	.06	.01	-.08	.02	.02	-.04	.05	.00	.12*	.03	-
12. 1 - AUC	.03	.05	.07	.07	.13*	.15*	-.05	.11	.01	.11	-.12*

Note. STRAIN = Stress and Adversity Inventory for Adults; DERS-SF = Difficulties in Emotional Regulation Scale Short Form; AUDIT = Alcohol Use Disorders Identification Test; 1 unit = 8g pure ethanol; SUPPS-P = Shortened Urgency, Premeditation (lack of), Perseverance (lack of), Sensation Seeking, Positive Urgency, Impulsive Behaviour Scale; BART = average number of space bar presses for unburst balloons during the Balloon Analogue Risk Task; 1 – AUC = 1 minus the area under the curve scores (greater scores reflect greater delay discounting) for the Titrating Alternatives Delay Discounting Task.

* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 5.4 Summary of the mediation analysis examining whether emotional dysregulation mediates the effect between cumulative lifetime stress and lifetime alcohol use (N = 279).

		Consequent									
		***M* (DERS-SF)**					***Y* (Alcohol Use)**				
Antecedent		**B**	**β**	**SE**	**LL**	**UL**	**B**	**β**	**SE**	**LL**	**UL**
Constant	i_M	**44.02**		**5.91**	**32.88**	**56.09**	i_Y 32.60		21.44	-6.34	78.27
X (STRAIN)	*a*	**0.15**	**0.34**	**0.03**	**0.10**	**0.20**	*c'* 0.13	0.12	0.07	-0.005	0.26
M (DERS-SF)		-	-	-	-	-	*b* **0.47**	**0.19**	**0.19**	**0.10**	**0.85**
Age		**-0.34**	**-0.31**	**0.06**	**-0.45**	**-0.22**	-0.06	-0.02	0.17	-0.39	0.27
Sex = Male		-0.56	-0.02	1.47	-3.45	2.32	6.74	0.10	3.91	-1.09	14.34
Education											
GCSE & below		Ref.					Ref.				
A-levels & equivalent		4.75	0.16	3.32	-1.86	11.28	**-17.89**	**-0.24**	**9.36**	**-37.40**	**-0.55**
Undergraduate & higher		2.75	0.10	3.08	-3.44	8.75	**-19.55**	**-0.28**	**9.07**	**-38.76**	**-3.00**
Employment											
Unemployed		Ref.					Ref.				
Student		5.42	0.16	3.77	-2.20	12.66	-6.21	-0.07	17.19	-43.48	21.81
Employed		4.89	0.15	2.96	-1.24	10.37	-12.38	-0.15	15.60	-47.21	11.06
Household Income											
Low		Ref.					Ref.				
Medium		-1.33	-0.03	2.21	-5.47	3.18	-1.93	-0.02	5.01	-11.29	8.37
High		-1.56	-0.05	1.79	-4.98	2.06	0.02	0.00	4.95	-9.23	10.12
Subjective Social Status											
Low		Ref.					Ref.				
Medium		-0.91	-0.03	2.18	-5.23	3.25	2.56	0.03	6.73	-9.77	16.66
High		-2.08	-0.08	1.91	-5.82	1.69	-0.01	0.00	4.73	-9.12	9.29
			R^2 = 0.22					R^2 = 0.10			
			$F(11, 267) = 7.13$, $p < .001$					$F(12, 266) = 2.64$, $p = .002$			

Note. Models were adjusted for age, sex, highest level of education achieved, employment status, and household income. LL and UL represent the lower and upper limit of the bootstrapped 95% CI (10,000 bootstraps), respectively. STRAIN = Stress and Adversity Inventory for Adults Stressor Severity Index; DERS-SF = Difficulties in Emotional Regulation Scale Short Form; 1 unit = 8g pure ethanol. Significant effects ($p < .05$) are in boldface.

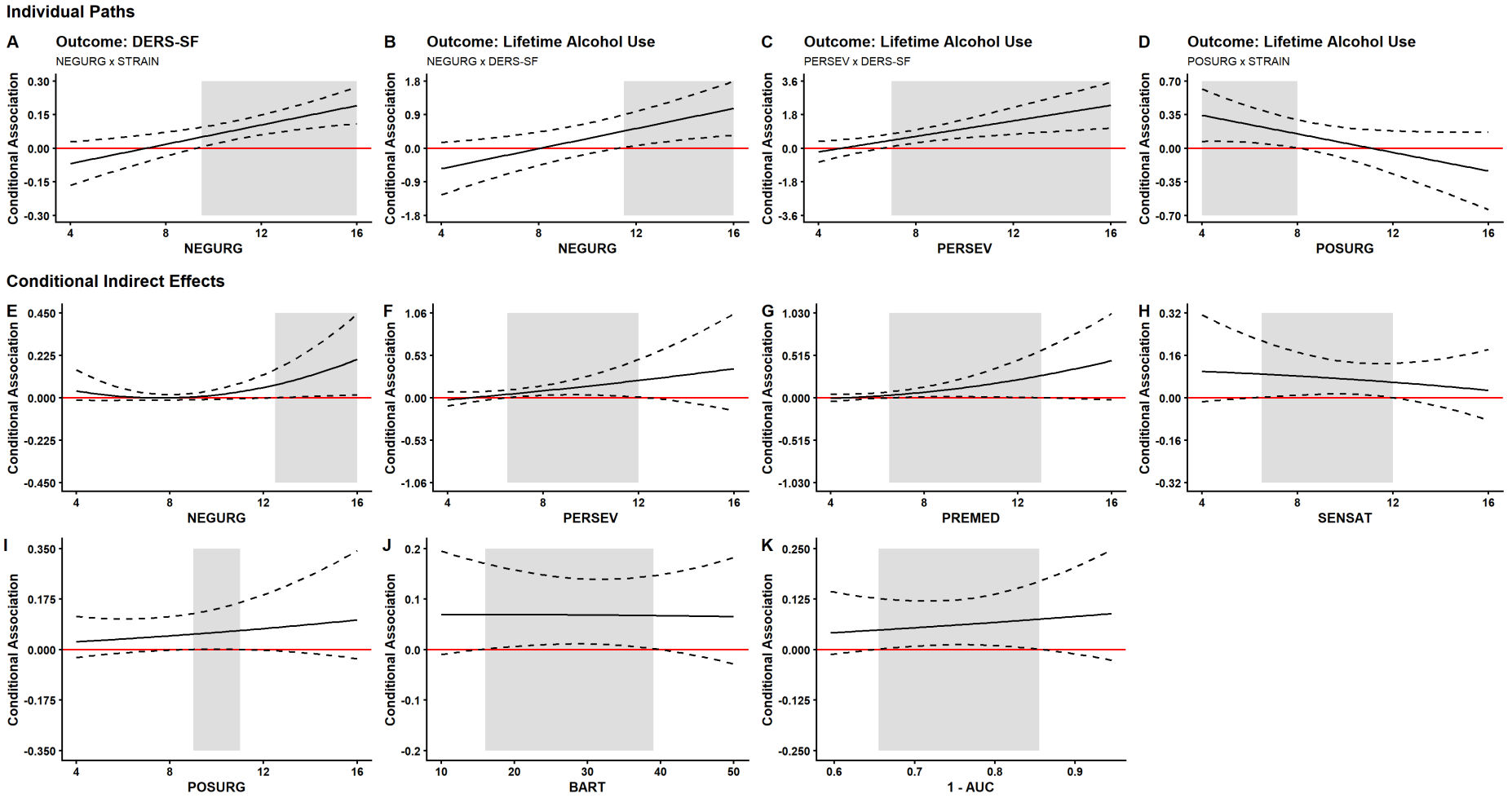
Johnson-Neyman plots (see Figure 5.2) revealed that associations between cumulative lifetime stressor exposure and emotional dysregulation, emotional dysregulation and alcohol use, and the indirect association (i.e., the relation between cumulative lifetime stressor exposure and lifetime alcohol use, through emotional dysregulation) were strengthened as negatively urgency increased from 9.5, 11.5 and 12.5, respectively. A similar pattern was observed for lack of perseverance (values ≥ 7) and the association between emotional dysregulation and alcohol use. However, the opposite was observed for positive urgency and the association between cumulative lifetime stressor exposure and alcohol use, where the relationship was weakened as positive urgency increased (values ≤ 8 were significant). In terms of other modified indirect effects (Figure 5.2, panels F–K), middling values tended to be significant. However, the slopes, as values of impulsivity increased, were relatively less steep. Therefore, these findings suggest that moderators in Figure 5.2 F–K were relatively less effective in predicting lifetime alcohol use.

5.4. Discussion

A theoretically–driven model of risk factors for lifetime alcohol use was tested in this study. Specifically, the aim of this study was to determine: (a) if emotional dysregulation mediates the relation between cumulative lifetime stressor exposure and lifetime alcohol use; and (b) whether these associations were strengthened by greater impulsivity, operationally defined in terms of both self-report and behavioural methods. Consistent with our pre–registered hypotheses, we found statistical evidence that emotional dysregulation fully mediated the association between cumulative lifetime stressor exposure and lifetime alcohol use, demonstrated by a significant indirect (ab) effect and non-significant direct effect (c). We also found that urgency (both negative and positive) and perseverance are crucial moderators of these associations. Contrary to our hypothesis, self-report premeditation and sensation seeking, and our behavioural measures of impulsivity, were less useful regarding the prediction of lifetime alcohol use.

The individual contributions of stressor exposure, emotional dysregulation and impulsivity to increased alcohol use are well established (Blaine & Sinha, 2017; Carbia et al., 2021). Consistent with this research, we found that greater self-reported impulsivity was independently related to increased

Figure 5.2 Johnson-Neyman plots illustrating the conditional process analysis results. Note. The shaded area represents the region of significance (p < .05).NEGURG = Negative Urgency; PERSEV = Lack of Perseverance; PREMED = Lack of Premeditation; SENSAT = Sensation Seeking; POSURG = Positive Urgency; BART = average number of space bar presses for unburst balloons during the Balloon Analogue Risk Task; 1 – AUC = 1 minus the area under the curve score (so that greater scores reflect greater delay discounting) for the Titrating Alternatives Delay Discounting Task; STRAIN = Stress and Adversity Inventory for Adults Stressor Severity Index; DERS-SF = Difficulties in Emotional Regulation Scale Short Form



AUDIT score (except sensation seeking) and lifetime alcohol use (except sensation seeking and perseverance). Similarly, we found that that behavioural delay discounting (1 – AUC) was associated with increased lifetime alcohol use. In contrast, behavioural risk-taking (BART) was not correlated with alcohol use behaviour.

The individual contributions of stressor exposure, emotional dysregulation and impulsivity to increased alcohol use are well established (Blaine and Sinha, 2017; Carbia et al., 2021). Consistent with this research, we found that greater self-reported impulsivity was independently related to increased AUDIT score (except sensation seeking) and lifetime alcohol use (except sensation seeking and perseverance). Similarly, we found that that behavioural delay discounting (1 – AUC) was associated with increased lifetime alcohol use. In contrast, behavioural risk-taking (BART) was not correlated with alcohol use behaviour.

There is clear evidence that stressor exposure causes emotional dysregulation (Sapolsky, 2007); that emotional dysregulation is greater in alcohol-dependent individuals (Sinha, 2012); and that impulsivity is a personality trait (Cyders et al., 2014), which is likely to manifest during development (Niv et al., 2012). To our knowledge, this is the first study to demonstrate that emotional dysregulation fully mediates the relation between cumulative lifetime stressor exposure and lifetime alcohol use. Moreover, it is the first to determine the different facets of impulsivity that moderate this mediated association. Collectively, this provides evidence that our variables of interest are temporally spaced, giving confidence that longitudinal follow-up studies would be likely to show similar results (Hayes, 2022). We argue that further testing of our model would lead to fruitful theoretical and, potentially, therapeutic advances. For instance, interventions which aim to improve emotional regulation may be beneficial in prevention and treatment efforts.

No studies have investigated mediated associations between stress, impulsivity, emotional dysregulation and alcohol use behaviour. However, Hamilton et al. (2013) tested multiple stress → impulsivity → hazardous drinking models, iterating over several stressor types, and found that self-report impulsivity partially mediated the relation between cumulative lifetime stress and alcohol use behaviour. Similarly, Kim et al. (2013) suggest that 'reflection impulsivity', 'response impulsivity', and

'aggression' *partially mediated* the association between early life stress and alcohol dependence. Here, we specified a cumulative lifetime stressor exposure → emotional dysregulation → lifetime alcohol use mode, providing evidence for *full mediation*. Finally, as Jakubczyk et al. (2018) found evidence that emotional dysregulation partially mediated the relation between AUD symptomology and increased impulsivity, it is probable that stressor exposure, emotion dysregulation and impulsivity are both risk-factors for, and consequences of, alcohol misuse. However, due to the cross-sectional design used in these studies, and here, it is impossible to determine directionality or causality. This should be an area of future research focus.

The interactive effects of impulsivity and acute (Clay et al., 2018; Clay and Parker, 2018), chronic (Clay et al., 2022, 2021), and cumulative (Fox et al., 2010) stress on alcohol use behaviour have been previously reported but not in the context of mediation. We found that the positive associations between cumulative lifetime stressor exposure and emotional dysregulation; emotional dysregulation and lifetime alcohol use; and cumulative lifetime stressor exposure and lifetime alcohol use, through emotional dysregulation were strengthened as values of negative urgency increased. Similarly, the association between emotional dysregulation and lifetime alcohol use was strengthened as (lack of) perseverance increased. Meanwhile – and as expected, given the pattern of negative urgency findings – the relation between cumulative lifetime stressor exposure and lifetime alcohol use became weaker as levels of positive urgency increased. Finally, all other measures of impulsivity, except sensation seeking and risk-taking (BART), strengthened the indirect effect. However, the moderation slopes were less steep (vs. negative urgency), and middling values tended to be significant. Therefore, we conclude that these measures are perhaps a less useful target for future research focus compared to negative urgency.

The biological mechanisms underlying these patterns remain unclear. However, several stress-related changes in biology could partly explain our findings. For instance, as stress exposure(s) cumulates over an individual's life, HPA axis sympathetic-adrenal-medullary axis, and systemic inflammatory activity is upregulated (Graham et al., 2006; Lupien et al., 2009), leading to increased allostatic load (i.e., biological 'wear and tear') (McEwen, 1998) and the associated risk for diseases, disorders and death (Lupien et al., 2009). People who begin to misuse alcohol may do so in an attempt

to ‘self-medicate’. However, as the hedonic effects wear off, their allostatic load is increased further by the distress of withdrawal, and overtime, after repeated binges, a change in their allostatic set-point leaves them vulnerable to alcohol misuse and related harm (Koob, 2001).

Furthermore, impulsivity and emotional dysregulation are thought to be partly heritable (Niv et al., 2012; Rappaport et al., 2020) and functional magnetic resonance imaging (fMRI) and event related potential (ERP) studies show that both emotional regulation and impulsivity share overlapping networks, situated predominantly in the prefrontal cortex (Brown et al., 2012; Messerotti Benvenuti et al., 2015). Therefore, it may be that those who are high in trait-impulsivity (particularly urgency) expend a great deal of cognitive resources on emotional processing, leaving limited resources for decision making (Jakubczyk et al., 2018; Seo et al., 2016). Ultimately, resulting in maladaptive decisions, such as alcohol misuse.

An alternative explanation is that, in line with the stress generation hypothesis (Hammen, 2006), those high in negative urgency tend to experience a greater number of negative dependent events (Liu and Kleiman, 2012). Similarly, negative urgency has been shown to moderate acute stress reactivity (Owens et al., 2018). Therefore, those high in negative urgency may exacerbate current, or generate new, stressful life events. Put differently, life may be more stressful for those high in negative urgency. This may help to explain why meta-analysis results show that negative urgency is one of the strongest impulsivity-related correlates of alcohol-related problems and dependence (Coskunpinar et al., 2013) and, in the present study, the model containing negative urgency explained 51% of the variance in emotional dysregulation and 14% of the variance in lifetime alcohol use. Therefore, interventions focussed on reducing negative urgency may prove useful.

5.4.1. Limitations

We acknowledge several limitations. First, although the cross-sectional mediation analysis provides initial support for our hypothesis, without complementary longitudinal analyses we cannot make firm conclusions regarding causality or temporal onset (Hayes and Rockwood, 2020). Second, due to technical limitations (i.e., having to pass participants between software systems), our measures were not fully counterbalanced (i.e., the STRAIN was always completed last). This may have caused

uncontrolled order effects. It should be noted, however, that measures were counterbalanced within blocks and the most cognitively demanding tasks (i.e., the behavioural computer tasks) were presented at the beginning of the study. Third, our stop-signal task data (see Appendix D) was unreliable and the psychometric properties of the BART have been questioned in prior research (Steiner and Frey, 2021). Finally, self-report measures are prone to measurement error owing to reliance on participants' accurate memory and/or response biases and demand characteristics. For example, individuals typically under-estimate their alcohol consumption during questionnaires (Northcote and Livingston, 2011) and self-report impulsivity measures may reflect self-identified behaviours rather than the construct that is intended to be assessed (Lane et al., 2003). Therefore, future research should focus on creating behavioural measurement of UPPS-P constructs, which would also enable subsequent translational (i.e., animal) research.

5.4.2. Conclusion

The present cross-sectional scoping study extends prior research by testing a theoretically driven model of alcohol use. We found evidence to suggest that individuals who have higher cumulative lifetime stressor exposure tend to have higher alcohol use due to also having higher levels of emotional dysregulation. Furthermore, these relations were stronger in those with high negative urgency. These findings have important implications for both researchers and clinicians. For instance, highlighting clinical utility for lifetime stress exposure screening and identifying potential targets for personalised treatment interventions for alcohol misuse which focus on improving emotional regulation and reducing negative urgency.

Chapter 6. General Discussion

6.1. Overview of thesis

The overall aim of this thesis was to test the hypothesis that poor impulse control would increase the impact of stress on alcohol use. In other words, it was hypothesised that stress would cause increases in alcohol use behaviour, and that this effect would be greater among those who were more impulsive. This work builds on previous research which demonstrated that in times of acute psychosocial stress (i.e., following the TSST), risky decision making (i.e., risk–taking) strengthens stress–induced alcohol craving and consumption (Clay et al., 2018; Clay & Parker, 2018). Specifically, the hypothesis that the impact of stress on alcohol use behaviour is strengthened by impulsivity was tested across several studies in the context of acute, chronic, and cumulative lifetime stress.

A range of methods were implemented to accomplish the overarching thesis aim. For instance, earlier work (Clay et al., 2018; Clay & Parker, 2018) was extended in Chapter 2 by comparing drinking behaviour in response to acute physical (i.e., pain), psychosocial, and mixed (i.e., pain + psychosocial) stressors, and by investigating the influence of impulse control on stress–induced craving and consumption of alcohol in each condition.

In Chapter 3 and Chapter 4, the COVID–19 pandemic and associated "lockdowns" (i.e., unprecedented periods of social isolation; Anderson et al., 2020) were used to investigate the central hypothesis of this thesis during *chronic stress*. In other words, the lockdowns represented lengthy periods of naturally occurring stress for many (e.g., Bhattacharjee & Acharya, 2020; Gavin et al., 2020; Ornell et al., 2020; Pfefferbaum & North, 2020), and could therefore be utilised by stress researchers interested in the impact of chronic stress on health behaviour (Arora & Grey, 2020). More specifically, in Chapter 3, large datasets collected from four nationally representative birth cohorts (the Millennium Cohort Study, aged 19; Next Steps, aged 30; the 1970 British Cohort Study, aged 50; and the National Child Development Study, aged 62) were exploited. While a strength of this work was the large sample size and excellent generalisability (due to the data being nationally representative), a limitation was that the assessments of stress and impulsivity used in this study were single–item measures. As such, these measures may not capture the entirety of these constructs. Therefore, in Chapter 4, two studies which utilised commonly used, standardised, and psychometrically valid procedures were carried out: in the

first study, cross–sectional data from the first wave of the pandemic (07 April – 03 May 2020) was collected and analysed, and in second study, a subgroup was followed up until September 2020. Here, in addition to the primary aim of this thesis, the role of boredom and drinking motives in pandemic–related drinking was also investigated. Furthermore, the efficacy of a personal feedback intervention (i.e., a summary of self–report drinking behaviours and consequences, normative comparisons, and information about strategies to reduce drinking) was tested.

Finally, in Chapter 5, the main hypothesis of this thesis was tested and extended in the context of *cumulative lifetime stress* using conditional process (i.e., moderated mediation) analysis. Specifically, it was hypothesised that the association between cumulative lifetime stressor exposure and lifetime alcohol use would be mediated by emotional dysregulation, and that increased impulsivity would strengthen these relationships.

6.2. Summary of key findings from each study

6.2.1. Acute stress

The experimental laboratory study reported in Chapter 2 aimed to assess the impact of impulse control on craving for, and consumption of, alcohol following either: acute pain (CPT), acute psychosocial stress (TSST), or an acute mixed stressor (MAST). The first hypothesis in this study predicted that acute stress exposure would increase craving and reduce the time to consume an alcoholic beverage (50mL of 37.5% ABV vodka diluted with 250mL of mixer). The second hypothesis predicted that more impulsive individuals would show higher levels of stress–induced craving and drinking. However, the data in Chapter 2 did not support the first hypothesis as statistically significant between–group differences in craving and drinking time were not detected. This may have been due to the CPT and MAST stress manipulations not eliciting a sufficiently large stress response. This was despite previous work demonstrating that both the CPT (e.g., Velasco et al., 1997) and MAST (e.g., Smeets et al., 2012) reliably activate the HPA system. Therefore, the exact procedural parameters which elicit a robust stress response in acute stress tasks which are commonly used in laboratory–based alcohol research should be determined. Nevertheless, in the TSST group, where a robust stress response was observed, poor impulse control (specifically negative urgency) and increased negative affect were associated with

increased levels of alcohol craving as in previous work (e.g., Bresin et al., 2018; Clay et al., 2018). However, craving was not significantly associated with consumption, despite being a hallmark feature of addiction (e.g., Addolorato et al., 2005). Therefore, as the sample in Chapter 2 were non–dependent drinkers, craving may only be an important predictor of alcohol use behaviour among those who drink excessively. Taken together, the results arising from Chapter 2 highlight the importance of negative urgency (i.e., the tendency to act rashly under extreme negative emotions) and negative affect (i.e., the feeling that occurs when one is not satisfied with the present circumstances) in understanding stress–induced alcohol craving.

6.2.2. Chronic stress

The studies on chronic stress (i.e., Chapter 3 and Chapter 4) revealed that a minority of participants reported consuming more alcohol and feeling more stressed during the first wave of the COVID–19 pandemic. Stress and impulsivity, however, were independently associated with increased alcohol use behaviour in the birth cohort study (Chapter 3), and in Chapter 4, where more precise measures were used, increased pandemic–related alcohol use behaviour was driven by increased levels of impulsive traits (i.e., risk–taking, sensation seeking, and lack of premeditation) and the tendency to drink to cope with negative affect. In both chapters, however, the direction of the negative affect and personality interactions went in the opposite direction to that which was predicted in the primary hypothesis of this thesis. More specifically, those who reported less stress (Chapter 3) or lower levels of boredom (Chapter 4) *and* were more impulsive were more likely to reported increased pandemic drinking behaviour. This may reflect that chronic stress interacts with impulsivity via completely different mechanisms than acute stress generally. Alternatively, it may be that COVID–19–related stress was qualitatively different to other life–stressors. Thus, future research should aim to understand and compare how different types of chronic stress (e.g., adverse childhood experiences vs. social isolation) may alter the pathway to alcohol misuse and addiction.

6.2.3. Cumulative lifetime stress

The cumulative effect of stress over the life course is known to be detrimental to emotional regulation ability (e.g., Birditt et al., 2023; Zeier et al., 2022). Furthermore, evidence shows that repeated stressor

exposure results in neurophysiological changes in areas related to the processing of emotions and stress reactivity (e.g., Casement et al., 2015; Kim et al., 2013). Most theories attempting to understand what drives substance misuse – from relatively simple historical explanations, such as the self–medication hypothesis (for review, see: Khantzian, 1997), to more modern and sophisticated frameworks such as the Etiologic, Theory–based, Ontogenetic Hierarchical (ETOH) Framework (for review, see: Boness et al., 2021) – include self–regulation of negative emotions as a core component. Therefore, the primary hypothesis of this thesis was extended in Chapter 5 to investigate the role of emotional dysregulation in the link between cumulative lifetime stressor exposure and lifetime alcohol use. Specifically, it was hypothesised that cumulative lifetime stressor exposure would decrease the ability for individuals to regulate their own emotions, which would lead to increased lifetime alcohol. It was also hypothesised that poor impulse control would exacerbate these effects. The data in Chapter 5 supported these predictions. Importantly, however, negative urgency was the only facet of impulsivity to strengthen all pathways; in other words: (1) the link between stress and emotional dysregulation; (2) the relationship between emotional dysregulation and lifetime alcohol use; and (3) the indirect of stress on alcohol use via emotional dysregulation. Therefore, similar to the results presented in previous chapters, trait impulsivity – specifically negative urgency (Chapter 2) – and the ability to cope with negative emotionality (Chapter 2 and Chapter 4) are critical in understanding alcohol misuse and addiction.

6.3. Implications

6.3.1. Theoretical implications

The importance of clearly characterising the multifaceted nature of both impulsivity and stress in future research cannot be understated. For instance, cumulative knowledge generation on both constructs are beset by *"jingle"* and *"jangle"* fallacies (Block, 1995); where the jingle fallacy refers to when two separate constructs are given the same name, and the jangle fallacy refers to the same construct being given different names. In other words, simply understanding that both stress and impulsivity are risk–factors for alcohol misuse and addiction is naïve at best and ignorant at worst. For instance, it is clear that the experience of stress is modified by both the timescale and severity of the stressor (Crosswell & Lockwood, 2020; Epel et al., 2018). Similarly, it is also clear that impulsivity covers a plethora of traits

and behaviours that are sometimes not even statistically correlated with each other (Dalley & Robbins, 2017; Strickland & Johnson, 2020). Indeed, the between–study heterogeneity of results reported in this thesis provide evidence that a nuanced understanding of both stress and impulsivity is required. Moreover, following the research reported in this thesis, it is clear that impulsivity and stress interactions are crucial in understanding the transition from alcohol use, to misuse, to addiction. In particular, aspects of personality which are related to the processing of negative emotions, such as negative urgency (Chapter 2 and Chapter 5), negative affect (Chapter 2, Chapter 3, and Chapter 4), and emotional dysregulation (Chapter 5) are demonstrably important.

Figure 6.1 A theoretical model of stress–induced alcohol use based on the results of this thesis, integrating aspects of negative emotionality and impulsivity.

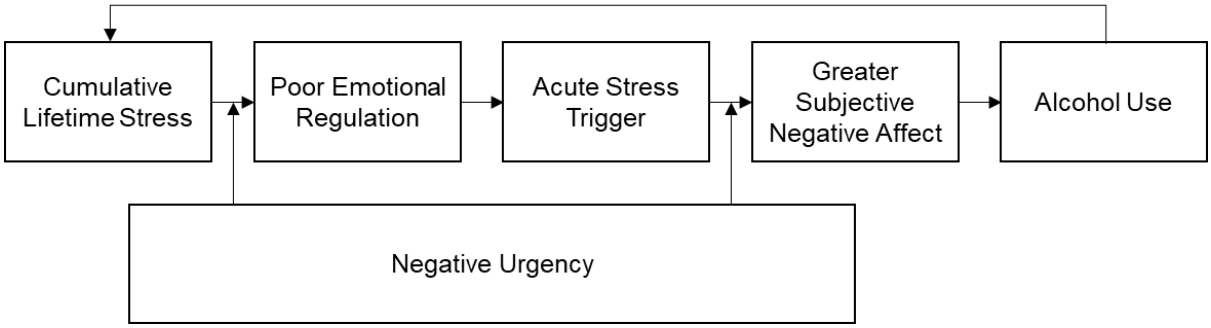


Figure 6.1 illustrates how these factors may fit together into an integrated model of testable hypotheses. To build this model, the original model presented in Chapter 5 is extended and refined, whereby cumulative lifetime stressor exposure results in lower abilities to regulate emotion. In turn, individuals who are unable to self–regulate experience greater levels of subjective negative affect following exposure to acute stress, and this heightened level of negative affect is what ultimately drives increased alcohol use behaviour (e.g., binge–intoxication in non–dependent drinkers and heightened craving, and ultimately drinking, among heavy drinkers). Importantly, negative urgency is hypothesised to strengthen the links between cumulative lifetime stressor exposure and emotional dysregulation (see Chapter 5), and between the acute stress triggers and the experience of high levels of negative affect (see Chapter 2 and Owens et al., 2018). Finally, based on evidence which suggests that chronic HPA axis activation – resulting from increased cumulative lifetime stress and alcohol misuse in this case –

causes neurophysiological changes in stress and reward pathways (Koob & Kreek, 2007; Koob & Schulkin, 2019; Milivojevic & Sinha, 2018), a reciprocal relationship between alcohol use and increased cumulative lifetime stress is specified, thus also (in part) representing the cycle / spiral of addiction.

6.3.2. Clinical implications

Current treatments for AUDs are only modestly effective (Ray et al., 2019). However, most existing treatments for addictive disorders typically focus on proximal factors of substance use (e.g., drinking motives and drinking contexts), rather than the distal factors which underlie them, such as trait impulsivity (Kiluk et al., 2010; Magill et al., 2015). Therefore, novel (endo)phenotype specific (i.e., personalised) interventions may pave the way to more favourable treatment outcomes. Such interventions could plausibly be created for any one of the factors outlined in Figure 6.1. However, some are likely to be more practicable than others. For instance, trying to limit the number of stressful events that someone is exposed to would require significant and intrusive control over their lives. Instead, focusing on reducing the impact of emotional dysregulation or negative urgency may be more beneficial as the individual receiving the intervention will be better equipped to deal with the inevitable experience of future stress.

At first glance, the idea that giving an individual the tools needed to self–regulate their emotional reactions is not new. After all, the theory underlying cognitive behavioural therapy is that cognitions and behaviour are both the cause of, and the means to resolve, emotional dysregulation (Lee et al., 2021). However, meta–analysis results show that such interventions are still only moderately effective when treating substance use disorders (pooled effect, $g = 0.15$), with diminishing returns over time (6–9 month follow–up, $g = 0.12$; 12–month follow–up, $g = 0.10$) (Magill & Ray, 2009; McHugh et al., 2010). Therefore, it may be better for future interventions to target negative urgency instead (Halcomb et al., 2019).

Negative urgency reflects the between–person variation in the ability to self–regulate intense emotionally–driven urges (Segerstrom & Smith, 2019). It is only moderately correlated with measures of emotional regulation (Cyders & Smith, 2008), thus both negative urgency and emotional regulation

likely reflect separate constructs. Results from a recent meta–analysis suggest that high negative urgency predicts lower treatment effectiveness and that current treatments barely reduce negative urgency (Hershberger et al., 2017). Therefore, novel treatments which focus on reducing the impact of negative urgency may prove to be more efficacious than existing treatments which focus on either proximal factors of addiction or emotional regulation, specifically. This idea is further strengthened by the fact that as impulsivity reduces with age due to maturation, so does substance use (Littlefield et al., 2009). In other words, as a natural reduction in impulsivity seems to reduce substance use, interventions which target specific impulsive traits, such as negative urgency, are likely to be effective. Furthermore, several studies suggest that both coping and enhancement motives mediate the negative urgency–alcohol use relationship (e.g., Adams et al., 2012; Menary et al., 2015; Wolkowicz et al., 2021). Therefore, future interventions which successfully reduce negative urgency may also reduce the influence of proximal factors (e.g., drinking motives) on alcohol use outcomes. Finally, as empirical evidence suggests that negative urgency is a transdiagnostic trait that is implicated in many psychopathologies, which are often comorbid with addiction (e.g., depression and anxiety) (e.g., Berg et al., 2015), new interventions focussed on reducing negative urgency may be beneficial elsewhere other than in addiction treatment. However, before effective treatments can be developed to reduce negative urgency, translational measures, which can be applied in both humans and animals, must be created (Halcomb et al., 2019). These models will allow researchers to characterise the neurobiological and genetic underpinnings of negative urgency as well as testing novel pharmacological compounds.

6.4. Limitations

It is important to consider the methodological limitations of the empirical chapters presented in this thesis when appraising this body of work, and to take the impact of such limitations on the general outcomes of this thesis into account. Alongside the study–specific limitations described in each chapter, there are two key limitations which are relevant to the thesis as a whole: measurement error and sampling error. Both are commonplace in psychological research (e.g., Henrich et al., 2010; Schmidt & Hunter, 1996).

Measurement error refers to the difference between a recorded quantity and its true value (Schmidt & Hunter, 1996). All assessments, including objective measures (e.g., the N–ray blunder in physics Nye, 1980) suffer from some degree of measurement error. In psychology, measures are often completed by the participants themselves, thus they are affected by (for example): mood, fatigue, specific knowledge about a procedure, social desirability bias, fluctuations in memory and performance. Such measures (e.g., self–report questionnaires and behavioural data) were used in each of the studies reported in this thesis. However, care was taken to select psychometrically valid measures during primary data collection. Similarly, during analysis of secondary data (i.e., in Chapter 3), the validity of single–item measures was considered by comparing scores against validated assessments. Furthermore, the internal consistency (for questionnaire–based assessments), inter–rater reliability (for behavioural drinking data), and reliability data from computer tasks (e.g., the SST) were calculated and assessed where appropriate. Therefore, taken together, substantial attention was taken when selecting measurements, though some degree of measurement error is likely to have occurred.

Sampling error is defined as the difference between an estimated parameter and the true population value (Holliday, 2014). In other words, if a sample does not represent the population of interest, estimated parameters are unlikely to generalise outside of the sample (Henrich et al., 2010). As the samples in several of the studies presented in this thesis were self–selecting, and therefore likely non–representative of the UK population as a whole, there are constraints on generality which must be considered when evaluating these findings (Simons et al., 2017). This was a particular issue in Chapter 2 where data collection was hindered by the onset of the COVID–19 pandemic and – like much of psychology, cognitive science, and economics research (Henrich et al., 2010) – our recruitment pool was mostly limited to students at the host institution (in this case, the University of Portsmouth). An incentive of either course credits or £15 was offered to participants who completed the study reported in Chapter 2 to try and bolster recruitment. Ultimately, however, it remains difficult to recruit participants for laboratory studies (Patel et al., 2003). Thus, finding ways to translate similar research designs to utilise online research methods (e.g., Chapter 3 and Chapter 5), secondary data analyses (e.g., Chapter 3), and high–throughput animal work may have utility.

There was a trade–off between large nationally representative samples with less precise measures or smaller, less representative samples with more precise measures. For instance, the data analysed in Chapter 3 were nationally representative however the measures were single–item. Therefore, we can be relatively confident that these data generalise to the wider UK population. However, we can be less confident in the quality of the measures. On the other hand, the data analysed in Chapter 4 were more nuanced and precise than that reported in Chapter 3. However, the sample was WEIRD (i.e., western, educated, industrialised, rich and democratic; Henrich et al., 2010). Therefore, we can be confident that there is relatively less measurement error associated with the assessments used in Chapter 4, but less confident in the generalisability of these results. Nationally representative longitudinal data focus on risk–factors for alcohol misuse and addiction would provide a solution to both issues. However, such data does not currently exist. Thus, this should be a focus of future research efforts.

6.5. Future directions

The results presented in this thesis demonstrate the role of impulse control on alcohol use in the context of acute, chronic, and cumulative lifetime stress. In particular, these results highlight the importance, and potential clinical utility, negative urgency and emotional regulation abilities (see Figure 6.1 for example). However, several unanswered questions also arose from this thesis. A summary of selected open questions that can be used as a guideline for future research are shown in Table 6.1.

Most of these questions are underpinned by a need for methodological precision alongside longitudinal, nationally representative data. For instance, how key methodological choices made during the research design process (e.g., the alcohol consumption task used to assess drinking behaviour) affect study results and conclusions is not currently clear. More specifically, for example, would the conclusions made based on results from a laboratory study utilising a progressive ratio schedule of reinforcement differ from those obtained using an ad libitum alcohol consumption task, all other things being equal?

Table 6.1 Selected open questions originating from this work.

Open Questions
Procedural questions
● What are the exact procedural parameters required to elicit a robust stress response in acute stress tasks which are commonly used in laboratory–based alcohol research?
● How do laboratory–based drinking paradigms (e.g., a progressive ratio schedule vs. an *ad libitum* alcohol consumption task) affect study results?
Clinical questions
● Can effective (endo)phenotype specific treatments (either pharmacological or psychotherapeutic) be developed?
● Can sophisticated statistical modelling techniques (e.g., machine learning) be a cost–effective solution to personalised diagnosis and management?
Theoretical questions
● At which point of the cycle of addiction does alcohol craving become an important component?
● Are the effects reported here and elsewhere in the literature generalisable to hard–to–reach populations, such as heavily dependent drinkers?
● Do the results reported here hold in "real world" settings, and if so, how do contextual factors (e.g., daily stressors, drinking contexts) effect this?
● How does the type of chronic stress experienced (e.g., childhood adversity vs. pandemic–related social isolation) alter the pathway to alcohol misuse?
● Can novel behavioural measures represent UPPS–P constructs be created to allow high–throughput translational (i.e., animal) work to be conducted in this area?

Speaking to the point of the need for longitudinal, nationally representative data, we are currently unable to know whether the results presented in this thesis will generalise to either hard–to–reach populations (e.g., socioeconomically disadvantaged groups; Bonevski et al., 2014) or "real world" settings (e.g., offline and outside of the laboratory). Furthermore, as the data in Chapter 5 are not longitudinal, causal inference is precluded. Nationally representative longitudinal data would help to overcome both of these limitations. First, non–response and study attrition are associated with very low or very high levels of alcohol consumption (Torvik et al., 2012). Thus, the national representation and the derivation of population weights (to account for attrition; Seaman & White, 2013) would allow confident conclusions about hard–to–reach populations to be made. Second, longitudinal data will facilitate causal claims (Raudenbush, 2001). However, such longitudinal data typically utilise less nuanced measurements (Holditch-Davis & Levy, 2010). Therefore, such analyses are not currently possible. Thus, going forward, a longitudinal study focused on addiction is warranted.

The creation and validation of behavioural measures of UPPS–P constructs, which can be translated for use between humans and animals, is also urgently required (Halcomb et al., 2019). This would allow the relevant neurobiological and genetic pathways associated with UPPS–P constructs to be characterised. Subsequently, novel treatments for addition–related UPPS–P factors (e.g., negative urgency) can then be created and tested.

As a final note, recent technological advances, such as the near universal smartphone ownership, and machine learning techniques, may offer new avenues of investigation. For instance, smart phones allow for ecological momentary assessments (i.e., repeated, real–time measurements) to be carried out (Perski et al., 2022). Thus, investigations of alcohol use behaviours in the context of day–to–day life can be investigated. Thus far, such studies suggest that moment–to–moment fluctuations in state impulsivity predict alcohol outcomes (Halvorson et al., 2020; Stamates et al., 2019), and similar to the results presented here, that negative urgency is of particular importance (Halvorson et al., 2020). Therefore, future research should aim to test the main hypothesis of this thesis in the context of daily stress to investigate whether moment–to–moment fluctuations in impulsivity *and* stress interact to

predict alcohol use behaviour. Similarly, machine learning could be used to assist in the investigation and delivery of precision medicine going forward (Wilkinson et al., 2020).

6.6. Conclusions

Some researchers have suggested that that the use of psychoactive drugs is intrinsic to most i.e., *"humans have an innate desire to get high"* (Carney, 2016), and alcohol consumption can cause pleasurable effects, such as relaxation, inhibition, and euphoria (Paton, 2005). Furthermore, drinking alcohol is woven into the fabric of many cultures and alcohol use is typically socially accepted (Hanson, 2013). Unsurprisingly, therefore, alcohol is one of the most popular psychoactive drugs throughout the world (World Health Organization, 2018a). However, alcohol use does not come without risk. For instance, alcohol is a causal factor in over 200 diseases and conditions (World Health Organization, 2018a), and chronic, unmanaged alcohol misuse can develop into a full–blown addiction (e.g., Saunders et al., 2019).

The present thesis utilised both primary and secondary data to investigate the role of impulse control on alcohol use in the context of acute, chronic, and cumulative lifetime stress. This programme of work advances the field in multiple ways. First, the research presented in this thesis is the first to demonstrate stress x impulsivity interactions across multiple contexts. Second, based on the results of this thesis, a novel and theoretically–driven model, which incorporates several previously identified risk–factors for alcohol misuse, is presented. Third, this programme of research highlights the importance of negative urgency and emotional regulation in the relationship between stress and alcohol use and makes suggestions for the creation and testing of novel interventions. Finally, this thesis demonstrates both the strengths and limitations of longitudinal birth cohort data, recommending that nationally representative, longitudinal, and addiction–specific data would pave the way for decades of fruitful research.

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Appendix A: Chapter 2 Supplementary Materials

Favourable Ethical Approval

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2 Nov 2020

FAVOURABLE ETHICAL OPINION – NOTIFICATION OF SUBSTANTIAL AMENDMENT

Study Title: Investigating cognitive factors related to alcohol use.

Reference Number: SHFEC 2019-123A

Date Submitted: 2 Nov 2020

Thank you for submitting your proposal amendment to the Science and Health Faculty Ethics Committee (SHFEC) for ethical review in accordance with current procedures.

I am pleased to inform you that SHFEC was content to grant a favourable ethical opinion of this proposal amendment on the basis described in the submitted documents listed at Annex A, and subject to standard general conditions (*See Annex B)*.

Please note that the favourable opinion of SHFEC does not grant permission or approval to undertake the research. Management permission or approval must be obtained from any host organisation, including the University of Portsmouth or supervisor, prior to the start of the study.

Wishing you every success in your research

Dr Paul Morris
Chair, Science and Health Faculty Ethics Committee

Annexes

A - Documents reviewed
B - After ethical review - Guidance for researchers

Stop Signal Task Descriptive Statistics

We report several task-related descriptive statistics in Table a1, following "A consensus guide to capturing the ability to inhibit actions and impulsive behaviors in the stop-signal task" (Verbruggen et al., 2019). P(Response|Signal) should ≈ 0.50 (Band et al., 2003) and, at the very least, individual SSRTs should not be estimated when P(Response|Signal) is lower than 0.25 or higher than 0.75 (Congdon et al., 2012). Applying this rule in the present study resulted in 45.79% of the data being excluded, suggesting that the SST data were unreliable for a significant number of the participants. We did not include this measure in our analyses due to the questionable reliability of the data in the present study and the reduction in sample size and statistical power following the exclusion of the unreliable data.

Table A1. Stop Signal Task descriptive statistics (*M* and *SD*).

Variable	**Total (SD)**	**Female (SD)**	**Male (SD)**
P(Response|Signal)	0.35 (0.22)	0.34 (0.22)	0.37 (0.23)
P(No Response|Go Trial)	0.08 (0.14)	0.09 (0.18)	0.06 (0.04)
P(Choice Error|Go Trial)	0.01 (0.02)	0.01 (0.03)	0.01 (0.02)
M RT on Go Trials	630.24 (94.37)	627.55 (96.95)	634.27 (91.38)
Intra-subject *SD* for *M* RT on Go Trials	130.61 (24.73)	127.60 (24.16)	135.12 (25.17)
Mean Stop Signal Delay	152.62 (24.73)	149.69 (73.28)	157.14 (74.73)
Mean RT for Unsuccessful Stop Trials	497.57 (60.88)	497.61 (59.45)	497.51 (63.70)

Taste Test Procedure

Phenylthiocarbamide (PTC) impregnated taste test strips (Breckland Scientific, UK) and 1M glucose solution (Iatridi et al., 2019) were used to quantify each participant's ability to detect and preference for bitter and sweet tastes, respectively. Before each taste test, participants were asked to drink a small (40ml) glass of chilled water. Following each taste test, participants completed two visual analogue scales anchored with "no sensation" and "strongest imaginable sensation of any kind" or "strongest imaginable unpleasant experience of any kind" and strongest imaginable pleasant experience of any kind". Here, participants were instructed to "consider each rating scale across all sensory domains (i.e., touch, taste, smell, sight, and hearing).

Supplementary Results

Table A2. Descriptive statistics (*M* and *SD*) for stress and craving data by timepoint and group.

	Timepoint		
Variable	**T1**	**T2**	**T3**
		TSST (*n* = 22)	
SBP	122.32 (13.14)		125.82 (12.67)
DBP	70.73 (9.52)		77.09 (7.67)
MAP	87.75 (9.01)		93.17 (8.74)
HR	81.26 (10.45)	95.17 (10.89)	82.22 (9.74)
SDNN	110.06 (109.33)	90.50 (34.69)	101.22 (38.23)
RMSSD	94.18 (169.27)	31.81 (14.65)	43.60 (22.49)
Positive Affect (PANAS)	30.41 (8.52)		26.68 (9.19)
Negative Affect (PANAS)	12.55 (2.58)		18.77 (7.05)
State Anxiety (STAI)	8.91 (2.24)		14.09 (3.99)
Craving (DAQ)	29.09 (6.60)		31.13 (9.14)
		MAST (*n* = 31)	
SBP	117.97 (13.64)		120.97 (13.69)
DBP	69.23 (7.35)		73.96 (8.35)
MAP	85.31 (7.92)		89.48 (8.63)
HR	84.59 (84.14)	84.14 (12.67)	81.68 (14.61)
SDNN	88.51 (37.35)	86.10 (27.82)	114.04 (41.92)
RMSSD	51.35 (29.15)	49.02 (20.69)	62.05 (27.93)
Positive Affect (PANAS)	30.89 (6.57)		27.94 (7.17)
Negative Affect (PANAS)	12.19 (2.34)		14.10 (4.56)
State Anxiety (STAI)	9.19 (2.36)		11.74 (3.61)
Craving (DAQ)	28.87 (6.99)		28.87 (7.07)
		CPT (*n* = 24)	
SBP	124.08 (13.89)		121.29 (17.60)
DBP	74.08 (10.32)		74.25 (12.88)
MAP	90.58 (10.88)		89.77 (13.75)
HR	89.14 (13.34)	83.07 (11.67)	81.69 (11.52)
SDNN	84.13 (43.09)	80.55 (33.51)	103.27 (39.64)
RMSSD	46.88 (26.35)	48.39 (25.34)	52.79 (27.83)
Positive Affect (PANAS)	27.04 (7.47)		26.67 (8.60)
Negative Affect (PANAS)	13.46 (3.07)		15.25 (8.35)
State Anxiety (STAI)	11.42 (2.41)		12.41 (4.34)
Craving (DAQ)	26.29 (6.36)		27.17 (5.83)
		Control (*n* = 30)	
SBP	124.77 (14.19)		114.97 (11.40)
DBP	72.9 (7.99)		70.33 (8.90)
MAP	89.73 (7.76)		85.06 (8.59)
HR	83.66 (15.27)	77.62 (13.18)	80.93 (13.55)

SDNN	78.96 (28.51)	79.08 (26.68)	104.90 (40.02)
RMSSD	47.07 (27.59)	50.18 (29.65)	50.31 (29.58)
Positive Affect (PANAS)	27.73 (6.56)		24.60 (6.89)
Negative Affect (PANAS)	13.23 (2.92)		11.90 (2.56)
State Anxiety (STAI)	10.07 (2.29)		10.17 (2.42)
Craving (DAQ)	27.20 (4.14)		27.77 (5.71)

Note. TSST = Trier Social Stress Test; MAST = Maastricht Acute Stress Test; CPT = Cold Pressor Task SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HR = Heart Rate (BPM) = heart rate; SDNN (ms) = standard deviation of normal–to–normal intervals; RMSSD (ms) = root mean square of successive differences between normal heartbeats; PANAS = Positive Negative Affect Schedule; STAI = State–Trait Anxiety Inventory; DAQ = Desires for Alcohol Questionnaire; T1 = baseline measurement; T2 = measurement taken during the manipulation; T3 = measurement taken immediately after manipulation.

Table A3. Group–wise correlations (Spearman's rank values) between impulsivity, stress and craving (change in Desires for Alcohol Questionnaire Score).

		Control	CPT	TSST	MAST
SUPPS-P Negative Urgency	*r*	.00	-.12	.46	.22
	p-value	0.981	0.586	0.030	0.230
SUPPS-P Premeditation	*r*	.23	-.32	.40	.24
	p-value	0.221	0.126	0.062	0.186
SUPPS-P Perseverance	*r*	.06	-.18	.26	.29
	p-value	0.734	0.392	0.238	0.107
SUPPS-P Sensation Seeking	*r*	-.11	.07	-.50	-.31
	p-value	0.564	0.736	0.017	0.086
SUPPS-P Positive Urgency	*r*	.03	-.16	-.19	.04
	p-value	0.864	0.442	0.395	0.844
BART	*r*	.19	.06	.29	-.03
	p-value	0.311	0.788	0.198	0.890
1 - AUC	*r*	-.14	.15	-.06	-.06
	p-value	0.469	0.470	0.775	0.735
MAP Reactivity	*r*	.02	.09	.19	-.07
	p-value	0.899	0.690	0.392	0.702
HR Reactivity	*r*	-.19	.18	.32	.05
	p-value	0.315	0.411	0.146	0.776
HR Recovery	*r*	-.15	-.12	-.23	-.22
	p-value	0.440	0.580	0.293	0.226
PA Reactivity	*r*	.07	-.12	.00	.00
	p-value	0.699	0.586	0.995	0.980
NA Reactivity	*r*	.00	-.18	.54	.03
	p-value	0.997	0.403	0.009	0.880

Note. TSST = Trier Social Stress Test; MAST = Maastricht Acute Stress Test; CPT = Cold Pressor Task; SUPPS-P = The Shortened Urgency, Premeditation (lack of), Perseverance (lack of), Sensation Seeking, Positive Urgency, Impulsive Behaviour Scale; BART = average number of space bar presses for unburst balloons during the Balloon Analogue Risk Task; 1 – AUC = 1 minus the area under the curve score (so that greater scores reflect greater delay discounting) for the Titrating Alternatives Delay Discounting Task; MAP = mean arterial pressure; HR = heart rate; PA = positive affect; NA = negative affect.

Table A4. Group–wise correlations (Spearman's rank values) between impulsivity, stress, craving and drinking (total drinking time).

		Control	CPT	TSST	MAST
DAQ Change	*r*	-.18	-.13	.19	.35
	p-value	.352	.558	.408	.057
SUPPS-P Negative Urgency	*r*	.02	-.43	-.04	.23
	p-value	.937	.034	.852	.206
SUPPS-P Premeditation	*r*	.12	-.04	.22	.08
	p-value	.537	.844	.320	.673
SUPPS-P Perseverance	*r*	.08	-.04	.43	.12
	p-value	.669	.846	.048	.512
SUPPS-P Sensation Seeking	*r*	-.22	-.35	-.11	-.20
	p-value	.243	.093	.631	.284
SUPPS-P Positive Urgency	*r*	.03	-.24	-.39	.00
	p-value	.892	.261	.074	.994
BART	*r*	-.26	.13	-.08	-.13
	p-value	.171	.538	.710	.499
1 - AUC	*r*	.21	-.09	-.13	-.16
	p-value	.261	.665	.577	.393
MAP Reactivity	*r*	.14	.13	-.23	.00
	p-value	.451	.552	.306	.988
HR Reactivity	*r*	.30	.30	-.02	.00
	p-value	.110	.156	.930	.979
HR Recovery	*r*	-.19	.24	-.15	.13
	p-value	.326	.250	.519	.501
PA Reactivity	*r*	-.18	.48	-.28	-.46
	p-value	.336	.017	.205	.009
NA Reactivity	*r*	-.28	-.27	.18	-.01
	p-value	.137	.197	.415	.965

Note. TSST = Trier Social Stress Test; MAST = Maastricht Acute Stress Test; CPT = Cold Pressor Task; DAQ = Desires for Alcohol Questionnaire; SUPPS-P = The Shortened Urgency, Premeditation (lack of), Perseverance (lack of), Sensation Seeking, Positive Urgency, Impulsive Behaviour Scale; BART = average number of space bar presses for unburst balloons during the Balloon Analogue Risk Task; 1 – AUC = 1 minus the area under the curve score (so that greater scores reflect greater delay discounting) for the Titrating Alternatives Delay Discounting Task; MAP = mean arterial pressure; HR = heart rate; PA = positive affect; NA = negative affect.

Appendix B: Chapter 3 Supplementary Materials

Favourable Ethical Approval

Thank you for using the online ethics Ethics Screening tool.

You have indicated that your study does not include any of the following:

- Human participants (taking tests, being observed, answering questionnaires, taking part in interviews/focus groups etc.)
- Gathers or uses confidential information that might identify human participants
- Includes "Relevant material" as defined by the Human Tissue Act 2004
- Includes animals (either vertebrate or invertebrate)
- Has an environmental impact
- Impacts our cultural heritage (excavation, destructive sampling etc.)
- Requires review from an external ethics committee (NHS, MOD, PHE, HMPPS etc.)
- Has health and safety concerns that cannot be met by normal risk assessment

If this is correct then please use this email as evidence of ethics review.
Your reference number is: **ETHICS-10155.**

Supplementary Methods

Figure B1. Study sample overview. MCS = Millennium Cohort Study (born 2000 – 02); NSHD = MRC National Survey of Health and Development (born 1946); NCDS = 1958 National Child Development Study; BCS70 = 1970 British Cohort Study; the Next Steps cohort were born 1989 – 90. Opted out = Those who requested not to be contacted further via phone, email, or by clicking the "opt-out" button, which was included in the invitation email. Responded = Anyone who completed the first block of the questionnaire. Data from Brown et al. (2020).

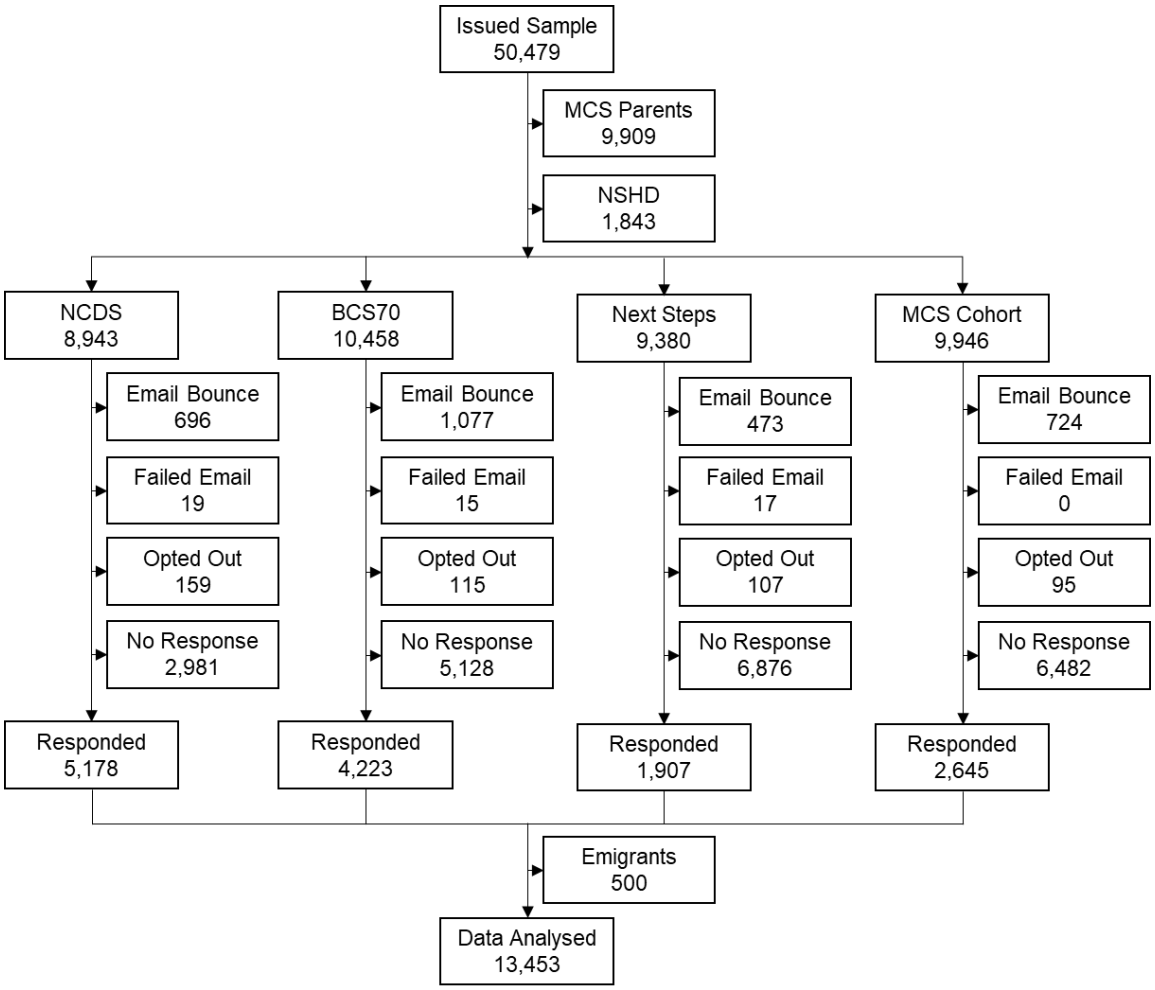

Table B1. Linear regression models used to assess the association between stress and PHQ–4 score.

Variable	Coef.	LL	UL	SE	*t*	*p*
MCS (n = 2,644)						
Unadjusted model						
Stress						
Same - no change	Ref					
Less than before	-0.95	-1.4	-0.51	0.23	-4.18	**< .001**
More than before	2.45	2.02	2.88	0.22	11.21	**< .001**
Adjusted model						
Stress						
Same - no change	Ref					
Less than before	-0.41	-0.94	0.12	0.27	-1.52	0.13
More than before	2.68	1.97	3.39	0.36	7.41	**< .001**
Sex						
Male	Ref					
Female	0.68	0.05	1.32	0.32	2.13	**0.03**
Ethnicity						
White	Ref					
Non-white	0.04	-0.85	0.94	0.45	0.09	0.93
NS-SEC 2010 analytical classes						
Higher managerial	Ref					
Lower managerial	0.47	-1.39	2.33	0.94	0.5	0.62
Intermediate occupations	0.28	-1.22	1.78	0.76	0.37	0.71
Small employer and self-employed	0.25	-1.98	2.48	1.13	0.22	0.83
Lower supervisory and technical	0.04	-1.71	1.8	0.89	0.05	0.96
Semi-routine occupations	0.38	-1.22	1.98	0.81	0.47	0.64
Routine occupations	1.02	-0.67	2.71	0.86	1.19	0.24
Uncategorised	1.3	-0.35	2.96	0.84	1.55	0.12
Economic activity						
Employed	Ref					
Self-employed	-1	-3.04	1.04	1.04	-0.96	0.34
In unpaid/voluntary work	2.02	-0.76	4.8	1.41	1.43	0.15
Apprenticeship	-0.39	-1.56	0.79	0.6	-0.65	0.52
Unemployed	0.16	-0.73	1.04	0.45	0.35	0.73
Permanently sick or disabled	2.86	-2.31	8.02	2.62	1.09	0.28
Looking after home or family	-1.3	-2.68	0.08	0.7	-1.86	0.07
In education	0.34	-1.27	1.96	0.82	0.42	0.68
Retired	-	-	-	-	-	-
Uncategorised	-1.12	-2.39	0.15	0.64	-1.73	0.08
Next Steps (*n* = 1,852)						
Unadjusted model						
Stress						
Same - no change	Ref					
Less than before	-0.4	-0.8	-0.01	0.2	-2	**0.046**

More than before	2.6	2.17	3.04	0.22	11.72	< **.001**
Adjusted model						
Stress						
Same - no change	Ref					
Less than before	-0.21	-0.62	0.19	0.2	-1.04	0.297
More than before	2.49	2.07	2.91	0.21	11.65	< **.001**
Sex						
Male	Ref					
Female	0.5	0.09	0.91	0.21	2.42	**0.016**
Ethnicity						
White	Ref					
Non-white	0.05	-0.37	0.47	0.21	0.24	0.809
NS-SEC 2010 analytical classes						
Higher managerial	Ref					
Lower managerial	-0.18	-0.73	0.37	0.28	-0.64	0.525
Intermediate occupations	0.53	-0.23	1.3	0.39	1.38	0.169
Small employer and self-employed	0.43	-0.56	1.43	0.51	0.85	0.394
Lower supervisory and technical	-0.37	-0.95	0.21	0.29	-1.25	0.212
Semi-routine occupations	0.57	-0.24	1.39	0.41	1.38	0.169
Routine occupations	-0.02	-1.13	1.09	0.56	-0.04	0.97
Uncategorised	0.53	-0.59	1.65	0.57	0.93	0.352
Economic activity						
Employed	Ref					
Self-employed	-0.37	-1.14	0.41	0.4	-0.92	0.356
In unpaid/voluntary work	-0.41	-2.82	2	1.23	-0.33	0.738
Apprenticeship	0.44	-1.05	1.94	0.76	0.58	0.559
Unemployed	1.39	-0.01	2.78	0.71	1.95	0.051
Permanently sick or disabled	4.28	1.63	6.93	1.35	3.17	**0.002**
Looking after home or family	0.9	-0.58	2.39	0.75	1.2	0.231
In education	-	-	-	-	-	-
Retired	-	-	-	-	-	-
Uncategorised	-0.01	-1.26	1.25	0.64	-0.01	0.992
BCS70 (*n* = 3,997)						
Unadjusted model						
Stress						
Same - no change	Ref					
Less than before	0.2	-0.16	0.57	0.19	1.1	0.272
More than before	2.16	1.78	2.54	0.19	11.09	< **.001**
Adjusted model						
Stress						
Same - no change	Ref					
Less than before	0.28	-0.03	0.59	0.16	1.76	0.078
More than before	1.96	1.72	2.19	0.12	16.47	< **.001**
Sex						
Male	Ref					

Female	0.01	-0.22	0.24	0.12	0.07	0.941
Ethnicity						
White	Ref					
Non-white	-0.22	-0.58	0.13	0.18	-1.24	0.213
NS-SEC 2010 analytical classes						
Higher managerial	Ref					
Lower managerial	0	-0.25	0.25	0.13	0.03	0.976
Intermediate occupations	0.31	0.02	0.6	0.15	2.08	**0.038**
Small employer and self-employed	-0.17	-0.52	0.18	0.18	-0.95	0.343
Lower supervisory and technical	0.57	-0.02	1.17	0.3	1.89	0.059
Semi-routine occupations	0.53	0.1	0.95	0.22	2.42	0.016
Routine occupations	0.4	-0.07	0.87	0.24	1.67	0.095
Uncategorised	0.46	0.03	0.88	0.22	2.11	**0.035**
Economic activity						
Employed	Ref					
Self-employed	-0.22	-0.47	0.03	0.13	-1.73	0.084
In unpaid/voluntary work	-1.39	-1.95	-0.84	0.28	-4.9	**< .001**
Apprenticeship	-	-	-	-	-	-
Unemployed	0.67	-0.22	1.56	0.45	1.47	0.141
Permanently sick or disabled	3.65	2.31	5	0.69	5.32	**< .001**
Looking after home or family	-0.03	-0.53	0.46	0.25	-0.13	0.894
In education	-	-	-	-	-	-
Retired	-0.61	-1.16	-0.06	0.28	-2.17	**0.03**
Uncategorised	0.07	-1.36	1.49	0.73	0.09	0.925
NCDS (*n* = 4,960)						
Unadjusted model						
Stress						
Same - no change	Ref					
Less than before	0	-0.28	0.29	0.15	0.02	0.983
More than before	2.16	1.89	2.44	0.14	15.48	**< .001**
Adjusted model						
Stress						
Same - no change	Ref					
Less than before	0.06	-0.2	0.32	0.13	0.44	0.657
More than before	1.95	1.68	2.23	0.14	13.87	**< .001**
Sex						
Male	Ref					
Female	0.27	0.02	0.52	0.13	2.12	**0.034**
Ethnicity						
White	Ref					
Non-white	0.25	-0.31	0.81	0.29	0.87	0.385
NS-SEC 2010 analytical classes						
Higher managerial	Ref					
Lower managerial	0.08	-0.18	0.34	0.13	0.61	0.544
Intermediate occupations	0.19	-0.09	0.47	0.14	1.33	0.183

Small employer and self-employed	0.18	-0.19	0.54	0.19	0.95	0.344
Lower supervisory and technical	-0.11	-0.59	0.37	0.25	-0.45	0.655
Semi-routine occupations	0.49	0.04	0.94	0.23	2.15	**0.032**
Routine occupations	0.41	-0.1	0.91	0.26	1.57	0.117
Uncategorised	0.38	-0.11	0.86	0.25	1.51	0.131
Economic activity						
Employed	Ref					
Self-employed	0.05	-0.31	0.41	0.18	0.26	0.795
In unpaid/voluntary work	-0.07	-1.21	1.07	0.58	-0.12	0.908
Apprenticeship	-	-	-	-	-	-
Unemployed	0.26	-0.34	0.87	0.31	0.86	0.392
Permanently sick or disabled	3.32	1.87	4.77	0.74	4.5	**< .001**
Looking after home or family	-0.26	-0.86	0.35	0.31	-0.84	0.401
In education	-	-	-	-	-	-
Retired	-0.25	-0.75	0.25	0.25	-0.98	0.326
Uncategorised	0.46	-0.67	1.59	0.58	0.8	0.422

Note. MCS = Millennium Cohort Study, BCS70 = 1970 British Cohort Study, NCDS = National Child Development Study; NS-SEC = National Statistics Socio-economic class prior to the outbreak. Economic activity reflects activity during the pandemic.

Table B2. Associations between single-item assessments, and behavioural assessments of inhibitory control.

	Spearman's Correlation	**OLS Coef.**
Risk-taking item	0.35	0.20
Patience item	-0.40	-0.17

Note. Values represent the association between the single-item measures, and behavioural assessments, of risk-taking, and patience utilised in Falk et al. (2018). The Spearman's correlations were calculated using raw data, while the linear regression coefficients were calculated using standardised scores. *N* = 409. Adapted from Falk et al. (2016). OLS = ordinary least squares.

Potential confounders

Confounders included: respondent's sex (male or female); ethnicity (white or non-white); National Statistics Socioeconomic Class prior to the lockdown (NS-SEC, grouped into eight categories: higher managerial, lower managerial, intermediate occupations, small employers and self-employed, lower supervisory and technical, semi-routine occupations, routine occupations, and Uncategorised), and economic activity during the pandemic (grouped into ten categories: employed, self-employed, unpaid/voluntary work, apprenticeship, unemployed, permanently sick or disabled, looking after the home or family, in education, retired, and Uncategorised). The Office for National Statistics have published a detailed description of the NS-SEC (Office for National Statistics, 2016).

The selection of potential confounding variables was driven by the author's substantive knowledge about established risk factors that could plausibly be related to our outcome variables. For instance, there are several sociocultural factors that should be accounted for when researching alcohol misuse using human participants (Room, 2013). Historical data suggests that binge drinking is highest among younger individuals and declines with age (Office for National Statistics, 2018). However, recently emerging evidence suggests a devaluation of alcohol among Generation Z (born between 1996 and 2015) (Kraus et al., 2020). Similarly, in Western cultures men tend to drink more than woman, yet data from the US suggests a shift in the pattern, whereby rates of AUD have increased by around 85% among women (White et al., 2015). One explanation for this may be sex differences in susceptibility to stress (Peltier et al., 2019). In terms of ethnicity, binge drinking tends to be more prevalent among white people (Twigg & Moon, 2013). This is thought to be partly attributable to the way alcohol consumption is often stigmatised among ethnic minorities (Room, 2005; Zapolski et al., 2014). Nevertheless, due to this stigmatisation, individuals that belong to these cultural groups tend to be disproportionately affected by alcohol-related harm (Zapolski et al., 2014). Further, having a lower socioeconomic status has been previously reported as being associated with lower total alcohol consumption, yet being at the greatest risk of hazardous drinking and alcohol-related harm, perhaps due to higher levels of heavy episodic (binge) drinking among more deprived groups (Mackenbach et al., 2008; Probst et al., 2020).

Missing data

Weights were derived from logistic regression models by the Centre for Longitudinal Studies team using several variables associated with non-response. For example, ¬sex, ethnicity, social class, cognitive ability, indicators mental health, educational achievement, internet access prior to the web survey, economic activity, indicators of physical health, and non-response during previous sweeps ¬– see Brown et al. (Brown et al., 2020) for a detailed description of the procedure used to calculate weights.

Table B3. Percentage of missing data by variable.

Variable	Overall	MCS	Next Steps	BCS70	NCDS
n	13,453	2,644	1,852	3,997	4,960
Sex	0.00%	0.00%	0.00%	0.00%	0.00%
Ethnicity	13.29%	3.82%	1.84%	10.56%	24.82%
Relationship status	2.70%	3.59%	2.92%	2.20%	2.54%
COVID-19 status	0.01%	0.00%	0.00%	0.03%	0.02%
Economic activity at time of survey	5.07%	8.17%	5.56%	3.53%	4.48%
Key worker	5.29%	8.28%	5.72%	0.00%	4.82%
NS–SEC 2010 analytical classes	0.00%	0.00%	0.00%	0.00%	0.00%
Change in drinking	7.87%	9.68%	6.70%	6.35%	8.57%
Alcohol misuse at time of survey	8.55%	10.25%	7.67%	6.96%	9.25%
Change in stress	8.07%	12.67%	9.67%	6.05%	6.65%
Risk-taking	7.83%	12.29%	8.96%	3.70%	6.43%
Impatience	8.02%	12.41%	8.96%	6.08%	6.88%

PHQ–4	0.00%	0.00%	0.00%	0.00%	0.00%

Note. NS-SEC = National Statistics Socio-economic Class; PHQ–4 = Patient Health Questionnaire – 4. The overall percentage of missing data was 23.43%.

Supplementary Results

Change in alcohol use during the first lockdown

Table B4. Ordinal logistic regression results for the Millennium Cohort Study with change in alcohol use as the outcome.

	Model 1			Model 2		
Variable	**OR (95% CI)**	**SE**	***p***	**OR (95% CI)**	**SE**	***p***
Sex						
Male	Ref.			Ref.		
Female	0.77 (0.51, 1.14)	0.16	0.190	0.83 (0.55, 1.27)	0.18	0.400
Ethnicity						
White	Ref.			Ref.		
Non-white	1.39 (0.64, 3.01)	0.55	0.404	1.58 (0.72, 3.47)	0.63	0.251
NS-SEC analytical classes						
Higher managerial	Ref.			Ref.		
Lower managerial	3.77 (1.00, 14.23)	2.54	0.050	3.27 (0.81, 13.27)	2.33	0.096
Intermediate occupations	1.81 (0.68, 4.81)	0.90	0.235	1.84 (0.67, 5.07)	0.95	0.239
Small employers and self employed	1.11 (0.27, 4.54)	0.79	0.888	1.16 (0.25, 5.31)	0.90	0.851
Lower supervisory and technical	0.56 (0.18, 1.73)	0.32	0.309	0.56 (0.17, 1.85)	0.34	0.336
Semi-routine occupations	2.13 (0.83, 5.5)	1.03	0.118	2.1 (0.77, 5.77)	1.08	0.148
Routine occupations	1.93 (0.68, 5.43)	1.01	0.213	1.62 (0.55, 4.79)	0.89	0.380
Uncategorised	2.11 (0.72, 6.19)	1.15	0.173	2.13 (0.67, 6.8)	1.26	0.201
Economic activity						
Employed	Ref.			Ref.		
Self-employed	0.56 (0.15, 2.03)	0.37	0.375	0.55 (0.15, 2.05)	0.37	0.371
Unpaid/voluntary work	0.16 (0.02, 1.26)	0.17	0.081	0.16 (0.01, 1.74)	0.19	0.131
Apprenticeship	1.37 (0.71, 2.63)	0.45	0.343	1.38 (0.68, 2.79)	0.49	0.370
Unemployed	0.80 (0.42, 1.54)	0.27	0.505	0.70 (0.35, 1.41)	0.25	0.319

Permanently sick or disabled	0.71 (0.17, 2.96)	0.51	0.637	0.54 (0.13, 2.29)	0.40	0.405
Retired	-	-	-	-	-	-
Looking after home or family	0.70 (0.25, 1.97)	0.37	0.504	0.48 (0.17, 1.39)	0.26	0.177
In education	**0.10 (0.02, 0.59)**	**0.09**	**0.011**	**0.12 (0.03, 0.49)**	**0.09**	**0.004**
Uncategorised	0.95 (0.52, 1.73)	0.29	0.858	0.88 (0.44, 1.75)	0.31	0.709
Stress						
Same	Ref.			Ref.		
Less				0.21 (0.02, 1.98)	0.24	0.172
More				1.47 (0.39, 5.61)	1.00	0.568
Risk-taking				0.98 (0.88, 1.10)	0.06	0.775
Risk-taking x Stress						
Same	Ref.			Ref.		
Less				1.2 (0.92, 1.57)	0.16	0.181
More				1.05 (0.87, 1.26)	0.10	0.622
Impatience				**1.14 (1.06, 1.24)**	**0.05**	**0.001**
Impatience x Stress						
Same	Ref.			Ref.		
Less				0.92 (0.75, 1.12)	0.09	0.404
More				**0.87 (0.77, 0.99)**	**0.06**	**0.030**

Note. NS-SEC = National Statistics Socio-economic Class. Model 1: Demographics (sex, ethnicity, NS-SEC prior to the outbreak of Coronavirus, and economic activity during the pandemic). Model 2: The effect of inhibitory control (risk-taking and patience), stress, and the interaction between inhibitory control and stress, adjusting for demographics.

Table B5. Ordinal logistic regression results for the Next Steps cohort with change in alcohol use as the outcome.

	Model 1			Model 2		
Variable	**OR (95% CI)**	**SE**	***p***	**OR (95% CI)**	**SE**	***p***
Sex						
Male	Ref.			Ref.		
Female	1.14 (0.85, 1.54)	0.17	0.380	1.17 (0.86, 1.59)	0.18	0.315
Ethnicity						
White	Ref.			Ref.		
Non-white	**0.71 (0.55, 0.93)**	**0.10**	**0.012**	**0.70 (0.54, 0.91)**	**0.09**	**0.008**
NS-SEC analytical classes						
Higher managerial	Ref.			Ref.		
Lower managerial	0.90 (0.61, 1.32)	0.18	0.575	0.91 (0.62, 1.34)	0.18	0.640
Intermediate occupations	0.72 (0.45, 1.14)	0.17	0.161	0.64 (0.39, 1.05)	0.16	0.075
Small employers and self employed	0.96 (0.39, 2.39)	0.44	0.937	0.88 (0.35, 2.23)	0.42	0.788
Lower supervisory and technical	0.90 (0.31, 2.6)	0.49	0.850	0.91 (0.33, 2.5)	0.47	0.851
Semi-routine occupations	1.11 (0.63, 1.97)	0.32	0.713	1.19 (0.65, 2.15)	0.36	0.574
Routine occupations	1.01 (0.52, 1.96)	0.34	0.985	1.14 (0.51, 2.54)	0.46	0.743
Uncategorised	1.59 (0.81, 3.1)	0.54	0.175	1.36 (0.68, 2.7)	0.48	0.384
Economic activity						
Employed	Ref.			Ref.		
Self-employed	1.36 (0.82, 2.25)	0.35	0.235	1.22 (0.73, 2.04)	0.32	0.458
Unpaid/voluntary work	0.39 (0.05, 2.99)	0.40	0.363	0.47 (0.06, 3.62)	0.49	0.467
Apprenticeship	0.79 (0.54, 1.15)	0.15	0.212	0.89 (0.59, 1.33)	0.18	0.562
Unemployed	0.45 (0.19, 1.06)	0.20	0.069	0.51 (0.2, 1.29)	0.24	0.155
Permanently sick or disabled	0.39 (0.11, 1.35)	0.25	0.138	0.44 (0.12, 1.57)	0.29	0.207
Retired	-	-	-	-	-	-

Looking after home or family	0.47 (0.14, 1.54)	0.28	0.213	0.58 (0.17, 1.94)	0.36	0.377
In education	-	-	-	-	-	-
Uncategorised	**0.42 (0.17, 0.99)**	**0.19**	**0.049**	0.45 (0.18, 1.11)	0.21	0.083
Stress						
Same	Ref.			Ref.		
Less				0.59 (0.09, 3.71)	0.55	0.574
More				2.21 (0.99, 4.94)	0.90	0.053
Risk-taking				1.03 (0.95, 1.13)	0.05	0.479
Risk-taking x Stress						
Same	Ref.			Ref.		
Less				0.96 (0.74, 1.24)	0.13	0.760
More				0.98 (0.87, 1.09)	0.06	0.676
Impatience				1.05 (0.97, 1.14)	0.04	0.201
Impatience x Stress						
Same	Ref.			Ref.		
Less				**1.22 (1.00, 1.48)**	**0.12**	**0.047**
More				**0.88 (0.80, 0.98)**	**0.05**	**0.016**

Note. NS-SEC = National Statistics Socio-economic Class. Model 1: Demographics (sex, ethnicity, NS-SEC prior to the outbreak of Coronavirus, and economic activity during the pandemic). Model 2: The effect of inhibitory control (risk-taking and patience), stress, and the interaction between inhibitory control and stress, adjusting for demographics.

Table B6. Ordinal logistic regression results for the 1970 British Cohort Study with change in alcohol use as the outcome.

	Model 1			Model 2		
Variable	**OR (95% CI)**	**SE**	***p***	**OR (95% CI)**	**SE**	***p***
Sex						
Male	Ref.			Ref.		
Female	**1.27 (1.08, 1.50)**	**0.11**	**0.004**	**1.19 (1.01, 1.41)**	**0.10**	**0.043**
Ethnicity						
White	Ref.			Ref.		
Non-white	0.77 (0.51, 1.16)	0.16	0.205	0.78 (0.51, 1.18)	0.16	0.234
NS-SEC analytical classes						
Higher managerial	Ref.			Ref.		
Lower managerial	0.96 (0.76, 1.22)	0.12	0.753	0.95 (0.75, 1.22)	0.12	0.708
Intermediate occupations	**0.70 (0.54, 0.92)**	**0.10**	**0.010**	**0.70 (0.53, 0.92)**	**0.10**	**0.010**
Small employers and self employed	0.78 (0.49, 1.23)	0.18	0.287	0.83 (0.52, 1.34)	0.20	0.444
Lower supervisory and technical	0.99 (0.65, 1.51)	0.21	0.955	1.02 (0.66, 1.58)	0.23	0.932
Semi-routine occupations	**0.62 (0.46, 0.85)**	**0.10**	**0.003**	**0.59 (0.43, 0.81)**	**0.10**	**0.001**
Routine occupations	**0.62 (0.39, 0.98)**	**0.15**	**0.041**	**0.56 (0.36, 0.87)**	**0.12**	**0.009**
Uncategorised	1.02 (0.68, 1.54)	0.21	0.912	1.17 (0.8, 1.71)	0.23	0.432
Economic activity						
Employed	Ref.			Ref.		
Self-employed	1.11 (0.85, 1.45)	0.15	0.435	0.98 (0.76, 1.25)	0.12	0.851
Unpaid/voluntary work	1.44 (0.33, 6.35)	1.09	0.633	1.37 (0.32, 5.94)	1.03	0.673
Apprenticeship	-	-	-	-	-	-
Unemployed	0.73 (0.43, 1.25)	0.20	0.253	0.66 (0.38, 1.15)	0.19	0.145
Permanently sick or disabled	**0.40 (0.24, 0.66)**	**0.10**	**< 0.001**	**0.35 (0.21, 0.56)**	**0.09**	**< 0.001**
Retired	1.09 (0.67, 1.75)	0.27	0.732	0.97 (0.61, 1.55)	0.23	0.900

Looking after home or family	0.87 (0.47, 1.6)	0.27	0.655	0.91 (0.49, 1.7)	0.29	0.766
In education	-	-	-	-	-	-
Uncategorised	**0.36 (0.14, 0.94)**	**0.18**	**0.036**	**0.33 (0.12, 0.9)**	**0.17**	**0.031**
Stress						
Same	Ref.			Ref.		
Less				1.40 (0.58, 3.38)	0.63	0.455
More				0.87 (0.51, 1.47)	0.23	0.594
Risk-taking				0.98 (0.94, 1.03)	0.02	0.533
Risk-taking x Stress						
Same	Ref.			Ref.		
Less				0.96 (0.85, 1.08)	0.06	0.478
More				1.06 (0.98, 1.15)	0.04	0.152
Impatience				0.98 (0.93, 1.03)	0.02	0.370
Impatience x Stress						
Same	Ref.			Ref.		
Less				1.01 (0.9, 1.14)	0.06	0.846
More				1.05 (0.97, 1.13)	0.04	0.216

Note. NS-SEC = National Statistics Socio-economic Class. Model 1: Demographics (sex, ethnicity, NS-SEC prior to the outbreak of Coronavirus, and economic activity during the pandemic). Model 2: The effect of inhibitory control (risk-taking and patience), stress, and the interaction between inhibitory control and stress, adjusting for demographics.

Table B7. Ordinal logistic regression results for the National Child Development Study with change in alcohol use as the outcome.

	Model 1			Model 2		
Variable	**OR (95% CI)**	**SE**	***p***	**OR (95% CI)**	**SE**	***p***
Sex						
Male	Ref.			Ref.		
Female	**1.23 (1.02, 1.50)**	**0.12**	**0.035**	1.19 (0.98, 1.44)	0.12	0.081
Ethnicity						
White	Ref.			Ref.		
Non-white	0.54 (0.26, 1.09)	0.19	0.083	0.53 (0.26, 1.08)	0.19	0.081
NS-SEC analytical classes						
Higher managerial	Ref.			Ref.		
Lower managerial	0.85 (0.6, 1.2)	0.15	0.354	0.88 (0.61, 1.25)	0.16	0.467
Intermediate occupations	0.9 (0.62, 1.31)	0.17	0.593	0.91 (0.62, 1.32)	0.17	0.610
Small employers and self employed	0.73 (0.44, 1.22)	0.19	0.235	0.81 (0.5, 1.34)	0.21	0.418
Lower supervisory and technical	**0.45 (0.24, 0.84)**	**0.14**	**0.012**	**0.45 (0.24, 0.86)**	**0.15**	**0.015**
Semi-routine occupations	0.96 (0.59, 1.57)	0.24	0.870	1.03 (0.63, 1.69)	0.26	0.896
Routine occupations	0.76 (0.48, 1.22)	0.18	0.260	0.79 (0.49, 1.28)	0.19	0.334
Uncategorised	0.7 (0.4, 1.22)	0.20	0.206	0.71 (0.39, 1.28)	0.21	0.257
Economic activity						
Employed	Ref.			Ref.		
Self-employed	0.94 (0.7, 1.26)	0.14	0.669	0.91 (0.66, 1.25)	0.15	0.566
Unpaid/voluntary work	1.61 (0.45, 5.71)	1.04	0.462	1.51 (0.42, 5.46)	0.99	0.529
Apprenticeship	-	-	-	-	-	-
Unemployed	0.69 (0.21, 2.22)	0.41	0.532	0.65 (0.21, 2)	0.37	0.453
Permanently sick or disabled	1.11 (0.61, 2.02)	0.34	0.743	1.13 (0.6, 2.13)	0.37	0.706
Retired	1.00 (0.59, 1.71)	0.27	0.988	1.01 (0.58, 1.75)	0.28	0.978

Looking after home or family	1.04 (0.55, 1.98)	0.34	0.903	1.05 (0.54, 2.06)	0.36	0.881
In education	-	-	-	-	-	-
Uncategorised	1.08 (0.32, 3.7)	0.68	0.902	1.04 (0.28, 3.91)	0.70	0.957
Stress						
Same	Ref.			Ref.		
Less				1.38 (0.42, 4.51)	0.83	0.590
More				0.90 (0.54, 1.48)	0.23	0.670
Risk-taking				0.99 (0.94, 1.03)	0.02	0.508
Risk-taking x Stress						
Same	Ref.			Ref.		
Less				0.97 (0.82, 1.13)	0.08	0.674
More				1.07 (0.98, 1.16)	0.04	0.118
Impatience				0.99 (0.95, 1.03)	0.02	0.504
Impatience x Stress						
Same	Ref.			Ref.		
Less				1.01 (0.89, 1.15)	0.07	0.869
More				0.99 (0.93, 1.07)	0.04	0.875

Note. NS-SEC = National Statistics Socio-economic Class. Model 1: Demographics (sex, ethnicity, NS-SEC prior to the outbreak of Coronavirus, and economic activity during the pandemic). Model 2: The effect of inhibitory control (risk-taking and patience), stress, and the interaction between inhibitory control and stress, adjusting for demographics.

Risk of alcohol–related harm due to hazardous drinking during the first lockdown

Table B8. Ordinal logistic regression results for the Millennium Cohort Study with risk of alcohol-related harm due to hazardous drinking as the outcome.

	Model 1			**Model 2**		
Variable	**OR (95% CI)**	**SE**	***p***	**OR (95% CI)**	**SE**	***p***
Sex						
Male	Ref.			Ref.		
Female	0.60 (0.36, 1.02)	0.16	0.062	0.58 (0.34, 1.02)	0.16	0.057
Ethnicity						
White	Ref.			Ref.		
Non-white	0.55 (0.23, 1.33)	0.25	0.185	0.71 (0.3, 1.67)	0.31	0.427
NS-SEC analytical classes						
Higher managerial	Ref.			Ref.		
Lower managerial	1.76 (0.39, 8.04)	1.36	0.463	1.47 (0.28, 7.77)	1.24	0.649
Intermediate occupations	0.67 (0.17, 2.59)	0.46	0.557	0.71 (0.16, 3.15)	0.54	0.655
Small employers and self employed	1.46 (0.33, 6.5)	1.11	0.618	1.04 (0.21, 5.14)	0.84	0.963
Lower supervisory and technical	0.34 (0.08, 1.53)	0.26	0.159	0.29 (0.06, 1.48)	0.24	0.135
Semi-routine occupations	0.74 (0.18, 3.09)	0.54	0.681	0.69 (0.14, 3.36)	0.56	0.647
Routine occupations	0.68 (0.17, 2.75)	0.48	0.588	0.56 (0.12, 2.71)	0.45	0.472
Uncategorised	1.08 (0.27, 4.33)	0.76	0.917	1.36 (0.3, 6.21)	1.05	0.687
Economic activity						
Employed	Ref.			Ref.		
Self-employed	0.84 (0.27, 2.58)	0.48	0.757	0.65 (0.2, 2.17)	0.40	0.485
Unpaid/voluntary work	**5.60E-07 (1.30E-07, 2.42E-06)**	**4.16E-07**	**< 0.001**	**6.40E-07 (1.04E-07, 3.96E-06)**	**5.92E-07**	**< 0.001**
Apprenticeship	**0.29 (0.11, 0.78)**	**0.15**	**0.015**	**0.25 (0.08, 0.77)**	**0.14**	**0.016**

Unemployed	0.79 (0.39, 1.61)	0.29	0.521	0.72 (0.37, 1.4)	0.24	0.333
Permanently sick or disabled	**0.11 (0.01, 1.03)**	**0.12**	**0.053**	**0.07 (0.01, 0.68)**	**0.08**	**0.021**
Retired	-	-	-	-	-	-
Looking after home or family	**6.91E-07 (2.37E-07, 2.01E-06)**	**3.75E-07,**	**< 0.001**	**5.86E-07 (9.92E-08, 3.46E-06)**	**5.29E-07**	**< 0.001**
In education	**2.63 (1.37, 5.05)**	**0.87**	**0.004**	2.16 (0.93, 5.04)	0.93	0.075
Uncategorised	**0.18 (0.05, 0.62)**	**0.11**	**0.007**	**0.11 (0.03, 0.46)**	**0.08**	**0.003**
Stress						
Same	Ref.			Ref.		
Less				0.25 (0.01, 4.98)	0.38	0.361
More				0.82 (0.18, 3.65)	0.62	0.794
Risk-taking				0.98 (0.8, 1.19)	0.10	0.836
Risk-taking x Stress						
Same	Ref.			Ref.		
Less				1.32 (0.89, 1.96)	0.27	0.172
More				1.13 (0.91, 1.41)	0.12	0.258
Impatience				**1.20 (1.05, 1.38)**	**0.08**	**0.010**
Impatience x Stress						
Same	Ref.			Ref.		
Less				0.9 (0.73, 1.12)	0.10	0.359
More				0.95 (0.8, 1.14)	0.09	0.603

Note. NS-SEC = National Statistics Socio-economic Class. Model 1: Demographics (sex, ethnicity, NS-SEC prior to the outbreak of Coronavirus, and economic activity during the pandemic). Model 2: The effect of inhibitory control (risk-taking and patience), stress, and the interaction between inhibitory control and stress, adjusting for demographics.

Table B9. Ordinal logistic regression results for the Next Steps cohort with risk of alcohol-related harm due to hazardous drinking as the outcome.

	Model 1			Model 2		
Variable	**OR (95% CI)**	**SE**	***p***	**OR (95% CI)**	**SE**	***p***
Sex						
Male	Ref.			Ref.		
Female	**0.60 (0.42, 0.85)**	**0.11**	**0.004**	**0.56 (0.39, 0.82)**	**0.11**	**0.003**
Ethnicity						
White	Ref.			Ref.		
Non-white	**0.55 (0.34, 0.91)**	**0.14**	**0.02**	**0.44 (0.27, 0.73)**	**0.11**	**0.002**
NS-SEC analytical classes						
Higher managerial	Ref.			Ref.		
Lower managerial	1.03 (0.65, 1.63)	0.24	0.893	1.02 (0.65, 1.6)	0.24	0.935
Intermediate occupations	0.6 (0.37, 0.97)	0.15	0.037	0.59 (0.35, 0.99)	0.16	0.047
Small employers and self employed	1.43 (0.57, 3.58)	0.67	0.448	1.2 (0.41, 3.52)	0.66	0.741
Lower supervisory and technical	1.03 (0.36, 2.93)	0.55	0.956	1.00 (0.35, 2.87)	0.54	0.998
Semi-routine occupations	0.99 (0.49, 1.97)	0.35	0.971	0.93 (0.44, 1.94)	0.35	0.845
Routine occupations	0.55 (0.23, 1.32)	0.25	0.181	0.62 (0.24, 1.61)	0.3	0.327
Uncategorised	0.63 (0.23, 1.7)	0.32	0.357	0.6 (0.21, 1.69)	0.32	0.328
Economic activity						
Employed	Ref.			Ref.		
Self-employed	1.19 (0.64, 2.22)	0.38	0.584	1.12 (0.57, 2.2)	0.38	0.732
Unpaid/voluntary work	0.66 (0.03, 14.5)	1.04	0.793	0.64 (0.02, 19.07)	1.1	0.795
Apprenticeship	1.73 (0.41, 7.28)	1.27	0.454	2.53 (0.62, 10.32)	1.81	0.196
Unemployed	1.78 (0.56, 5.68)	1.05	0.327	2.20 (0.72, 6.74)	1.25	0.167
Permanently sick or disabled	0.68 (0.08, 5.73)	0.74	0.721	0.91 (0.11, 7.71)	0.99	0.928
Retired	-	-	-	-	-	-

Looking after home or family	1.73 (0.36, 8.33)	1.38	0.496	2.38 (0.44, 12.94)	2.05	0.315
In education	-	-	-	-	-	-
Uncategorised	0.64 (0.13, 3.24)	0.53	0.589	0.66 (0.13, 3.42)	0.55	0.617
Stress						
Same	Ref.			Ref.		
Less				0.36 (0.06, 2.15)	0.33	0.259
More				**3.77 (1.15, 12.28)**	**2.27**	**0.028**
Risk-taking				**1.18 (1.05, 1.32)**	**0.07**	**0.006**
Risk-taking x Stress						
Same	Ref.			Ref.		
Less				0.97 (0.77, 1.22)	0.11	0.813
More				0.88 (0.77, 1.02)	0.07	0.098
Impatience				0.97 (0.9, 1.06)	0.04	0.531
Impatience x Stress						
Same	Ref.			Ref.		
Less				**1.31 (1.10, 1.57)**	**0.12**	**0.002**
More				0.95 (0.83, 1.07)	0.06	0.396

Note. NS-SEC = National Statistics Socio-economic Class. Model 1: Demographics (sex, ethnicity, NS-SEC prior to the outbreak of Coronavirus, and economic activity during the pandemic). Model 2: The effect of inhibitory control (risk-taking and patience), stress, and the interaction between inhibitory control and stress, adjusting for demographics.

Table B10. Ordinal logistic regression results for the Next Steps cohort with risk of alcohol-related harm due to hazardous drinking as the outcome.

	Model 1			Model 2		
Variable	**OR (95% CI)**	**SE**	***p***	**OR (95% CI)**	**SE**	***p***
Sex						
Male	Ref.			Ref.		
Female	**0.64 (0.53, 0.76)**	**0.06**	**< 0.001**	**0.64 (0.53, 0.77)**	**0.06**	**< 0.001**
Ethnicity						
White	Ref.			Ref.		
Non-white	**0.44 (0.23, 0.84)**	**0.15**	**< 0.001**	**0.41 (0.21, 0.81)**	**0.14**	**0.010**
NS-SEC analytical classes						
Higher managerial	Ref.			Ref.		
Lower managerial	1.03 (0.83, 1.29)	0.12	0.775	1.03 (0.82, 1.29)	0.12	0.808
Intermediate occupations	0.79 (0.6, 1.04)	0.11	0.098	0.82 (0.62, 1.08)	0.12	0.164
Small employers and self employed	0.85 (0.55, 1.32)	0.19	0.478	0.87 (0.56, 1.36)	0.20	0.536
Lower supervisory and technical	0.70 (0.42, 1.18)	0.19	0.183	0.76 (0.45, 1.27)	0.20	0.289
Semi-routine occupations	0.85 (0.61, 1.18)	0.14	0.335	0.84 (0.6, 1.18)	0.15	0.321
Routine occupations	0.70 (0.44, 1.11)	0.17	0.132	**0.65 (0.44, 0.96)**	**0.13**	**0.030**
Uncategorised	0.91 (0.6, 1.38)	0.19	0.652	1.02 (0.68, 1.53)	0.21	0.939
Economic activity						
Employed	Ref.			Ref.		
Self-employed	1.1 (0.82, 1.48)	0.16	0.512	1.01 (0.77, 1.32)	0.14	0.944
Unpaid/voluntary work	1.12 (0.24, 5.14)	0.87	0.884	1.12 (0.24, 5.1)	0.87	0.886
Apprenticeship	-	-	-	-	-	-
Unemployed	0.83 (0.43, 1.6)	0.28	0.584	0.77 (0.41, 1.45)	0.25	0.422
Permanently sick or disabled	**0.24 (0.10, 0.58)**	**0.11**	**0.002**	**0.21 (0.09, 0.53)**	**0.10**	**0.001**
Retired	0.89 (0.54, 1.47)	0.23	0.653	0.82 (0.49, 1.36)	0.21	0.437

Looking after home or family	0.7 (0.26, 1.86)	0.35	0.475	0.72 (0.28, 1.85)	0.35	0.498
In education	-	-	-	-	-	-
Uncategorised	0.7 (0.28, 1.75)	0.33	0.442	0.69 (0.27, 1.78)	0.33	0.442
Stress						
Same	Ref.			Ref.		
Less				1.00 (0.35, 2.89)	0.54	0.999
More				1.29 (0.73, 2.25)	0.37	0.380
Risk-taking				**1.06 (1.01, 1.12)**	**0.03**	**0.017**
Risk-taking x Stress						
Same	Ref.			Ref.		
Less				0.95 (0.82, 1.1)	0.07	0.504
More				1.01 (0.93, 1.09)	0.04	0.834
Impatience				1.00 (0.95, 1.04)	0.02	0.859
Impatience x Stress						
Same	Ref.			Ref.		
Less				**1.17 (1.04, 1.31)**	**0.07**	**0.007**
More				1.00 (0.93, 1.08)	0.04	0.943

Note. NS-SEC = National Statistics Socio-economic Class. Model 1: Demographics (sex, ethnicity, NS-SEC prior to the outbreak of Coronavirus, and economic activity during the pandemic). Model 2: The effect of inhibitory control (risk-taking and patience), stress, and the interaction between inhibitory control and stress, adjusting for demographics.

Table B11. Ordinal logistic regression results for the National Child Development Study with risk of alcohol-related harm due to hazardous drinking as the outcome.

	Model 1			Model 2		
Variable	**OR (95% CI)**	**SE**	***p***	**OR (95% CI)**	**SE**	***p***
Sex						
Male	Ref.			Ref.		
Female	**0.64 (0.52, 0.78)**	**0.07**	**< 0.001**	**0.62 (0.5, 0.76)**	**0.07**	**< 0.001**
Ethnicity						
White	Ref.			Ref.		
Non-white	**0.26 (0.12, 0.56)**	**0.10**	**< 0.001**	**0.27 (0.13, 0.58)**	**0.11**	**< 0.001**
NS-SEC analytical classes						
Higher managerial	Ref.			Ref.		
Lower managerial	0.84 (0.57, 1.24)	0.17	0.378	0.82 (0.56, 1.19)	0.16	0.295
Intermediate occupations	0.75 (0.51, 1.12)	0.15	0.160	0.75 (0.51, 1.1)	0.15	0.138
Small employers and self employed	0.71 (0.39, 1.28)	0.21	0.256	0.75 (0.42, 1.33)	0.22	0.324
Lower supervisory and technical	0.66 (0.38, 1.13)	0.18	0.133	0.71 (0.42, 1.22)	0.19	0.213
Semi-routine occupations	0.83 (0.49, 1.4)	0.22	0.483	0.83 (0.49, 1.41)	0.22	0.500
Routine occupations	**0.56 (0.33, 0.96)**	**0.15**	**0.035**	**0.55 (0.32, 0.96)**	**0.16**	**0.035**
Uncategorised	0.85 (0.52, 1.37)	0.21	0.501	0.93 (0.57, 1.52)	0.23	0.784
Economic activity						
Employed	Ref.			Ref.		
Self-employed	0.74 (0.51, 1.08)	0.14	0.120	0.66 (0.46, 0.96)	0.12	0.029
Unpaid/voluntary work	1.36 (0.67, 2.78)	0.50	0.394	1.32 (0.64, 2.73)	0.49	0.449
Apprenticeship	-	-	-	-	-	-
Unemployed	0.87 (0.37, 2.08)	0.39	0.761	0.77 (0.32, 1.82)	0.34	0.548
Permanently sick or disabled	0.59 (0.26, 1.33)	0.24	0.203	0.45 (0.2, 1.01)	0.19	0.054
Retired	1.05 (0.7, 1.59)	0.22	0.807	0.88 (0.4, 1.93)	0.35	0.750

Looking after home or family	0.76 (0.44, 1.3)	0.21	0.312	0.95 (0.62, 1.44)	0.20	0.797
In education	-	-	-	-	-	-
Uncategorised	0.92 (0.42, 2)	0.36	0.831	0.88 (0.4, 1.93)	0.35	0.750
Stress						
Same	Ref.			Ref.		
Less				0.74 (0.25, 2.16)	0.41	0.585
More				0.88 (0.49, 1.60)	0.27	0.680
Risk-taking				1.00 (0.95, 1.05)	0.03	0.945
Risk-taking x Stress						
Same	Ref.			Ref.		
Less				1.04 (0.89, 1.22)	0.08	0.631
More				1.08 (0.99, 1.18)	0.05	0.091
Impatience				1.02 (0.97, 1.06)	0.02	0.480
Impatience x Stress						
Same	Ref.			Ref.		
Less				1.04 (0.94, 1.16)	0.06	0.435
More				1.00 (0.92, 1.09)	0.04	0.972

Note. NS-SEC = National Statistics Socio-economic Class. Model 1: Demographics (sex, ethnicity, NS-SEC prior to the outbreak of Coronavirus, and economic activity during the pandemic). Model 2: The effect of inhibitory control (risk-taking and patience), stress, and the interaction between inhibitory control and stress, adjusting for demographics.

Change in stress during the first lockdown

Table B12. Ordinal logistic regression results for the Millennium Cohort Study with change in stress as the outcome.

Variable	OR (95% CI)	SE	*p*
Sex			
Male	Ref.		
Female	**1.54 (1.08, 2.20)**	**0.28**	**0.017**
Ethnicity			
White	Ref.		
Non-white	1.66 (0.75, 3.66)	0.67	0.213
NS-SEC analytical classes			
Higher managerial	Ref.		
Lower managerial	1.11 (0.26, 4.8)	0.83	0.885
Intermediate occupations	0.7 (0.17, 2.85)	0.5	0.613
Small employers and self employed	0.81 (0.13, 5.1)	0.76	0.818
Lower supervisory and technical	0.32 (0.07, 1.39)	0.24	0.128
Semi-routine occupations	0.79 (0.19, 3.29)	0.57	0.749
Routine occupations	0.55 (0.12, 2.45)	0.42	0.431
Uncategorised	0.39 (0.09, 1.67)	0.29	0.205
Economic activity			
Employed	Ref.		
Self-employed	**5.53 (1.56, 19.57)**	**3.55**	**0.008**
Unpaid/voluntary work	5.33 (0.27, 104.22)	8.05	0.269
Apprenticeship	0.54 (0.28, 1.02)	0.17	0.056
Unemployed	**1.75 (1.08, 2.83)**	**0.43**	**0.024**
Permanently sick or disabled	1.36 (0.47, 3.92)	0.73	0.567
Retired	-	-	-

Looking after home or family	0.99 (0.35, 2.8)	0.52	0.979
In education	0.39 (0.04, 3.61)	0.44	0.407
Uncategorised	0.85 (0.46, 1.59)	0.27	0.618

Note. NS-SEC = National Statistics Socio-economic Class.

Table B13. Ordinal logistic regression results for the Next Steps cohort with change in stress as the outcome.

Variable	OR (95% CI)	SE	*p*
Sex			
Male	Ref.		
Female	**1.93 (1.39, 2.70)**	**0.33**	**< 0.001**
Ethnicity			
White	Ref.		
Non-white	0.93 (0.66, 1.32)	0.17	0.691
NS-SEC analytical classes			
Higher managerial	Ref.		
Lower managerial	0.80 (0.52, 1.23)	0.18	0.314
Intermediate occupations	1.26 (0.77, 2.08)	0.32	0.355
Small employers and self employed	0.48 (0.18, 1.29)	0.24	0.148
Lower supervisory and technical	0.61 (0.36, 1.02)	0.16	0.060
Semi-routine occupations	1.30 (0.79, 2.15)	0.33	0.308
Routine occupations	1.76 (0.87, 3.56)	0.63	0.114
Uncategorised	1.35 (0.68, 2.64)	0.46	0.388
Economic activity			
Employed	Ref.		
Self-employed	**2.14 (1.15, 3.98)**	**0.68**	**0.017**
Unpaid/voluntary work	2.77 (0.4, 19.05)	2.72	0.300
Apprenticeship	0.36 (0.04, 3.42)	0.41	0.375
Unemployed	1.26 (0.55, 2.91)	0.54	0.586
Permanently sick or disabled	0.57 (0.2, 1.66)	0.31	0.306
Retired	-	-	-
Looking after home or family	0.71 (0.29, 1.73)	0.32	0.452

In education	-	-	-
Uncategorised	0.87 (0.39, 1.96)	0.36	0.734

Note. NS-SEC = National Statistics Socio-economic Class.

Table B14. Ordinal logistic regression results for the Next Steps cohort with change in stress as the outcome.

Variable	OR (95% CI)	SE	*p*
Sex			
Male	Ref.		
Female	**1.62 (1.37, 1.92)**	**0.14**	**< 0.001**
Ethnicity			
White	Ref.		
Non-white	0.72 (0.4, 1.3)	0.22	0.280
NS-SEC analytical classes			
Higher managerial	Ref.		
Lower managerial	1.08 (0.86, 1.36)	0.13	0.498
Intermediate occupations	1.13 (0.88, 1.44)	0.14	0.335
Small employers and self employed	1.11 (0.74, 1.66)	0.23	0.607
Lower supervisory and technical	0.89 (0.58, 1.37)	0.19	0.602
Semi-routine occupations	1.28 (0.92, 1.78)	0.22	0.151
Routine occupations	0.92 (0.69, 1.22)	0.13	0.549
Uncategorised	1.08 (0.71, 1.63)	0.23	0.728
Economic activity			
Employed	Ref.		
Self-employed	1.21 (0.92, 1.6)	0.17	0.180
Unpaid/voluntary work	1.58 (0.36, 6.8)	1.18	0.542
Apprenticeship	-	-	-
Unemployed	1.33 (0.63, 2.8)	0.5	0.460
Permanently sick or disabled	2.07 (0.99, 4.31)	0.78	0.053
Retired	1.41 (0.86, 2.32)	0.36	0.171
Looking after home or family	0.5 (0.17, 1.44)	0.27	0.199

In education	-	-	-
Uncategorised	1.45 (0.67, 3.17)	0.58	0.348

Note. NS-SEC = National Statistics Socio-economic Class.

Table B15. Ordinal logistic regression results for the Next Steps cohort with change in stress as the outcome.

Variable	OR (95% CI)	SE	*p*
Sex			
Male	Ref.		
Female	**2.03 (1.66, 2.48)**	**0.21**	**< 0.001**
Ethnicity			
White	Ref.		
Non-white	0.88 (0.5, 1.56)	0.26	0.662
NS-SEC analytical classes			
Higher managerial	Ref.		
Lower managerial	1.01 (0.67, 1.51)	0.21	0.968
Intermediate occupations	1.3 (0.9, 1.87)	0.24	0.166
Small employers and self employed	0.96 (0.56, 1.64)	0.26	0.887
Lower supervisory and technical	1.58 (0.93, 2.67)	0.43	0.092
Semi-routine occupations	1.41 (0.93, 2.14)	0.30	0.110
Routine occupations	1.14 (0.67, 1.91)	0.30	0.632
Uncategorised	1.26 (0.79, 1.99)	0.30	0.336
Economic activity			
Employed	Ref.		
Self-employed	1.32 (0.94, 1.85)	0.23	0.108
Unpaid/voluntary work	1.24 (0.48, 3.21)	0.60	0.661
Apprenticeship	-	-	-
Unemployed	1.09 (0.51, 2.33)	0.42	0.818
Permanently sick or disabled	1.45 (0.79, 2.66)	0.45	0.235
Retired	1.05 (0.69, 1.6)	0.22	0.809
Looking after home or family	1.04 (0.58, 1.87)	0.31	0.884

In education	-	-	-
Uncategorised	1.60 (0.76, 3.37)	0.61	0.213

Note. NS-SEC = National Statistics Socio-economic Class.

Appendix C: Chapter 4 Supplementary Materials

Favourable Ethical Approval

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6 April 2020

FAVOURABLE ETHICAL OPINION – FOLLOWING RESUBMISSION

Study Title: AlCovid-19: The effects of social isolation on alcohol use

Reference Number: SFEC 2020-030

Date Resubmitted: 6 April 2020

Thank you for resubmitting your application to the Science and Health Faculty Ethics Committee (SFEC) for ethical review in accordance with current procedures, for making the requested changes following the first SFEC review, and for the clarifications provided.

I am pleased to inform you that SFEC was content to grant a favourable ethical opinion of the above research on the basis described in the submitted documents listed at Annex A, and subject to standard general conditions (*See Annex B)*.

Please note that the favourable opinion of SFEC does not grant permission or approval to undertake the research. Management permission or approval must be obtained from any host organisation, including the University of Portsmouth or supervisor, prior to the start of the study.

Wishing you every success in your research

Dr Paul Morris
Chair, Science and Health Faculty Ethics Committee

Annexes

A - Documents reviewed
B - After ethical review - Guidance for researchers

Study 1

Socioeconomic status index

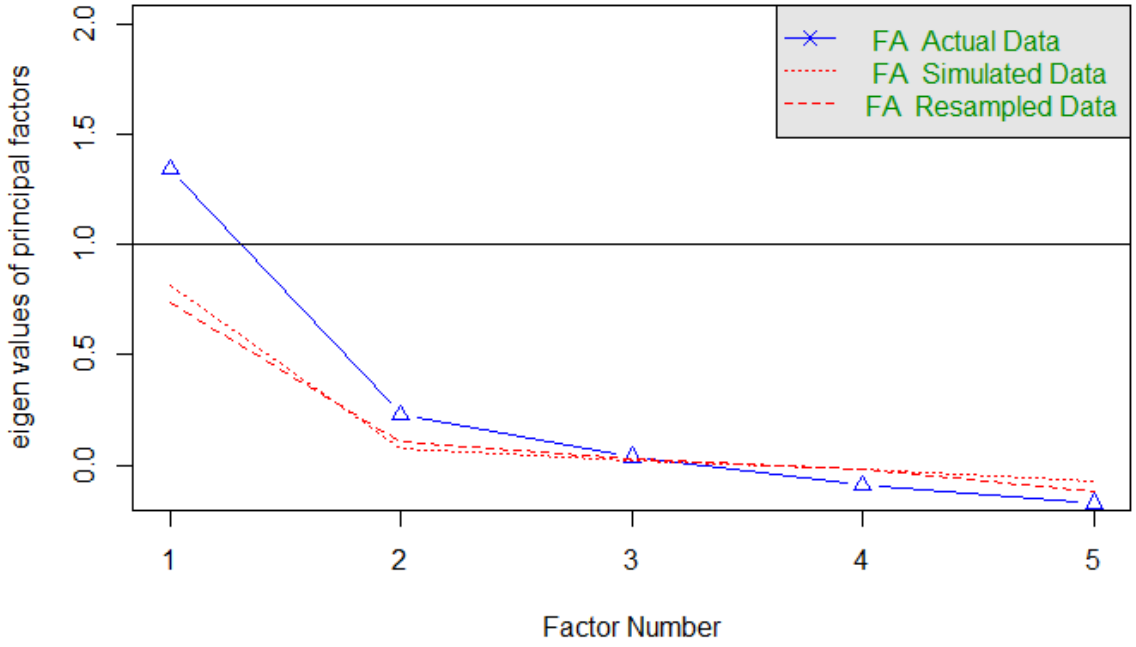
An exploratory factor analysis (EFA) was used to create an index of socioeconomic status using the *psych* package (version 2.1.3) in R (version 4.0.4). The variables entered into the EFA included:

1. Highest level of education completed (secondary education [e.g., GED/GCSE], high school diploma/A–levels, undergraduate degree [BA/BSc/other], graduate degree [MA/MSc/MPhil/other], doctoral degree or higher).
2. Occupation (full–time student, never worked/long–term unemployed, consultant, skilled labourer, trained professional, support staff, administrative staff, junior management, middle management, upper management).
3. Key worker status (yes, no).
4. Relative income (continuous).
5. Subjective social status (working class, lower–middle class, upper–middle class, upper–class).

The following EFA analyses were conducted using guidelines outlined in Preacher and MacCallum (2003). Bartlett's test indicated correlation adequacy, χ^2 (10) = 204.02, $p < .001$, the KMO test indicated sampling adequacy, *MSA* = 0.67, and the determinant ($|R| = .544$) was well above the specified cut off of .00001, suggesting that the data were not multicollinear. As that dataset that underwent EFA included a mix of continuous, polytomous and dichotomous variables, the *mixed.cor* function from the *psych* package was used to calculate the correlation matrix that was subjected to EFA.

It is important to use multiple methods to determine how many factors to retain (Harrison, 2020). Here, parallel analysis, scree plot examination, and the K1 criterion (Kaiser, 1974) were utilised. The K1 criterion, scree plot and theoretical assumptions suggested that a one factor model was appropriate. However, the results of the parallel analysis suggested two–factors. Therefore, taken together, a one–factor model was tested. The scree plot and parallel analysis are shown in Figure C1.

Figure C1. Parallel analysis scree plots.



Maximum likelihood was used with direct oblimin rotation. After testing all five variables, one item, key worker status, had a factor loading less than the criterion of .300, so it was omitted from further analyses. Another one–factor model was tested, and the factor loadings and communalities are presented in Table C1. This model achieved simple structure with each item loading onto a single factor. Factor scores were calculated using the regression method, where factor loadings are adjusted to take account of the initial correlations between variables, thus differences in the unit of measurement and variances are stabilised (Field, 2013).

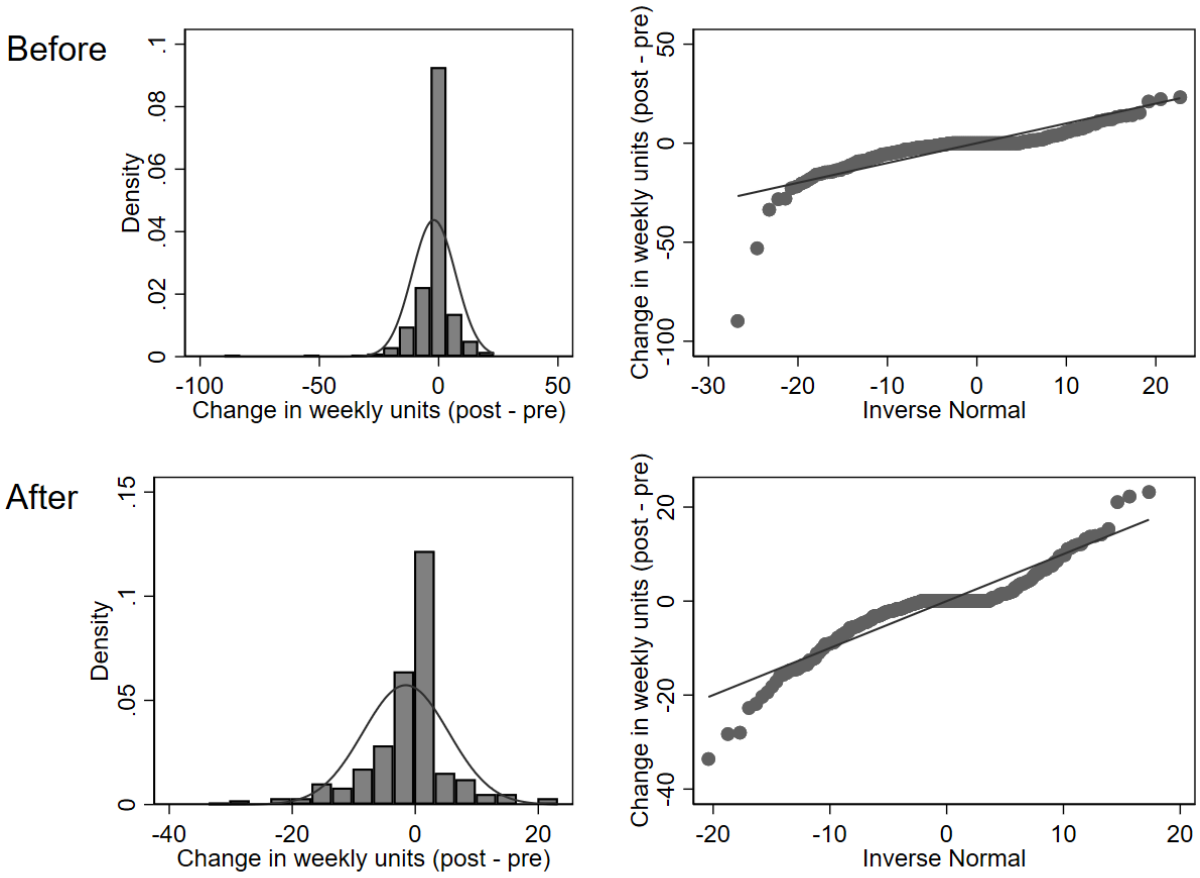
Table C1. One–factor model communalities and loadings.

Variable	Factor loadings	Communalities
Relative income	.68	.46
Education	.59	.34
Occupation	.55	.30
Subjective social status	.46	.21

Distribution plot showing outliers

Two participants reported drinking more than 50 units less during social isolation compared to before. As shown in Figure C2, these were clear outliers and were removed from the dataset prior to analysis.

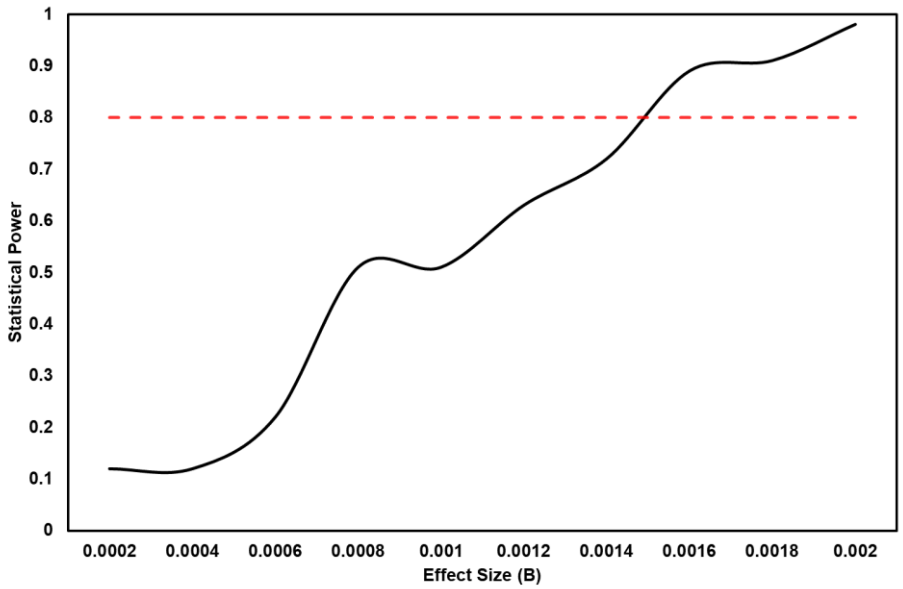
Figure C2. Distributional plots of change in units per week (post – pre) showing two clear outliers.



Sensitivity power analysis

The following procedure is partially described in Green and Macleod (2016)and Lakens (2022). We refitted the model where change in heavy drinking days was regressed on our hypothesised two–way boredom x premeditation interaction and several covariates: age, gender, ethnicity, socioeconomic status, the number of COVID–19 symptoms experienced, and whether the participant was isolated with children. We chose to use this model in our sensitivity analysis as it was (a) the most complex in terms of the number of parameters included in the model, thus will logically require the largest sample size to reliably detect an effect should one exist; and (b) because it was the model where the smallest significant effect was estimated. We used lme4 (Bates et al., 2015) to refit the model to the first imputed dataset. We then sequentially substituted our estimated effect size for our model with effect sizes ranging from B = 0.0002 to B = 0.002. Early simulation runs revealed that the upper limit of B = 0.002 is sufficient to achieve a statistical power of 98%. For each of these effect sizes, we conduced simulation–based power analyses with the simr package (Green & Macleod, 2016). This revealed that our design had sufficient statistical power (1 – β) = 80% to detect an effect size of B = 0.0015. Results are shown in Figure C3.

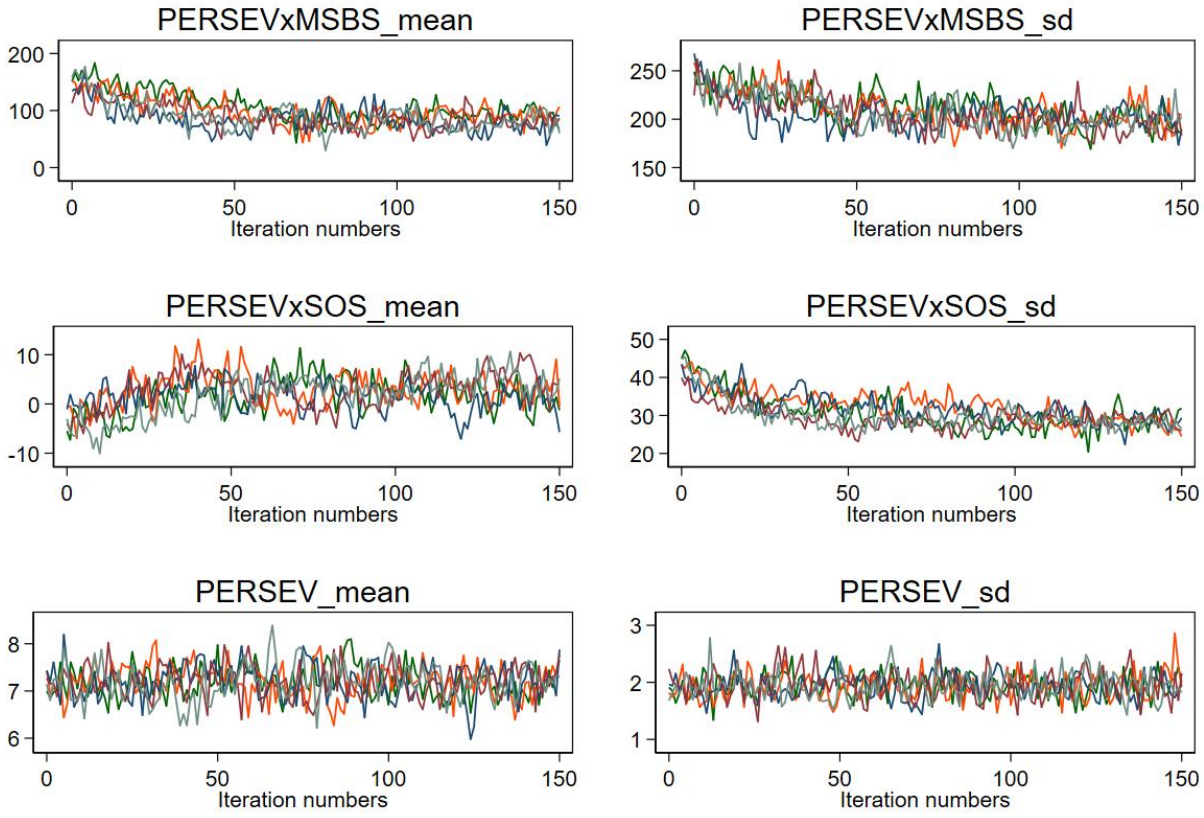
Figure C3. Results of a sensitivity power analysis for a range of effect sizes. The dashed line represents statistical power (1 – β) = 80%.



Multiple imputation

Convergence following multiple imputation can be assessed by plotting one or more parameters against the iteration number; the lines should be interwoven with no obvious pattern (Enders, 2010; Van Buuren, 2018). Figure C4 displays times–series plots for the mean and standard deviation of the interactions between lack of perseverance, boredom and stress. We time–series plots for interaction variables as they are the most likely to experience problematic convergence, thus if convergence can be established for these terms, then it is likely that convergence was also achieved for parameters that converge more quickly.

Figure C4. Time-series plot showing mean and standard deviation of the imputed values plotted against the iteration number for the first five imputed datasets. Each line represents an imputed dataset. Note. PERSEV = Lack of Perseverance; MSBS = Multidimensional State Boredom Scale, SOS = Short Stress Overload Scale.



Supplementary results

The association between perceived stress reactivity and a change in stress

We had planned to test the hypothesis that those with greater perceived stress reactivity scores would have higher stress–induced alcohol use. Before running the moderation analysis, we tested the association between perceived stress reactivity and a change in stress. This was tested by calculating the Pearson correlation coefficient (see Table C2), which was non–significant and indicated no relationship, $r(335) = 0.00$, $p = 0.967$. We then used a median split to categorise perceived stress reactivity into high (49.96%, MSOS–S = 0.93, SDSOS–S = 6.97) and low (51.04%, MSOS–S = 0.73, SDSOS–S = 4.69) groups. Subsequently, using linear mixed effects models with sub–region as a random effect, we regressed the change in stress score on our new perceived stress reactivity group variable (analogous to an independent samples t–test). Finally, we adjusted this model by including the following covariates: age, gender, ethnicity, socioeconomic status, the number of symptoms experienced, and whether the respondent was isolating with children. Like the correlation analysis, both the unadjusted ($B = -0.17$, $p = 0.81$) and adjusted models ($B = -0.15$, $p = 0.84$) were non–significant. Thus, indicating that those who perceive themselves to be more stress reactive did not report an increase in stress during the first wave of the pandemic. It is for this reason that the planned moderation analysis was not conducted.

Bivariate relationships

Table C2. Matrix of Pearson correlations (below the diagonal) and the corresponding *p*–values (above the diagonal) between the main study variables (*N* = 337).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. AUDIT		0.035	0.190	0.009	< .001	< .001	< .001	< .001	< .001	0.966	0.741	0.100	< .001	0.001	0.958	< .001	< .001
2. Alcohol Units	**-0.14**		< .001	< .001	0.183	0.614	0.426	0.476	0.227	0.370	0.414	0.656	0.717	0.414	0.162	0.963	0.710
3. Drinking Days	-0.08	**0.75**		< .001	0.109	0.576	0.837	0.508	0.626	0.421	0.550	0.054	0.831	0.228	0.639	0.536	0.982
4. Heavy Days	**-0.17**	**0.60**	**0.29**		0.304	0.566	0.663	0.792	0.121	0.716	0.218	0.121	0.370	0.023	0.123	0.968	0.506
5. APQ	-0.31	0.09	0.10	0.07		0.002	0.011	0.212	0.005	0.837	0.170	0.533	0.015	0.245	0.879	0.100	0.035
6. DMQ-R Soc	**0.46**	-0.03	0.03	-0.04	**-0.18**		< .001	< .001	< .001	0.787	0.496	0.004	< .001	0.041	0.444	< .001	< .001
7. DMQ-R Cop	**0.54**	-0.05	-0.01	0.03	**-0.16**	**0.51**		< .001	< .001	0.402	0.000	0.561	< .001	0.019	0.242	0.663	< .001
8. DMQ-R Enh	**0.54**	-0.04	0.04	-0.02	-0.07	**0.68**	**0.55**		< .001	0.889	0.943	0.022	0.012	0.024	0.826	0.001	< .001
9. DMQ-R Con	**0.32**	-0.07	-0.03	-0.10	**-0.16**	**0.50**	**0.35**	**0.29**		0.601	0.026	0.026	< .001	< .001	0.001	0.056	< .001
10. SOS-S	0.00	-0.05	-0.05	-0.02	0.01	-0.02	-0.05	0.01	0.03		0.967	< .001	0.065	0.165	0.954	0.704	0.482
11. PSRS	-0.02	0.05	0.04	0.07	-0.08	0.04	**0.24**	0.00	**0.13**	0.00		0.741	< .001	0.517	0.335	0.011	0.066
12. MSBS	0.10	-0.03	0.11	-0.09	0.04	**0.17**	0.03	**0.13**	**0.13**	**0.30**	-0.02		0.191	0.470	0.272	0.461	0.542
13. Negative Urgency	**0.30**	-0.02	0.01	-0.05	**-0.15**	**0.23**	**0.31**	**0.16**	**0.31**	0.10	**0.37**	0.08		< .001	0.487	0.003	< .001
14. Premeditation	0.19	-0.05	0.07	**-0.15**	-0.07	**0.13**	**0.15**	**0.13**	**0.26**	-0.08	0.04	-0.04	**0.24**		< .001	0.002	< .001

15. Perseverance	0.00	-0.08	-0.03	-0.09	-0.01	0.05	0.07	0.01	**0.19**	0.00	-0.06	-0.06	-0.04	**0.42**		0.268	0.291
16. Sensation Seeking	**0.22**	0.00	0.04	0.00	-0.09	**0.21**	0.03	**0.19**	0.12	0.02	**-0.15**	0.04	**0.17**	**0.18**	-0.07		< .001
17. Positive Urgency	**0.32**	-0.02	0.00	-0.04	**-0.13**	**0.26**	**0.27**	**0.21**	**0.38**	-0.04	0.12	0.04	**0.60**	**0.41**	0.06	**0.34**	
18. DOSPERT	**0.30**	**-0.14**	-0.08	-0.10	**-0.18**	**0.28**	**0.22**	**0.26**	**0.33**	-0.06	-0.08	0.04	**0.21**	**0.23**	0.05	**0.53**	**0.37**

Note. Correlations were calculated using imputed data (m = 40). Alcohol Units, Drinking Days, Heavy Days, APQ, SOS–S and MSBS reflect change. 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; DMQ–R = Revised Drinking Motives Questionnaire; Soc = Social Motives; Cop = Coping Motives; Enh = Enhancement Motives; Con = Conformity Motives; SOS–S = Short Stress Overload Scale; PSRS = Perceived Stress Reactivity Scale Total Score; MSBS = Multidimensional State Boredom Scale total score; Negative Urgency, Premeditation, Perseverance, Sensation Seeking and Positive Urgency are facets of The Shortened Urgency, Premeditation (lack of), Perseverance (lack of), Sensation Seeking, Positive Urgency (SUPPS–P) Impulsive Behaviour Scale; DOSPERT = Domain–Specific Risk–taking Scale total score.

Significant effects ($p < .05$) are in boldface

Changes in alcohol use, stress, and boredom

Table C3. Null models (*N* = 337).

Model	Parameter	B	SE	95% CI
Alcohol Units Change	Intercept	-1.53	0.41	-2.34 to -0.71
Drinking Days Change	Intercept	-0.21	0.09	-0.37 to -0.04
Heavy Days Change	Intercept	-0.09	0.05	-0.20 to 0.02
APQ Change [a]	Intercept	-1.47	0.22	-1.90 to -1.04
SOS–S Change	Intercept	-0.77	0.36	-1.48 to -0.07
MSBS Change	Intercept	18.16	2.08	14.08 to 22.25

Note. Models fit using imputed data (*m* = 40). 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; SOS-S = Short Stress Overload Scale; MSBS = Multidimensional State Boredom Scale.

[a] model fit using the Broyden-Fletcher-Goldfarb-Shanno algorithm to allow convergence.

Table C4. Linear mixed effects model assessing change in units while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, and whether the participant was isolating with children (*N* = 337).

Variable	B	SE	95% CI
Intercept	-1.00	1.35	-3.66 to 1.65
Age	-0.01	0.04	-0.08 to 0.06
Male	-2.55	0.90	-4.31 to -0.78
Non–white	-0.48	1.83	-4.06 to 3.10
SES	-0.13	0.52	-1.14 to 0.89
No. Symp.	0.33	0.75	-1.14 to 1.79
Iso. w. Child.	0.66	0.88	-1.05 to 2.38

Note. Models fit using imputed data (*m* = 40). 1 unit = 8g pure ethanol. SES = Socioeconomic status.

Table C5. Linear mixed effects model assessing change in drinking days while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, and whether the participant was isolating with children (*N* = 337).

Variable	B	SE	95% CI
Intercept	-0.10	0.17	-0.43 to 0.24
Age	-0.01	0.01	-0.02 to 0.01
Male	-0.55	0.20	-0.94 to -0.16
Non–white	-0.55	0.40	-1.34 to 0.25
SES	0.03	0.11	-0.19 to 0.25
No. Symp.	0.00	0.16	-0.31 to 0.31
Iso. w. Child.	0.10	0.19	-0.27 to 0.48

Note. Models fit using imputed data (*m* = 40). SES = Socioeconomic status.

Table C6. Linear mixed effects model assessing change in heavy drinking days while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, and whether the participant was isolating with children (*N* = 337).

Variable	B	SE	95% CI
Intercept	-0.10	0.12	-0.34 to 0.14
Age	0.00	0.00	-0.01 to 0.01
Male	0.01	0.08	-0.15 to 0.16
Non–white	0.01	0.16	-0.31 to 0.34
SES	0.00	0.04	-0.09 to 0.09
No. Symp.	0.08	0.06	-0.04 to 0.21
Iso. w. Child.	0.00	0.08	-0.15 to 0.15

Note. Models fit using imputed data (*m* = 40). 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women. SES = Socioeconomic status.

Table C7. Linear mixed effects model assessing change in alcohol–related problems while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, and whether the participant was isolating with children (*N* = 337).

Variable	B	SE	95% CI
Intercept	-1.43	0.30	-2.02 to -0.83
Age	0.01	0.01	-0.01 to 0.03
Male	0.21	0.27	-0.33 to 0.75
Non–white	-1.22	0.57	-2.33 to -0.11
SES	-0.17	0.17	-0.5 to 0.16
No. Symp.	-0.30	0.23	-0.74 to 0.15
Iso. w. Child.	-0.05	0.27	-0.58 to 0.48

Note. Models fit using imputed data (*m* = 40). SES = Socioeconomic status.

Table C8. Linear mixed effects model assessing change in stress while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, whether the participant was isolating with children, and perceived stress reactivity (N = 337).

Variable	B	SE	95% CI
Intercept	0.15	0.64	-1.11 to 1.40
Age	-0.01	0.03	-0.07 to 0.05
Male	0.28	0.77	-1.22 to 1.79
Non–white	-0.31	1.53	-3.31 to 2.69
SES	0.38	0.42	-0.45 to 1.2
No. Symp.	0.00	0.53	-1.04 to 1.04
Iso. w. Child.	-1.48	0.71	-2.88 to -0.09
PSRS	0.01	0.05	-0.09 to 0.10

Note. Models fit using imputed data (*m* = 40). SES = Socioeconomic status; PSRS = Perceived Stress Reactivity Scale.

Table C9. Linear mixed effects model assessing change in boredom while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, and whether the participant was isolating with children (N = 337).

Variable	B	SE	95% CI
Intercept	21.22	3.50	14.36 to 28.07
Age	-0.38	0.16	-0.68 to -0.07
Male	-1.71	4.10	-9.76 to 6.33
Non–white	-14.01	8.51	-30.68 to 2.66
SES	-1.67	2.33	-6.25 to 2.90
No. Symp.	-5.78	2.90	-11.47 to -0.09
Iso. w. Child.	-2.39	3.93	-10.08 to 5.310

Note. Models fit using imputed data (m = 40). SES = Socioeconomic status.

Associations between drinking motives and alcohol use behaviour

Table C10. Linear mixed effects models assessing the association between social drinking motives and alcohol use behaviour while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, and whether the participant was isolating with children (*N* = 337).

	Units			Drinking Days			Heavy Days			APQ		
Variable	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**
Intercept	-1.24	0.82	-2.85 to 0.37	-0.09	0.17	-0.43 to 0.25	-0.09	0.08	-0.25 to 0.06	-1.49	0.27	-2.02 to -0.96
DMQ–R Soc	-0.04	0.09	-0.21 to 0.13	0.01	0.02	-0.03 to 0.04	0.00	0.01	-0.02 to 0.02	-0.09	0.02	-0.14 to -0.04
Age	-0.01	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.00	0.01	-0.03 to 0.02
Male	-2.50	0.91	-4.29 to -0.72	-0.56	0.20	-0.95 to -0.16	0.01	0.08	-0.15 to 0.16	0.30	0.27	-0.23 to 0.83
Non–white	-0.51	1.83	-4.10 to 3.07	-0.54	0.40	-1.33 to 0.26	0.01	0.17	-0.31 to 0.34	-1.37	0.55	-2.46 to -0.29
SES	-0.15	0.52	-1.17 to 0.87	0.04	0.11	-0.18 to 0.26	0.00	0.05	-0.09 to 0.09	-0.22	0.17	-0.55 to 0.1
No. Symp.	0.30	0.75	-1.19 to 1.78	0.01	0.16	-0.31 to 0.32	0.08	0.06	-0.04 to 0.21	-0.37	0.23	-0.82 to 0.08
Iso. w. Child.	0.72	0.89	-1.02 to 2.46	0.10	0.19	-0.28 to 0.48	0.00	0.08	-0.15 to 0.15	0.09	0.27	-0.44 to 0.61

Note. Models fit using imputed data (*m* = 40). 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; DMQ–R Soc = Revised Drinking Motives Questionnaire (Social Motives); SES = socioeconomic status.

Table C11. Linear mixed effects models assessing the association between coping drinking motives and alcohol use behaviour while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, and whether the participant was isolating with children (*N* = 337).

	Units			Drinking Days			Heavy Days			APQ		
Variable	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**
Intercept	-1.24	0.81	-2.83 to 0.35	-0.10	0.17	-0.44 to 0.23	-0.09	0.08	-0.24 to 0.07	-1.46	0.29	-2.02 to -0.9
DMQ–R Cop	-0.12	0.12	-0.35 to 0.12	-0.01	0.03	-0.06 to 0.04	0.01	0.01	-0.02 to 0.03	-0.10	0.04	-0.18 to -0.03
Age	-0.01	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.01	0.01	-0.02 to 0.03
Male	-2.55	0.90	-4.31 to -0.79	-0.55	0.20	-0.94 to -0.16	0.01	0.08	-0.15 to 0.16	0.20	0.27	-0.33 to 0.74
Non–white	-0.44	1.82	-4.01 to 3.13	-0.54	0.40	-1.33 to 0.25	0.01	0.16	-0.31 to 0.34	-1.22	0.56	-2.31 to -0.12
SES	-0.22	0.52	-1.24 to 0.8	0.03	0.11	-0.2 to 0.25	0.00	0.05	-0.09 to 0.09	-0.25	0.17	-0.58 to 0.08
No. Symp.	0.37	0.75	-1.1 to 1.84	0.01	0.16	-0.31 to 0.32	0.08	0.06	-0.05 to 0.21	-0.26	0.23	-0.7 to 0.19
Iso. w. Child.	0.75	0.88	-0.97 to 2.47	0.11	0.19	-0.27 to 0.49	0.00	0.08	-0.15 to 0.15	0.03	0.27	-0.5 to 0.55

Note. Models fit using imputed data (*m* = 40). 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; DMQ-R Cop = Revised Drinking Motives Questionnaire (Coping Motives); SES = socioeconomic status.

Table C12. Linear mixed effects models assessing the association between coping drinking motives and alcohol use behaviour while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, and whether the participant was isolating with children (*N* = 337).

	Units			Drinking Days			Heavy Days			APQ		
Variable	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**
Intercept	-1.25	0.82	-2.87 to 0.36	-0.09	0.17	-0.43 to 0.25	-0.09	0.08	-0.25 to 0.07	-1.45	0.29	-2.03 to -0.87
DMQ–R Enh	-0.07	0.09	-0.25 to 0.11	0.01	0.02	-0.03 to 0.04	0.00	0.01	-0.02 to 0.02	-0.04	0.03	-0.09 to 0.02
Age	-0.01	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.01	0.01	-0.02 to 0.03
Male	-2.52	0.90	-4.29 to -0.75	-0.55	0.20	-0.94 to -0.16	0.01	0.08	-0.15 to 0.16	0.22	0.27	-0.31 to 0.76
Non–white	-0.54	1.82	-4.11 to 3.03	-0.54	0.40	-1.33 to 0.25	0.02	0.16	-0.31 to 0.34	-1.27	0.56	-2.38 to -0.16
SES	-0.14	0.52	-1.16 to 0.87	0.03	0.11	-0.19 to 0.25	0.00	0.04	-0.09 to 0.09	-0.18	0.17	-0.51 to 0.15
No. Symp.	0.32	0.75	-1.15 to 1.79	0.01	0.16	-0.31 to 0.32	0.08	0.06	-0.04 to 0.21	-0.30	0.23	-0.75 to 0.15
Iso. w. Child.	0.76	0.89	-0.99 to 2.5	0.09	0.19	-0.29 to 0.47	0.00	0.08	-0.15 to 0.15	0.01	0.27	-0.53 to 0.54

Note. Models fit using imputed data (*m* = 40). 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; DMQ–R Enh = Revised Drinking Motives Questionnaire (Enhancement Motives); SES = socioeconomic status.

Table C13. Linear mixed effects models assessing the association between conformity drinking motives and alcohol use behaviour while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, and whether the participant was isolating with children (*N* = 337).

	Units			Drinking Days			Heavy Days			APQ		
Variable	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**
Intercept	-1.33	0.81	-2.91 to 0.25	-0.11	0.17	-0.45 to 0.23	-0.11	0.08	-0.26 to 0.05	-1.47	0.28	-2.01 to -0.93
DMQ–R Con	-0.26	0.20	-0.66 to 0.13	-0.03	0.04	-0.11 to 0.05	-0.03	0.02	-0.06 to 0.01	-0.16	0.06	-0.28 to -0.04
Age	-0.02	0.04	-0.09 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.00	0.00	0.01	-0.02 to 0.02
Male	-2.47	0.90	-4.24 to -0.71	-0.54	0.20	-0.93 to -0.15	0.01	0.08	-0.14 to 0.17	0.24	0.27	-0.29 to 0.77
Non–white	-0.35	1.82	-3.91 to 3.21	-0.54	0.40	-1.33 to 0.25	0.03	0.16	-0.29 to 0.35	-1.18	0.56	-2.28 to -0.09
SES	-0.15	0.52	-1.17 to 0.86	0.03	0.11	-0.19 to 0.25	0.00	0.04	-0.09 to 0.08	-0.18	0.17	-0.51 to 0.14
No. Symp.	0.37	0.74	-1.09 to 1.83	0.01	0.16	-0.30 to 0.32	0.09	0.06	-0.04 to 0.21	-0.27	0.22	-0.71 to 0.17
Iso. w. Child.	0.81	0.88	-0.92 to 2.53	0.12	0.19	-0.26 to 0.50	0.01	0.08	-0.14 to 0.17	0.05	0.27	-0.48 to 0.58

Note. Models fit using imputed data (*m* = 40). 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; DMQ–R Con = Revised Drinking Motives Questionnaire (Conformity Motives); SES = socioeconomic status.

Associations between impulse control, stress, boredom and alcohol use

Table C14. Linear mixed effects models assessing the association between a change in stress and alcohol use behaviour while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, and whether the participant was isolating with children (*N* = 337).

	Units			Drinking Days			Heavy Days			APQ		
Variable	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**
Intercept	-1.1	0.83	-2.72 to 0.52	-0.08	0.17	-0.42 to 0.25	-0.09	0.08	-0.25 to 0.07	-1.42	0.30	-2.01 to -0.83
SOS–S Change	-0.06	0.07	-0.18 to 0.07	-0.01	0.01	-0.04 to 0.02	0.00	0.01	-0.02 to 0.01	0.01	0.02	-0.04 to 0.05
Age	-0.01	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.01	0.01	-0.02 to 0.03
Male	-2.53	0.96	-4.41 to -0.64	-0.57	0.21	-0.98 to -0.16	0.04	0.08	-0.12 to 0.2	0.12	0.28	-0.43 to 0.67
Non–white	-0.54	1.83	-4.12 to 3.05	-0.55	0.40	-1.34 to 0.24	0.01	0.16	-0.31 to 0.33	-1.22	0.57	-2.33 to -0.11
SES	-0.11	0.52	-1.12 to 0.91	0.03	0.11	-0.19 to 0.25	0.00	0.04	-0.08 to 0.09	-0.18	0.17	-0.51 to 0.15
No. Symp.	0.33	0.75	-1.15 to 1.8	0.01	0.16	-0.31 to 0.32	0.08	0.06	-0.05 to 0.2	-0.28	0.23	-0.73 to 0.17
Iso. w. Child.	0.59	0.87	-1.12 to 2.3	0.09	0.19	-0.28 to 0.47	-0.01	0.08	-0.16 to 0.14	-0.02	0.27	-0.55 to 0.51
PSRS	0.00	0.06	-0.12 to 0.13	0.00	0.01	-0.03 to 0.02	0.01	0.00	0.00 to 0.02	-0.02	0.02	-0.05 to 0.01

Note. Models fit using imputed data (*m* = 40). 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; SOS-S = Short Stress Overload Scale; PSRS = Perceived Stress Reactivity Scale.

Table C15. Linear mixed effects models assessing the association between a change in boredom and alcohol use behaviour while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, and whether the participant was isolating with children (*N* = 337).

	Units			Drinking Days			Heavy Days			APQ		
Variable	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**
Intercept	-1.16	0.81	-2.75 to 0.43	-0.11	0.17	-0.44 to 0.23	-0.09	0.08	-0.24 to 0.07	-1.43	0.30	-2.03 to -0.84
MSBS Change	-0.01	0.01	-0.03 to 0.02	0.00	0.00	0.00 to 0.01	0.00	0.00	0.00 to 0.00	0.00	0.00	-0.01 to 0.01
Age	-0.01	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.01	0.01	-0.01 to 0.03
Male	-2.56	0.90	-4.32 to -0.79	-0.54	0.20	-0.93 to -0.15	0.01	0.08	-0.15 to 0.16	0.21	0.27	-0.32 to 0.75
Non–white	-0.56	1.83	-4.14 to 3.02	-0.49	0.40	-1.28 to 0.3	0.00	0.17	-0.32 to 0.32	-1.19	0.57	-2.31 to -0.08
SES	-0.14	0.52	-1.15 to 0.88	0.04	0.11	-0.18 to 0.26	0.00	0.04	-0.09 to 0.09	-0.17	0.17	-0.50 to 0.17
No. Symp.	0.29	0.75	-1.19 to 1.77	0.03	0.16	-0.28 to 0.34	0.08	0.06	-0.05 to 0.2	-0.29	0.23	-0.73 to 0.16
Iso. w. Child.	0.64	0.87	-1.07 to 2.36	0.11	0.19	-0.26 to 0.49	0.00	0.08	-0.15 to 0.15	-0.04	0.27	-0.57 to 0.49

Note. Models fit using imputed data (*m* = 40). 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; MSBS = Multidimensional State Boredom Scale.

Table C16. Linear mixed effects models assessing the association between impulse control and alcohol use behaviour while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, and whether the participant was isolating with children (N = 337).

	Units			Drinking Days			Heavy Days			APQ		
Variable	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**
Negative Urgency												
Intercept	-1.19	0.81	-2.78 to 0.41	-0.10	0.17	-0.43 to 0.24	-0.09	0.08	-0.25 to 0.06	-1.41	0.28	-1.95 to -0.87
Neg. Urg.	-0.09	0.16	-0.41 to 0.23	0.00	0.04	-0.07 to 0.07	-0.01	0.01	-0.04 to 0.02	-0.12	0.05	-0.22 to -0.02
Age	-0.01	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.00	0.01	-0.02 to 0.03
Male	-2.55	0.90	-4.31 to -0.79	-0.55	0.20	-0.94 to -0.16	0.01	0.08	-0.15 to 0.16	0.20	0.27	-0.33 to 0.73
Non–white	-0.44	1.83	-4.02 to 3.14	-0.55	0.40	-1.34 to 0.24	0.02	0.17	-0.3 to 0.34	-1.19	0.56	-2.28 to -0.09
SES	-0.15	0.52	-1.16 to 0.87	0.03	0.11	-0.19 to 0.25	0.00	0.04	-0.09 to 0.09	-0.19	0.17	-0.52 to 0.14
No. Symp.	0.32	0.74	-1.14 to 1.78	0.00	0.16	-0.31 to 0.31	0.08	0.06	-0.04 to 0.21	-0.30	0.23	-0.76 to 0.15
Iso. w. Child.	0.68	0.88	-1.04 to 2.39	0.10	0.19	-0.27 to 0.48	0.00	0.08	-0.15 to 0.15	-0.03	0.27	-0.55 to 0.50
Lack of Premeditation												
Intercept	-1.16[a]	0.81	-2.75 to 0.44	-0.11	0.17	-0.44 to 0.23	-0.09	0.08	-0.24 to 0.07	-1.42	0.30	-2.01 to -0.83
Premed.	-0.19[a]	0.22	-0.63 to 0.24	0.05	0.05	-0.04 to 0.14	-0.05	0.02	-0.08 to -0.01	-0.07	0.07	-0.2 to 0.06
Age	-0.01[a]	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.00	0.01	0.01	-0.02 to 0.03
Male	-2.54[a]	0.90	-4.30 to -0.78	-0.55	0.20	-0.94 to -0.16	0.01	0.08	-0.14 to 0.16	0.22	0.27	-0.32 to 0.75
Non–white	-0.42[a]	1.83	-4.01 to 3.16	-0.56	0.40	-1.35 to 0.23	0.03	0.16	-0.29 to 0.35	-1.20	0.57	-2.31 to -0.09
SES	-0.17[a]	0.52	-1.19 to 0.84	0.04	0.11	-0.17 to 0.26	-0.01	0.04	-0.10 to 0.08	-0.19	0.17	-0.53 to 0.15
No. Symp.	0.36[a]	0.75	-1.11 to 1.82	0.00	0.16	-0.32 to 0.31	0.09	0.06	-0.03 to 0.21	-0.29	0.23	-0.73 to 0.16
Iso. w. Child.	0.61[a]	0.88	-1.11 to 2.34	0.12	0.19	-0.26 to 0.49	-0.01	0.08	-0.16 to 0.14	-0.06	0.27	-0.59 to 0.47

Lack of Perseverance												
Intercept	-1.15	0.81	-2.73 to 0.43	-0.10	0.17	-0.43 to 0.24	-0.09	0.08	-0.24 to 0.07	-1.43	0.30	-2.02 to -0.83
Persev.	-0.26	0.21	-0.67 to 0.14	-0.01	0.05	-0.11 to 0.08	-0.03	0.02	-0.07 to 0.01	-0.01	0.07	-0.14 to 0.12
Age	-0.01	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.01	0.01	-0.01 to 0.03
Male	-2.48	0.89	-4.24 to -0.73	-0.55	0.20	-0.94 to -0.16	0.01	0.08	-0.14 to 0.17	0.22	0.27	-0.32 to 0.75
Non–white	-0.46	1.82	-4.03 to 3.11	-0.55	0.40	-1.34 to 0.25	0.02	0.16	-0.31 to 0.34	-1.22	0.57	-2.33 to -0.11
SES	-0.18	0.52	-1.19 to 0.83	0.03	0.11	-0.19 to 0.25	-0.01	0.04	-0.09 to 0.08	-0.17	0.17	-0.51 to 0.17
No. Symp.	0.38	0.75	-1.09 to 1.85	0.01	0.16	-0.31 to 0.32	0.09	0.06	-0.04 to 0.21	-0.29	0.23	-0.74 to 0.15
Iso. w. Child.	0.57	0.88	-1.15 to 2.29	0.10	0.19	-0.28 to 0.48	-0.01	0.08	-0.16 to 0.14	-0.05	0.27	-0.58 to 0.48
Sensation Seeking												
Intercept	-1.14	0.82	-2.75 to 0.47	-0.08	0.17	-0.42 to 0.26	-0.09	0.08	-0.25 to 0.06	-1.47	0.30	-2.06 to -0.87
Sensat.	0.06	0.16	-0.25 to 0.37	0.03	0.03	-0.03 to 0.10	0.00	0.01	-0.03 to 0.03	-0.08	0.05	-0.17 to 0.01
Age	-0.01	0.04	-0.08 to 0.07	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.00	0.01	-0.02 to 0.03
Male	-2.62	0.91	-4.41 to -0.83	-0.59	0.20	-0.98 to -0.19	0.01	0.08	-0.15 to 0.17	0.30	0.28	-0.24 to 0.85
Non–white	-0.50	1.83	-4.08 to 3.08	-0.56	0.40	-1.35 to 0.23	0.02	0.16	-0.31 to 0.34	-1.20	0.56	-2.31 to -0.10
SES	-0.12	0.52	-1.14 to 0.9	0.04	0.11	-0.18 to 0.26	0.00	0.05	-0.09 to 0.09	-0.19	0.17	-0.52 to 0.15
No. Symp.	0.32	0.75	-1.15 to 1.78	0.00	0.16	-0.31 to 0.31	0.08	0.06	-0.04 to 0.21	-0.28	0.22	-0.72 to 0.16
Iso. w. Child.	0.65	0.88	-1.07 to 2.37	0.09	0.19	-0.28 to 0.47	0.00	0.08	-0.15 to 0.15	-0.02	0.27	-0.55 to 0.52
Positive Urgency												
Intercept	-1.21	0.82	-2.81 to 0.40	-0.09	0.17	-0.43 to 0.24	-0.10	0.08	-0.25 to 0.06	-1.47	0.29	-2.03 to -0.91
Pos. Urg.	-0.06	0.20	-0.45 to 0.33	0.00	0.04	-0.08 to 0.09	-0.01	0.02	-0.04 to 0.03	-0.13	0.06	-0.24 to -0.01

Age	-0.01	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.00	0.01	-0.02 to 0.03
Male	-2.52	0.90	-4.29 to -0.75	-0.55	0.20	-0.94 to -0.16	0.01	0.08	-0.14 to 0.16	0.26	0.27	-0.27 to 0.8
Non–white	-0.41	1.85	-4.03 to 3.21	-0.55	0.40	-1.34 to 0.24	0.02	0.17	-0.3 to 0.35	-1.10	0.57	-2.21 to 0.01
SES	-0.15	0.53	-1.19 to 0.88	0.03	0.11	-0.19 to 0.26	0.00	0.05	-0.09 to 0.09	-0.22	0.17	-0.56 to 0.11
No. Symp.	0.32	0.74	-1.14 to 1.79	0.01	0.16	-0.31 to 0.32	0.08	0.06	-0.04 to 0.21	-0.32	0.23	-0.77 to 0.12
Iso. w. Child.	0.69	0.88	-1.04 to 2.42	0.10	0.19	-0.28 to 0.48	0.00	0.08	-0.15 to 0.16	0.03	0.27	-0.51 to 0.56
						DOSPERT						
Intercept	-1.29[a]	0.82	-2.90 to 0.32	-0.11	0.17	-0.45 to 0.22	-0.10	0.08	-0.26 to 0.05	-1.53	0.32	-2.16 to -0.90
DOSPERT	-0.05[a]	0.02	-0.09 to 0.00	-0.01	0.00	-0.01 to 0.00	0.00	0.00	-0.01 to 0.00	-0.02	0.01	-0.04 to -0.01
Age	-0.02[a]	0.04	-0.09 to 0.05	-0.01	0.01	-0.02 to 0.00	0.00	0.00	-0.01 to 0.00	0.00	0.01	-0.02 to 0.02
Male	-2.24[a]	0.92	-4.04 to -0.45	-0.51	0.20	-0.91 to -0.11	0.03	0.08	-0.12 to 0.18	0.38	0.27	-0.16 to 0.91
Non–white	-0.65[a]	1.82	-4.21 to 2.92	-0.56	0.40	-1.35 to 0.23	0.00	0.16	-0.32 to 0.32	-1.27	0.56	-2.36 to -0.17
SES	-0.20[a]	0.52	-1.22 to 0.81	0.02	0.11	-0.20 to 0.24	-0.01	0.04	-0.09 to 0.08	-0.21	0.17	-0.54 to 0.12
No. Symp.	0.39[a]	0.74	-1.06 to 1.84	0.01	0.16	-0.30 to 0.32	0.09	0.06	-0.04 to 0.21	-0.27	0.22	-0.71 to 0.17
Iso. w. Child.	0.76[a]	0.87	-0.95 to 2.47	0.11	0.19	-0.26 to 0.49	0.01	0.08	-0.14 to 0.16	0.00	0.27	-0.52 to 0.53

Note. Models fit using imputed data (m = 40). 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; DOSPERT = Domain–Specific Risk–taking Scale.

[a] model fit using the Broyden–Fletcher–Goldfarb–Shanno algorithm to allow convergence.

Table C17. Linear mixed effects models assessing the interaction between impulse control and a change in stress in relation to alcohol use behaviour while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, and whether the participant was isolating with children (N = 337).

	Units			Drinking Days			Heavy Days			APQ		
Variable	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**
						Negative Urgency						
Intercept	-1.13[a]	0.83	-2.77 to 0.50	-0.08	0.17	-0.42 to 0.25	-0.10	0.08	-0.25 to 0.06	-1.42	0.28	-1.96 to -0.88
Interaction	-0.01[a]	0.03	-0.07 to 0.04	0.00	0.01	-0.01 to 0.01	0.00	0.00	-0.01 to 0.01	0.00	0.01	-0.02 to 0.02
Neg. Urg.	-0.09[a]	0.18	-0.44 to 0.25	0.01	0.04	-0.07 to 0.08	-0.02	0.02	-0.05 to 0.01	-0.12	0.06	-0.23 to -0.01
SOS–S Change	-0.05[a]	0.07	-0.18 to 0.08	-0.01	0.01	-0.04 to 0.02	0.00	0.01	-0.01 to 0.01	0.01	0.02	-0.03 to 0.05
Age	-0.01[a]	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.00	0.01	-0.02 to 0.02
Male	-2.49[a]	0.97	-4.4 to -0.59	-0.57	0.21	-0.99 to -0.16	0.05	0.08	-0.11 to 0.21	0.17	0.28	-0.39 to 0.72
Non–white	-0.46[a]	1.83	-4.05 to 3.12	-0.55	0.40	-1.34 to 0.24	0.02	0.16	-0.30 to 0.34	-1.18	0.56	-2.28 to -0.08
SES	-0.11[a]	0.52	-1.13 to 0.91	0.03	0.11	-0.19 to 0.26	0.00	0.04	-0.09 to 0.09	-0.19	0.17	-0.52 to 0.14
No. Symp.	0.29[a]	0.75	-1.18 to 1.76	0.00	0.16	-0.31 to 0.32	0.08	0.06	-0.05 to 0.20	-0.30	0.23	-0.75 to 0.15
Iso. w. Child.	0.63[a]	0.88	-1.09 to 2.35	0.09	0.19	-0.28 to 0.47	-0.01	0.08	-0.16 to 0.14	0.00	0.27	-0.53 to 0.52
PSRS	0.02[a]	0.07	-0.12 to 0.15	-0.01	0.01	-0.03 to 0.02	0.01	0.01	0.00 to 0.020	-0.01	0.02	-0.04 to 0.03
						Lack of Premeditation						
Intercept	-1.09	0.83	-2.72 to 0.55	-0.10	0.17	-0.44 to 0.24	-0.09	0.08	-0.24 to 0.07	-1.40	0.30	-2.00 to -0.81
Interaction	-0.04	0.05	-0.13 to 0.05	-0.01	0.01	-0.03 to 0.01	0.00	0.00	-0.01 to 0.00	0.01	0.01	-0.01 to 0.04
Premed.	-0.21	0.22	-0.64 to 0.23	0.05	0.05	-0.04 to 0.14	-0.05	0.02	-0.08 to -0.01	-0.07	0.07	-0.2 to 0.06
SOS–S Change	-0.06	0.07	-0.19 to 0.07	-0.01	0.01	-0.04 to 0.02	-0.01	0.01	-0.02 to 0.01	0.00	0.02	-0.04 to 0.05
Age	-0.01	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.00	0.01	-0.02 to 0.03

Male	-2.46	0.95	-4.32 to -0.60	-0.56	0.21	-0.96 to -0.16	0.05	0.08	-0.11 to 0.21	0.11	0.28	-0.45 to 0.66
Non–white	-0.49	1.82	-4.07 to 3.08	-0.56	0.40	-1.35 to 0.23	0.02	0.16	-0.30 to 0.34	-1.21	0.56	-2.31 to -0.10
SES	-0.19	0.52	-1.20 to 0.83	0.04	0.11	-0.18 to 0.26	-0.01	0.04	-0.09 to 0.08	-0.19	0.17	-0.53 to 0.15
No. Symp.	0.32	0.74	-1.14 to 1.77	-0.01	0.16	-0.32 to 0.3	0.08	0.06	-0.04 to 0.21	-0.26	0.23	-0.71 to 0.19
Iso. w. Child.	0.43	0.88	-1.31 to 2.16	0.09	0.19	-0.28 to 0.46	-0.03	0.08	-0.18 to 0.12	-0.01	0.27	-0.54 to 0.52
PSRS	0.00	0.06	-0.12 to 0.13	0.00	0.01	-0.03 to 0.02	0.01	0.00	0.00 to 0.02	-0.02	0.02	-0.05 to 0.01
Lack of Perseverance												
Intercept	-1.06	0.82	-2.67 to 0.54	-0.08	0.17	-0.42 to 0.26	-0.09	0.08	-0.24 to 0.07	-1.42	0.30	-2.00 to -0.83
Interaction	-0.02	0.04	-0.10 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.00	0.01	0.01	-0.01 to 0.03
Persev.	-0.27	0.21	-0.67 to 0.14	-0.02	0.05	-0.11 to 0.07	-0.03	0.02	-0.06 to 0.01	-0.01	0.07	-0.14 to 0.12
SOS–S Change	-0.06	0.06	-0.19 to 0.07	-0.01	0.01	-0.04 to 0.02	0.00	0.01	-0.02 to 0.01	0.01	0.02	-0.03 to 0.05
Age	-0.01	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.00	0.01	-0.02 to 0.03
Male	-2.42	0.95	-4.29 to -0.56	-0.54	0.21	-0.95 to -0.13	0.05	0.08	-0.11 to 0.21	0.10	0.28	-0.46 to 0.66
Non–white	-0.47	1.82	-4.03 to 3.09	-0.52	0.40	-1.31 to 0.26	0.02	0.16	-0.31 to 0.34	-1.25	0.57	-2.36 to -0.14
SES	-0.17	0.52	-1.19 to 0.85	0.03	0.11	-0.19 to 0.25	0.00	0.04	-0.09 to 0.09	-0.18	0.17	-0.52 to 0.15
No. Symp.	0.33	0.74	-1.13 to 1.79	-0.01	0.16	-0.33 to 0.30	0.08	0.06	-0.05 to 0.21	-0.26	0.23	-0.71 to 0.19
Iso. w. Child.	0.46	0.88	-1.26 to 2.18	0.07	0.19	-0.31 to 0.45	-0.02	0.08	-0.17 to 0.12	-0.01	0.27	-0.54 to 0.53
PSRS	0.00	0.06	-0.12 to 0.12	0.00	0.01	-0.03 to 0.02	0.01	0.00	0.00 to 0.02	-0.02	0.02	-0.05 to 0.01
Sensation Seeking												
Intercept	-1.08	0.82	-2.68 to 0.53	-0.06	0.17	-0.40 to 0.27	-0.09	0.08	-0.25 to 0.07	-1.46	0.29	-2.03 to -0.89
Interaction	0.02	0.02	-0.02 to 0.07	0.00	0.01	-0.01 to 0.01	0.00	0.00	0.00 to 0.01	-0.01	0.01	-0.02 to 0.01

Sensat.	0.08	0.16	-0.24 to 0.39	0.03	0.03	-0.03 to 0.1	0.00	0.01	-0.03 to 0.03	-0.09	0.05	-0.18 to 0.00
SOS–S Change	-0.05	0.07	-0.18 to 0.08	-0.01	0.01	-0.04 to 0.02	0.00	0.01	-0.01 to 0.01	0.00	0.02	-0.04 to 0.05
Age	-0.01	0.04	-0.08 to 0.07	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.00	0.01	-0.02 to 0.02
Male	-2.46	0.97	-4.37 to -0.55	-0.58	0.21	-0.99 to -0.17	0.05	0.08	-0.11 to 0.21	0.15	0.29	-0.41 to 0.72
Non–white	-0.75	1.83	-4.33 to 2.84	-0.60	0.40	-1.38 to 0.19	-0.01	0.16	-0.33 to 0.32	-1.12	0.56	-2.23 to -0.02
SES	-0.04	0.52	-1.07 to 0.99	0.05	0.11	-0.17 to 0.27	0.01	0.05	-0.08 to 0.10	-0.22	0.17	-0.55 to 0.11
No. Symp.	0.24	0.77	-1.26 to 1.75	-0.01	0.16	-0.33 to 0.31	0.07	0.06	-0.06 to 0.20	-0.23	0.23	-0.69 to 0.22
Iso. w. Child.	0.44	0.89	-1.30 to 2.18	0.06	0.19	-0.31 to 0.44	-0.02	0.08	-0.17 to 0.13	0.06	0.27	-0.47 to 0.60
PSRS	0.00	0.06	-0.12 to 0.13	0.00	0.01	-0.03 to 0.02	0.01	0.00	0.00 to 0.02	-0.02	0.02	-0.06 to 0.01
Positive Urgency												
Intercept	-1.17	0.84	-2.81 to 0.48	-0.10	0.17	-0.44 to 0.24	-0.09	0.08	-0.25 to 0.06	-1.47	0.28	-2.03 to -0.92
Interaction	-0.01	0.04	-0.09 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	0.00 to 0.01	-0.01	0.01	-0.03 to 0.01
Pos. Urg.	-0.06	0.20	-0.46 to 0.34	0.01	0.04	-0.08 to 0.09	-0.01	0.02	-0.04 to 0.02	-0.12	0.06	-0.24 to 0.00
SOS–S Change	-0.06	0.07	-0.19 to 0.07	-0.01	0.01	-0.04 to 0.02	0.00	0.01	-0.02 to 0.01	0.00	0.02	-0.04 to 0.05
Age	-0.01	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.00	0.01	-0.02 to 0.02
Male	-2.50	0.97	-4.41 to -0.6	-0.58	0.21	-0.99 to -0.16	0.05	0.08	-0.11 to 0.21	0.18	0.28	-0.38 to 0.74
Non–white	-0.36	1.84	-3.98 to 3.25	-0.50	0.40	-1.29 to 0.29	0.01	0.17	-0.31 to 0.34	-1.04	0.57	-2.16 to 0.07
SES	-0.12	0.53	-1.16 to 0.92	0.04	0.11	-0.18 to 0.27	0.00	0.05	-0.09 to 0.09	-0.22	0.17	-0.56 to 0.11
No. Symp.	0.35	0.76	-1.14 to 1.84	0.02	0.16	-0.29 to 0.34	0.07	0.06	-0.05 to 0.2	-0.30	0.23	-0.75 to 0.16
Iso. w. Child.	0.65	0.89	-1.09 to 2.39	0.10	0.19	-0.28 to 0.48	-0.01	0.08	-0.16 to 0.14	0.06	0.27	-0.48 to 0.59
PSRS	0.00	0.06	-0.12 to 0.13	-0.01	0.01	-0.03 to 0.02	0.01	0.00	0.00 to 0.02	-0.02	0.02	-0.05 to 0.02

						DOSPERT						
Intercept	-1.2	0.83	-2.83 to 0.42	-0.10	0.17	-0.44 to 0.24	-0.10	0.08	-0.26 to 0.06	-1.52	0.33	-2.16 to -0.88
Interaction	0.00	0.00	-0.01 to 0.01	0.00	0.00	0.00 to 0.00	0.00	0.00	0.00 to 0.00	0.00	0.00	0.00 to 0.00
DOSPERT	-0.05	0.02	-0.09 to -0.01	-0.01	0.00	-0.02 to 0.00	0.00	0.00	-0.01 to 0.00	-0.02	0.01	-0.04 to -0.01
SOS–S Change	-0.06	0.07	-0.19 to 0.06	-0.01	0.01	-0.04 to 0.02	0.00	0.01	-0.02 to 0.01	0.00	0.02	-0.04 to 0.04
Age	-0.03	0.04	-0.10 to 0.05	-0.01	0.01	-0.03 to 0.00	0.00	0.00	-0.01 to 0.01	0.00	0.01	-0.03 to 0.02
Male	-2.27	0.96	-4.15 to -0.38	-0.54	0.21	-0.95 to -0.13	0.07	0.08	-0.09 to 0.23	0.28	0.28	-0.27 to 0.84
Non–white	-0.73	1.82	-4.29 to 2.84	-0.56	0.40	-1.35 to 0.23	-0.02	0.16	-0.34 to 0.30	-1.31	0.56	-2.41 to -0.21
SES	-0.19	0.52	-1.20 to 0.82	0.02	0.11	-0.20 to 0.24	0.00	0.04	-0.09 to 0.09	-0.22	0.17	-0.55 to 0.10
No. Symp.	0.40	0.75	-1.07 to 1.87	0.02	0.16	-0.29 to 0.34	0.08	0.06	-0.05 to 0.20	-0.26	0.23	-0.70 to 0.19
Iso. w. Child.	0.68	0.87	-1.01 to 2.38	0.10	0.19	-0.27 to 0.48	0.00	0.08	-0.15 to 0.14	0.03	0.27	-0.49 to 0.55
PSRS	-0.01	0.06	-0.14 to 0.12	-0.01	0.01	-0.03 to 0.02	0.01	0.01	0.00 to 0.02	-0.03	0.02	-0.06 to 0.01

Note. Models fit using imputed data (m = 40). 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; DOSPERT = Domain–Specific Risk–taking Scale; SOS-S = Short Stress Overload Scale

[a] model fit using the Broyden–Fletcher–Goldfarb–Shanno algorithm to allow convergence.

Table C18. Linear mixed effects models assessing the interaction between impulse control and a change in boredom in relation to alcohol use behaviour while controlling for age, gender, ethnicity, SES, the number of COVID–19 symptoms, and whether the participant was isolating with children (*N* = 337).

	Units			Drinking Days			Heavy Days			APQ		
Variable	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**	**B**	**SE**	**95% CI**
						Negative Urgency						
Intercept	-1.18	0.80	-2.76 to 0.40	-0.11	0.17	-0.44 to 0.22	-0.09	0.08	-0.25 to 0.06	-1.42	0.28	-1.96 to -0.87
Interaction	-0.01	0.01	-0.02 to 0.01	0.00	0.00	0.00 to 0.00	0.00	0.00	0.00 to 0.00	0.00	0.00	0.00 to 0.00
Neg. Urg.	-0.09	0.16	-0.41 to 0.23	-0.01	0.04	-0.07 to 0.06	-0.01	0.01	-0.04 to 0.02	-0.12	0.05	-0.22 to -0.02
MSBS Change	-0.01	0.01	-0.03 to 0.02	0.00	0.00	0.00 to 0.01	0.00	0.00	0.00 to 0.00	0.00	0.00	-0.01 to 0.01
Age	-0.01	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.00	0.01	-0.02 to 0.03
Male	-2.6	0.90	-4.35 to -0.84	-0.55	0.20	-0.94 to -0.16	0.00	0.08	-0.15 to 0.16	0.20	0.27	-0.33 to 0.73
Non–white	-0.59	1.83	-4.17 to 2.99	-0.50	0.40	-1.29 to 0.29	0.00	0.17	-0.32 to 0.32	-1.16	0.56	-2.27 to -0.06
SES	-0.28	0.51	-1.29 to 0.72	0.01	0.11	-0.21 to 0.23	-0.01	0.04	-0.10 to 0.08	-0.20	0.17	-0.53 to 0.13
No. Symp.	0.45	0.76	-1.04 to 1.95	0.06	0.16	-0.25 to 0.37	0.08	0.06	-0.04 to 0.21	-0.27	0.23	-0.73 to 0.19
Iso. w. Child.	0.75	0.89	-0.99 to 2.49	0.13	0.19	-0.25 to 0.51	0.00	0.08	-0.15 to 0.15	-0.01	0.27	-0.54 to 0.51
						Lack of Premeditation						
Intercept	-1.15a	0.82	-2.76 to 0.45	-0.13	0.17	-0.46 to 0.20	-0.09	0.08	-0.24 to 0.07	-1.43	0.30	-2.03 to -0.84
Interaction	-0.02a	0.01	-0.04 to -0.01	0.00	0.00	-0.01 to 0.00	0.00	0.00	0.00 to 0.00	0.00	0.00	-0.01 to 0.00
Premed.	-0.14a	0.22	-0.58 to 0.30	0.07	0.05	-0.02 to 0.16	-0.04	0.02	-0.08 to 0.00	-0.06	0.07	-0.19 to 0.07
MSBS Change	-0.01a	0.01	-0.04 to 0.01	0.00	0.00	0.00 to 0.01	0.00	0.00	0.00 to 0.00	0.00	0.00	-0.01 to 0.01
Age	-0.02a	0.04	-0.08 to 0.05	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.00	0.01	0.01	-0.02 to 0.03
Male	-2.51a	0.89	-4.26 to -0.76	-0.53	0.20	-0.92 to -0.14	0.01	0.08	-0.14 to 0.16	0.22	0.27	-0.31 to 0.76

Non–white	-0.98a	1.80	-4.50 to 2.55	-0.58	0.40	-1.36 to 0.20	-0.03	0.16	-0.35 to 0.29	-1.23	0.57	-2.34 to -0.11
SES	-0.17a	0.51	-1.17 to 0.82	0.06	0.11	-0.16 to 0.27	-0.01	0.04	-0.10 to 0.07	-0.18	0.17	-0.52 to 0.15
No. Symp.	0.20a	0.74	-1.27 to 1.66	0.00	0.16	-0.32 to 0.31	0.07	0.06	-0.05 to 0.19	-0.29	0.23	-0.74 to 0.15
Iso. w. Child.	0.57a	0.87	-1.14 to 2.28	0.12	0.19	-0.25 to 0.49	-0.02	0.07	-0.16 to 0.13	-0.06	0.27	-0.59 to 0.47
Lack of Perseverance												
Intercept	-1.14	0.80	-2.72 to 0.43	-0.11	0.17	-0.44 to 0.22	-0.09	0.08	-0.24 to 0.06	-1.43	0.30	-2.02 to -0.84
Interaction	-0.01	0.01	-0.02 to 0.01	0.00	0.00	0.00 to 0.00	0.00	0.00	0.00 to 0.00	0.00	0.00	0.00 to 0.01
Persev.	-0.29	0.21	-0.70 to 0.11	-0.01	0.05	-0.11 to 0.08	-0.04	0.02	-0.07 to 0.00	0.00	0.07	-0.14 to 0.13
MSBS Change	-0.01	0.01	-0.03 to 0.01	0.00	0.00	0.00 to 0.01	0.00	0.00	0.00 to 0.00	0.00	0.00	-0.01 to 0.01
Age	-0.01	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.01	0.01	-0.01 to 0.03
Male	-2.48	0.89	-4.23 to -0.72	-0.54	0.20	-0.93 to -0.15	0.02	0.08	-0.13 to 0.17	0.21	0.27	-0.33 to 0.75
Non–white	-0.56	1.82	-4.13 to 3.01	-0.49	0.40	-1.28 to 0.30	0.00	0.16	-0.32 to 0.32	-1.20	0.57	-2.31 to -0.08
SES	-0.17	0.52	-1.18 to 0.84	0.04	0.11	-0.17 to 0.26	0.00	0.04	-0.09 to 0.08	-0.18	0.17	-0.51 to 0.16
No. Symp.	0.33	0.75	-1.15 to 1.81	0.03	0.16	-0.29 to 0.34	0.08	0.06	-0.04 to 0.20	-0.28	0.23	-0.73 to 0.16
Iso. w. Child.	0.51	0.88	-1.21 to 2.23	0.10	0.19	-0.27 to 0.48	-0.02	0.08	-0.17 to 0.13	-0.03	0.27	-0.56 to 0.49
Sensation Seeking												
Intercept	-1.08	0.83	-2.70 to 0.54	-0.08	0.17	-0.42 to 0.25	-0.08	0.08	-0.24 to 0.07	-1.45	0.30	-2.04 to -0.85
Interaction	0.00	0.00	-0.01 to 0.01	0.00	0.00	0.00 to 0.00	0.00	0.00	0.00 to 0.00	0.00	0.00	0.00 to 0.00
Sensat.	0.07	0.16	-0.24 to 0.38	0.03	0.03	-0.03 to 0.10	0.00	0.01	-0.03 to 0.03	-0.08	0.05	-0.17 to 0.01
MSBS Change	-0.01	0.01	-0.03 to 0.02	0.00	0.00	0.00 to 0.01	0.00	0.00	0.00 to 0.00	0.00	0.00	-0.01 to 0.01
Age	-0.01	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.00	0.01	-0.02 to 0.03

Male	-2.68	0.91	-4.47 to -0.89	-0.59	0.20	-0.99 to -0.20	0.00	0.08	-0.16 to 0.15	0.28	0.28	-0.27 to 0.82
Non–white	-0.64	1.83	-4.23 to 2.96	-0.50	0.40	-1.29 to 0.28	-0.01	0.17	-0.33 to 0.32	-1.19	0.56	-2.3 to -0.09
SES	-0.15	0.52	-1.17 to 0.87	0.04	0.11	-0.18 to 0.26	-0.01	0.05	-0.10 to 0.08	-0.20	0.17	-0.54 to 0.13
No. Symp.	0.30	0.76	-1.19 to 1.79	0.03	0.16	-0.28 to 0.34	0.08	0.06	-0.05 to 0.21	-0.25	0.23	-0.70 to 0.19
Iso. w. Child.	0.63	0.87	-1.08 to 2.34	0.10	0.19	-0.27 to 0.47	0.00	0.08	-0.15 to 0.15	-0.02	0.27	-0.55 to 0.51
Positive Urgency												
Intercept	-1.20a	0.81	-2.78 to 0.39	-0.11	0.17	-0.44 to 0.23	-0.09	0.08	-0.25 to 0.06	-1.47	0.29	-2.04 to -0.91
Interaction	-0.01a	0.01	-0.02 to 0.01	0.00	0.00	0.00 to 0.00	0.00	0.00	0.00 to 0.00	0.00	0.00	-0.01 to 0.00
Pos. Urg.	-0.02a	0.20	-0.41 to 0.37	0.00	0.04	-0.08 to 0.08	0.00	0.02	-0.03 to 0.03	-0.11	0.06	-0.23 to 0.01
MSBS Change	-0.01a	0.01	-0.03 to 0.01	0.00	0.00	0.00 to 0.01	0.00	0.00	0.00 to 0.00	0.00	0.00	-0.01 to 0.01
Age	-0.01a	0.04	-0.08 to 0.06	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.01	0.01	0.01	-0.02 to 0.03
Male	-2.57a	0.90	-4.33 to -0.81	-0.54	0.20	-0.93 to -0.15	0.00	0.08	-0.15 to 0.15	0.25	0.27	-0.28 to 0.78
Non–white	-0.37a	1.84	-3.99 to 3.24	-0.49	0.40	-1.28 to 0.30	0.03	0.17	-0.30 to 0.35	-1.02	0.57	-2.14 to 0.09
SES	-0.21a	0.53	-1.25 to 0.82	0.04	0.11	-0.18 to 0.26	-0.01	0.05	-0.1 to 0.07	-0.25	0.17	-0.58 to 0.09
No. Symp.	0.38a	0.76	-1.12 to 1.87	0.03	0.16	-0.28 to 0.34	0.09	0.06	-0.04 to 0.21	-0.27	0.23	-0.72 to 0.19
Iso. w. Child.	0.72a	0.88	-1.00 to 2.45	0.11	0.19	-0.27 to 0.49	0.01	0.08	-0.14 to 0.16	0.05	0.27	-0.48 to 0.58
DOSPERT												
Intercept	-1.31	0.81	-2.9 to 0.28	-0.13	0.17	-0.46 to 0.21	-0.09	0.08	-0.25 to 0.06	-1.53	0.33	-2.17 to -0.89
Interaction	0.00	0.00	0.00 to 0.00	0.00	0.00	0.00 to 0.00	0.00	0.00	0.00 to 0.00	0.00	0.00	0.00 to 0.00
DOSPERT	-0.05	0.02	-0.09 to 0.00	-0.01	0.00	-0.01 to 0.00	0.00	0.00	-0.01 to 0.00	-0.02	0.01	-0.04 to -0.01
MSBS Change	-0.01	0.01	-0.03 to 0.02	0.00	0.00	0.00 to 0.01	0.00	0.00	0.00 to 0.00	0.00	0.00	-0.01 to 0.01

Age	-0.03	0.04	-0.10 to 0.05	-0.01	0.01	-0.02 to 0.01	0.00	0.00	-0.01 to 0.00	0.00	0.01	-0.02 to 0.02
Male	-2.23	0.91	-4.03 to -0.44	-0.50	0.20	-0.90 to -0.10	0.02	0.08	-0.13 to 0.18	0.37	0.27	-0.16 to 0.91
Non–white	-0.71	1.81	-4.26 to 2.85	-0.50	0.40	-1.29 to 0.29	-0.02	0.16	-0.34 to 0.30	-1.24	0.56	-2.34 to -0.14
SES	-0.20	0.52	-1.21 to 0.82	0.03	0.11	-0.19 to 0.25	-0.01	0.04	-0.1 to 0.07	-0.22	0.17	-0.55 to 0.11
No. Symp.	0.33	0.75	-1.14 to 1.80	0.04	0.16	-0.28 to 0.35	0.09	0.06	-0.04 to 0.21	-0.25	0.22	-0.68 to 0.19
Iso. w. Child.	0.75	0.87	-0.96 to 2.46	0.13	0.19	-0.25 to 0.50	0.00	0.08	-0.14 to 0.15	0.01	0.27	-0.52 to 0.53

Note. Models fit using imputed data (m = 40). 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; DOSPERT = Domain–Specific Risk–taking Scale; MSBS = Multidimensional State Boredom Scale.

[a] model fit using the Broyden–Fletcher–Goldfarb–Shanno algorithm to allow convergence.

Study 2

Daily drinking diary

Please record what you drank last week. For each day, state the type and amount of beverage consumed. For each day, state the time the drinking session began and the time it finished. If you had two drinking sessions in one day, state the amount consumed and the length of each session.

Day	Beverage	% ABV	Total amount drunk	Start time	Finish time
Monday					
Tuesday					
Wednesday					
Thursday					
Friday					
Saturday					
Sunday					

Personal feedback

Dear Participant,

Thank you for completing the survey this week! Please follow the link below to complete this week's survey.

Based on the data you provided, you have consumed [*insert number of units*] standard UK units of alcohol this week. This is equivalent to [*insert number of grams*] g of pure alcohol.

According to statistics, someone in [*insert country of residence*] aged between [*insert lower age*] – [*insert upper age*] will drink 21.3g of alcohol per day in a typical week, which means that you have drunk [*insert number of grams of alcohol*] [*insert more or less*] than average.

You are also drinking [*insert more or less*] than the recommended amount someone should consume per week. The UK Government and NHS suggest that you should drink no more than 14 units per week (approximately 6 moderately sized drinks per week).

Long–term excessive drinking is associated with a range of health problems (including cancers of the mouth, throat and breast). Therefore, if you regularly drink as much as 14 units per week, it is best to spread your drinking evenly over 3 or more days. If you have one or two heavy drinking episodes per week, you increase your risk of long–term illness or injury.

For more information or support when cutting down, please see:

https://brainandbehaviourlab.jimdofree.com/research/drinking-advice/

Follow this link to take the survey: [*insert link*]

Follow this link to opt out of future emails: [*insert link*]

Thank you again. We will be in touch next week with another survey.

Best wishes,

James Clay (on behalf of the study team)

General feedback

Dear Participant,

Thank you for completing the survey this week! Please follow the link below to complete this week's survey.

Follow this link to take the survey: [*insert link*]

Follow this link to opt out of future emails: [*insert link*]

Thank you again. We will be in touch next week with another survey.

Best wishes,

James Clay (on behalf of the study team)

Missing data

Table C19. Percentage of missing data by variable.

Variable	% Missing
SES Index	33.33%
Weekly Units	19.17%
Weekly Drinking Days	19.17%
Weekly Heavy Days	19.17%
APQ	18.10%
Isolated With	18.10%
Timepoints Experienced COVID-19 Symptoms	18.10%
MSBS	18.10%
SOS–S	18.10%

Note. SES = socioeconomic status; 1 unit = 8g pure ethanol; 1 heavy day = consuming > 8 units per day for men or > 6 units per day for women; APQ = Alcohol Problems Questionnaire; MSBS = Multidimensional State Boredom Scale; SOS-S = Short Stress Overload Scale; symptoms included: (1) a high temperature, (2) a new, continuous cough, (3) a continuous headache, (4) a loss of taste and/or smell, (5) muscle aches, (6) a sore throat.

Appendix D: Chapter 5 Supplementary Materials

Favourable Ethical Approval

Science and Health Faculty Ethics Committee
Science and Health Faculty Office
University of Portsmouth
St Michael's Building
White Swan Road
PORTSMOUTH
PO1 2DT

023 9284 3379
ethics-sci@port.ac.uk

4 March 2021

Dr Matthew Parker
School of Pharmacy and Biomedical Sciences
University of Portsmouth

matthew.parker@port.ac.uk

FAVOURABLE ETHICAL OPINION – NOTIFICATION OF SUBSTANTIAL AMENDMENT

Study Title: The effect of cumulative life stress, emotional dysregulation, and inhibitory control on lifetime alcohol use: A moderated mediation analysis.

Reference Number: SHFEC 2021-022A

Date Submitted: 4 March 2021

Thank you for submitting your proposal amendment to the Science and Health Faculty Ethics Committee (SHFEC) for ethical review in accordance with current procedures.

I am pleased to inform you that SHFEC was content to grant a favourable ethical opinion of this proposal amendment on the basis described in the submitted documents listed at Annex A, and subject to standard general conditions (*See Annex B)*, and the following specific minor condition(s).

Conditions [1]

With respect to the proposed amendments, please ensure you address the following changes.

A. Please ensure that there is only one method of payment/honorarium/incentive for all participants. The two methods (£5 vs the chance to win £50 amazon voucher) are not equal. Please also ensure your advertisements reflect your now revised study details. Also, please remove the wording that the payment/honorarium/incentive is not a benefit of the research.

With respect to the conditions, please provide further details as the information presented is not clear.

B. Please provide this list of resources either in your PIS or debrief letter. Please ensure the resources are accessible and available.

[1] The favourable opinion given is dependent upon the study adhering to the conditions stated, which are based on the application document(s) submitted. It is appreciated that Chief Investigators may wish to challenge conditions or propose amendments to these. In that case, please consider the favourable opinion *suspended*, and simply make your case for amending or discarding conditions in writing as you would an application resubmission following ethical review.

C. Please ensure your advertisements are consistent and reflect your now revised study components. Please note the earlier details concerning using only one method of payment/honorarium/incentive for all participants.

If you would find it helpful to discuss any of the matters raised above or seek further clarification from a member of the Committee, you are welcome to contact ethics-sci@port.ac.uk who will circulate your queries to SHFEC

Please note that the favourable opinion of SHFEC does not grant permission or approval to undertake the research. Management permission or approval must be obtained from any host organisation, including the University of Portsmouth or supervisor, prior to the start of the study.

Wishing you every success in your research

Dr Paul Gorczynski
Vice Chair, Science and Health Faculty Ethics Committee

Annexes

A - Documents reviewed
B - After ethical review - Guidance for researchers

Information:

James Clay - Co-Investigator
Dr Lorenzo Stafford - Co-Investigator
Holly Seaton-Wood/Ruth Wills - Faculty Administrator

Statement of compliance

SHFEC is constituted in accordance with the Governance Arrangements set out by the University of Portsmouth

After Ethical Review

If unfamiliar, please consult the advice After Ethical Review (Annex B), which gives detailed guidance on reporting requirements for studies with a favourable opinion, including, notifying substantial amendments, notification of serious breaches of the protocol, progress reports and notifying SHFEC of the end of the study.

Power Analysis

The following procedure is partially described in Schoemann et al. (2017) and Lakens (2022). We implemented a simulation-based sensitivity (Monte Carlo) power analysis using an online Shiny App (https://schoemanna.shinyapps.io/mc_power_med/). The effect sizes entered into the power analysis were informed by taking the mean of correlation coefficients reported in previous literature. We assumed correlations of .283 for the relationship between X and M (Abravanel & Sinha, 2015; Burns et al., 2010); 449 for the relationship between M and Y (Aurora & Klanecky, 2016; Dragan, 2015; Khosravani et al., 2017; Mandavia et al., 2016; Petit et al., 2015); and .224 between X and Y (Dawson et al., 2005; Fox et al., 2010; Mandavia et al., 2016). 110 participants were required to achieve sufficient statistical power, $(1 - \beta) = 80\%$, to test our primary hypothesis (mediation effect). As we also planned to address our secondary hypotheses (moderation effects), we collected data from as many participants as our financial resources would allow (Lakens, 2022).

Stop Signal Task Descriptive Statistics

We report several task-related descriptive statistics in Table D1, following *"A consensus guide to capturing the ability to inhibit actions and impulsive behaviors in the stop-signal task"* (Verbruggen et al., 2019). P(Response|Signal) should ≈ 0.50 (Band et al., 2003) and, at the very least, individual SSRTs should *not* be estimated when P(Response|Signal) is lower than 0.25 or higher than 0.75 (Congdon et al., 2012). Applying this rule in the present study resulted in 58.11% of the data being excluded, suggesting that the SST data were unreliable for the majority of the participants. We did not include this measure in our analyses due to the questionable reliability of the data in the present study and the reduction in sample size and statistical power following the exclusion of the unreliable data.

Table D1. Stop Signal Task descriptive statistics.

Variable	**Total (SD)**	**Female (SD)**	**Male (SD)**
P(Response|Signal)	0.30 (0.23)	0.32 (0.24)	0.29 (0.22)
P(No Response|Go Trial)	0.07 (0.06)	0.07 (0.06)	0.07 (0.06)
P(Choice Error|Go Trial)	0.01 (0.01)	0.01 (0.02)	0.01 (0.01)
M RT on Go Trials	674.39 (82.39)	667.19 (81.99)	681.79 (82.43)
Intra-subject *SD* for *M* RT on Go Trials	123.27 (30.78)	123.57 (28.07)	122.96 (33.44)
Mean Stop Signal Delay	138.13 (74.99)	144.57 (78.02)	131.51 (71.41)
Mean RT for Unsuccessful Stop Trials	536.42 (64.17)	533.79 (62.90)	539.21 (65.61)

Missing Data

Table D2. Percentage of missing data by variable.

Variable	% Missing
Relationship Status	3.38%
Employment	3.38%
Education	1.35%
Household Income	1.01%
Age	1.01%
STRAIN	0.68%
BART	0.34%
1 - AUC	1.01%

Note. STRAIN = The Stress and Adversity Inventory for Adults; DERS-SF = Difficulties in Emotional Regulation Scale Short Form; BART = average number of space bar presses for unburst balloons during the Balloon Analogue Risk Task; 1 – AUC = 1 minus the area under the curve scores (greater scores reflect greater delay discounting) for the Titrating Alternatives Delay Discounting Task.

Exploratory Factor Analysis

We randomly split our dataset in half as per our preregistration as we had planned to run EFA on one half of the data and confirmatory factor analysis (CFA) on the other half. However, as the EFA analyses did not achieve simple structure (see below), CFA analysis was not performed.

Socioeconomic status index

An EFA was used to try to create an index of socioeconomic status using the *psych* package (version 2.1.3) in R version (4.2.1). The variables entered into this EFA included:

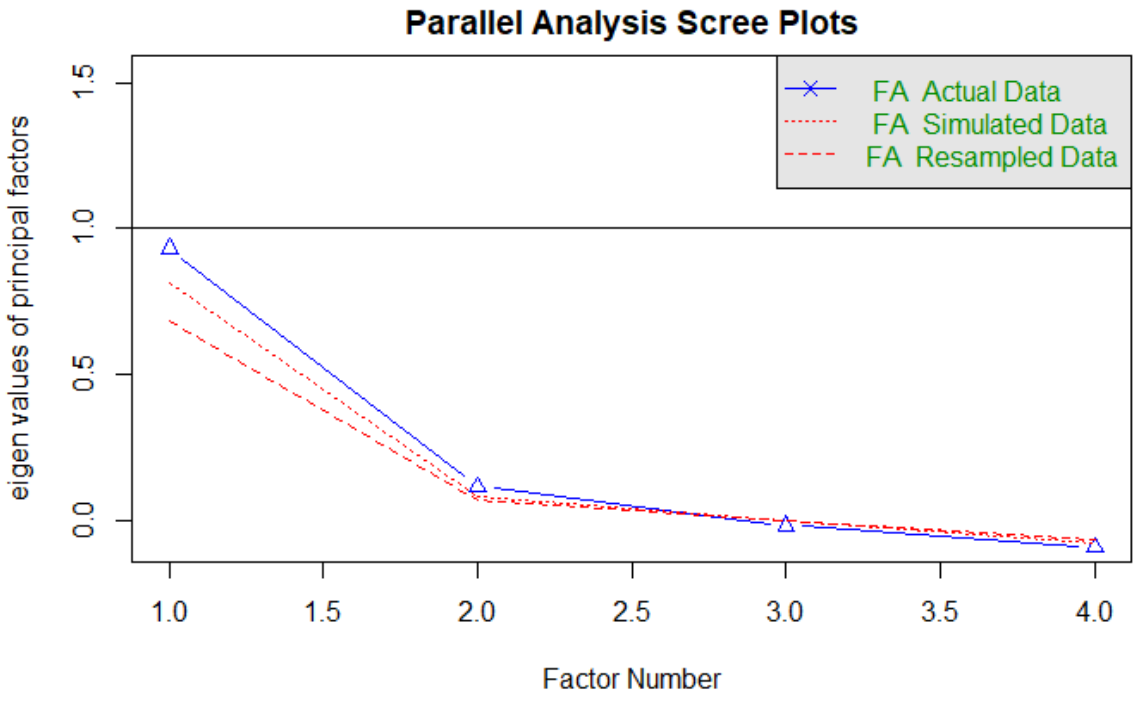
1. Highest level of education completed (no formal qualifications, secondary education [GCSE], A-levels, technical/community college, undergraduate degree [BA/BSc/other], graduate degree [MA/MSc/MPhil/other], doctoral degree or higher);
2. Employment status (unemployed, student, employed);
3. Household income (< 10,000, £10,000 - £15,999, £16,000 - £19,999, £20,000 - £29,999, £30,000 - £39,999, £40,000 - £49,999, £50,000 - £59,999, £60,000 - £69,999, £70,000 - £79,999, £80,000 - £89,999, £90,000 - £99,999, £100,000 - £149,999, > £150,000);
4. and subjective social status (1 to 10).

The EFA analysis was conducted using guidelines outlined in Preacher and MacCallum (2003). As that dataset that underwent EFA included a mix of continuous and polytomous variables, the *mixed.cor* function from the *psych* package was used to calculate the correlation matrix that was subjected to EFA (see Table D3). Bartlett's test indicated correlation adequacy, χ^2 (6) = 42.75, p < .001 and the determinant ($|R|$ = .733) was well above the specified cut off of .00001, suggesting that the data were not multicollinear. However, the KMO test indicated sampling inadequacy, *MSA* = 0.53. Similarly, individual KMO values were all 'unacceptable' (< 0.60) and three methods commonly used to determine how many factors to retain (parallel analysis, scree plot examination, and the K1 criterion) suggested that the data were not suitable for factor analysis (see Figure D1).

Table D3. Inter-correlations of socioeconomic status variables.

	1	2	3
1. Education	-		
2. Employment	.23	-	
3. Household Income	.12	.19	-
4. Subjective Status	.22	.05	.40

Figure D1. Parallel analysis scree plots.



Impulsivity index

The same EFA procedure reported above was followed to try to create a single index of impulsivity. The variables entered into this EFA included:

1. BART (continuous);
2. 1 – AUC (continuous);
3. SSRT (continuous);
4. SUPPS-P Negative Urgency (continuous);
5. SUPPS-P Perseverance (continuous);
6. SUPPS-P Premeditation (continuous);
7. SUPPS-P Sensation Seeking (continuous);
8. and SUPPS-P Positive Urgency (continuous).

The correlation matrix that was subjected to EFA is reported in Table D4. Bartlett's test indicated correlation adequacy, $\chi 2\ (28) = 198.74$, $p < .001$, the KMO test indicated sampling adequacy, MSA = 0.59, and the determinant ($|R| = .225$) was well above the specified cut off of .00001, suggesting that the data were not multicollinear. However, individual KMO values were 'unacceptable' (< .60) for BART, Perseverance, Sensation Seeking and Positive Urgency.

Parallel analysis and scree plot inspection (see Figure D2) suggested that a three-factor model was appropriate. Meanwhile the K1 criterion suggested that one factor solution. Therefore, we tested a three-factor solution. Maximum likelihood was used with direct oblimin rotation. The factor loadings are presented in Table D5. As simple structure was not achieved, we concluded, that like in prior research, impulsivity should be considered as a distinct set of separate constructs (Strickland & Johnson, 2020). Therefore, we estimated separate models, which aimed to test our hypotheses, for each construct.

Table D4. Inter-correlations of impulsivity variables.

	1	2	3	4	5	6	7
1. BART	-						
2. 1 - AUC	-.15	-					
3. SSRT	.04	-.02	-				
4. SUPPS-P Negative Urgency	-.06	.15	-.07	-			
5. SUPPS-P Perseverance	.10	-.09	.06	.22	-		
6. SUPPS-P Premeditation	.04	.06	.00	.45	.53	-	
7. SUPPS-P Sensation Seeking	.15	.12	-.13	.17	-.06	.12	-
8. SUPPS-P Positive Urgency	-.01	.14	-.11	.63	.07	.33	.44

Figure D2. Parallel analysis scree plots.

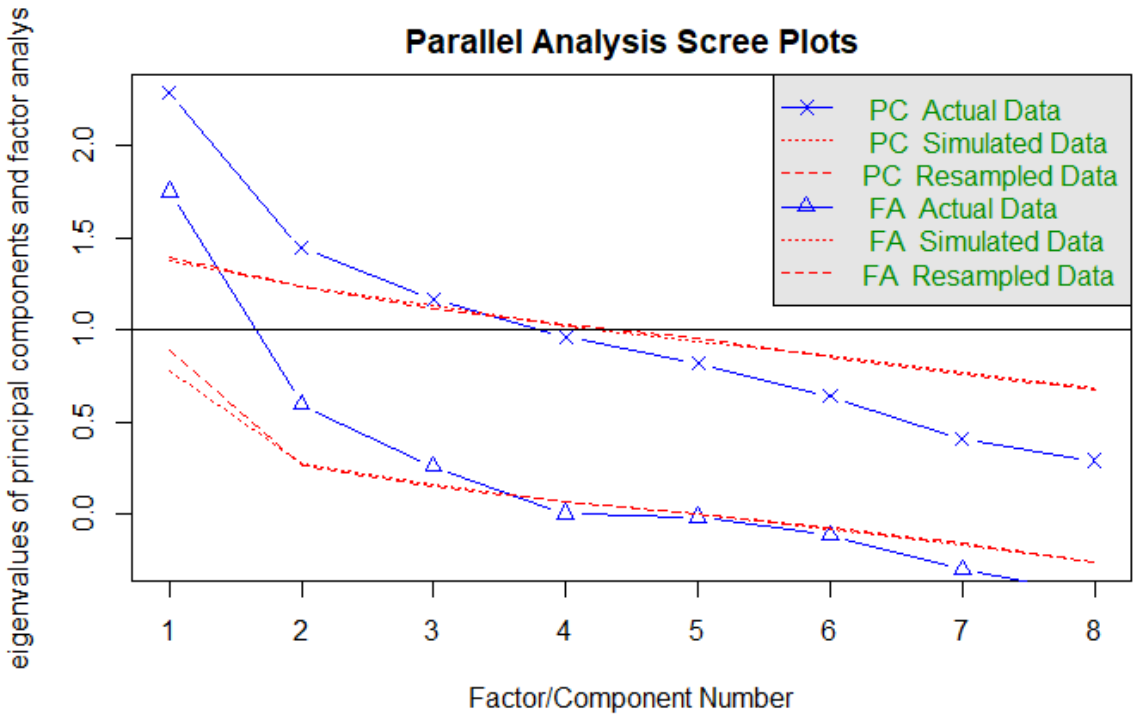


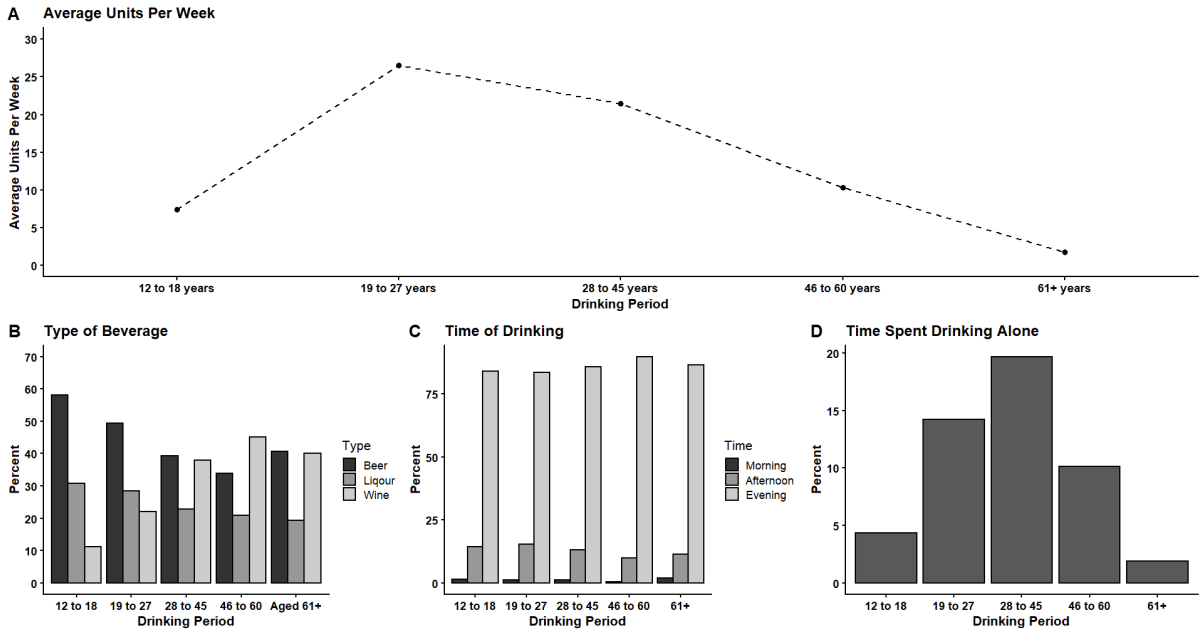
Table D5. Three-factor model loadings.

	1	**2**	**3**
BART	-.16	.16	.21
1 - AUC	.20	-.14	.05
SSRT	-.09	.08	-.10
SUPPS-P Negative Urgency	**.90**	.03	-.09
SUPPS-P Perseverance	-.03	**.94**	.00
SUPPS-P Premeditation	.35	**.49**	.04
SUPPS-P Sensation Seeking	.00	.00	**1.00**
SUPPS-P Positive Urgency	**.69**	-.06	.24

Note. Boldface font indicates factor loadings > .04.

Lifetime Alcohol Use Descriptive Statistics

Figure D3. Descriptive statistics for lifetime alcohol use in terms of average units of alcohol consumed per week (A), the type of beverage consumed (B), the time of drinking (C), the time of drinking, and drinking context (D). 1 UK alcohol unit = 8g of pure ethanol.



Conditional Process Analysis Results

Table D6. Summary of the mediation analysis examining whether negative urgency moderates the mediated relation between cumulative lifetime stressor exposure, emotional dysregulation and lifetime alcohol use (N = 279).

		Consequent								
		M **(DERS-SF)**					***Y*** **(Alcohol Use)**			
Antecedent		**B**	**SE**	**LL**	**UL**		**B**	**SE**	**LL**	**UL**
Constant	i_M	26.00	5.88	14.56	37.52	i_Y	72.88	35.41	10.16	146.74
X (STRAIN)	*a*	**-0.15**	**0.07**	**-0.29**	**-0.02**	*c'*	0.23	0.25	-0.28	0.70
M (DERS-SF)		-	-	-	-	b_1	-1.08	0.62	-2.29	0.12
W (NEGURG)		**1.48**	**0.37**	**0.76**	**2.20**	b_2	-3.70	2.65	-9.01	1.47
W x *X*		**0.02**	**0.01**	**0.01**	**0.03**	b_3	-0.01	0.02	-0.06	0.03
W x *M*		-	-	-	-	b_4	**0.13**	**0.07**	**0.005**	**0.26**
Age		**-0.17**	**0.05**	**-0.28**	**-0.06**		-0.06	0.16	-0.39	0.26
Sex = Male		1.13	1.15	-1.20	3.38		7.72	3.97	-0.16	15.50
Education										
GCSE & below		Ref.					Ref.			
A-levels & equivalent		4.26	2.39	-0.30	9.12		-13.73	9.91	-34.39	4.89
Undergraduate & higher		2.85	2.11	-1.17	7.07		-15.94	9.58	-35.90	1.67
Employment										
Unemployed		Ref.					Ref.			
Student		5.73	3.03	-0.35	11.58		-6.69	15.89	-40.31	20.23
Employed		4.23	2.47	-0.72	8.99		-11.58	13.92	-41.47	10.33
Household Income										
Low		Ref.					Ref.			
Medium		0.47	2.04	-3.48	4.54		0.29	5.01	-9.07	10.59
High		-1.45	1.41	-4.16	1.36		-0.10	5.09	-9.62	10.52

Subjective Social Status								
Low	Ref.				Ref.			
Medium	-0.13	1.74	-3.59	3.25	3.10	6.69	-8.80	17.38
High	-1.19	1.51	-4.18	1.74	-0.42	4.56	-9.16	8.60
		$R^2 = 0.51$				$R^2 = 0.14$		
		$F(13, 265) = 21.53$, $p < .001$				$F(15, 263) = 2.90$, $p < .001$		

Note. Models were adjusted for age, sex, highest level of education achieved, employment status, and household income. LL and UL represent the lower and upper limit of the bootstrapped 95% CI (10,000 bootstraps), respectively. STRAIN = Stress and Adversity Inventory for Adults Stressor Severity Index; DERS-SF = Difficulties in Emotional Regulation Scale Short Form; NEGURG = Negative Urgency subscale of the Shortened Urgency, Premeditation, Perseverance, Sensation Seeking, Positive Urgency, Impulsive Behaviour Scale; 1 unit = 8g pure ethanol. Significant effects ($p < .05$) are in boldface.

Table D7. Summary of the mediation analysis examining whether lack of perseverance moderates the mediated relation between cumulative lifetime stressor exposure, emotional dysregulation and lifetime alcohol use (*N* = 279).

		Consequent								
		***M* (DERS-SF)**					***Y* (Alcohol Use)**			
Antecedent		**B**	**SE**	**LL**	**UL**		**B**	**SE**	**LL**	**UL**
Constant	i_M	**34.66**	**7.53**	**19.86**	**49.30**	i_Y	**114.43**	**40.08**	**40.98**	**195.38**
X (STRAIN)	*a*	0.12	0.11	-0.08	0.34	*c'*	-0.21	0.29	-0.83	0.33
M (DERS-SF)		-	-	-	-	b_1	-1.02	0.62	-2.15	0.27
W (PERSEV)		1.23	0.67	-0.05	2.57	b_2	**-11.89**	**4.00**	**-19.62**	**-3.84**
W x *X*		0.003	0.01	-0.03	0.03	b_3	0.04	0.04	-0.03	0.12
W x *M*		-	-	-	-	b_4	**0.21**	**0.09**	**0.02**	**0.37**
Age		**-0.34**	**0.06**	**-0.45**	**-0.22**		-0.01	0.17	-0.34	0.30
Sex = Male		-0.56	1.43	-3.42	2.26		6.70	3.87	-0.90	14.30
Education										
GCSE & below		Ref.					Ref.			
A-levels & equivalent		4.24	3.10	-1.87	10.25		-16.07	9.62	-36.00	1.61
Undergraduate & higher		2.21	2.87	-3.44	7.87		-16.70	9.41	-36.47	0.30
Employment										
Unemployed		Ref.					Ref.			
Student		6.37	3.72	-1.21	13.52		-5.67	14.93	-37.01	20.37
Employed		5.57	2.95	-0.52	11.08		-12.15	13.83	-41.51	11.17
Household Income										
Low		Ref.					Ref.			
Medium		-1.44	2.21	-5.55	3.08		-1.23	4.82	-10.12	8.85
High		-2.03	1.80	-5.46	1.68		-0.07	4.97	-9.39	10.14

Subjective Social Status								
Low	Ref.				Ref.			
Medium	-0.52	2.07	-4.69	3.42	2.50	6.62	-9.16	16.85
High	-1.00	1.89	-4.77	2.71	-0.47	4.33	-8.76	8.20
		$R^2 = 0.27$				$R^2 = 0.16$		
		$F(13, 265) = 7.54$, $p < .001$				$F(15, 263) = 3.30$, $p < .001$		

Note. Models were adjusted for age, sex, highest level of education achieved, employment status, and household income. LL and UL represent the lower and upper limit of the bootstrapped 95% CI (10,000 bootstraps), respectively. STRAIN = Stress and Adversity Inventory for Adults Stressor Severity Index; DERS-SF = Difficulties in Emotional Regulation Scale Short Form; PERSEV = Perseverance subscale of the Shortened Urgency, Premeditation, Perseverance, Sensation Seeking, Positive Urgency, Impulsive Behaviour Scale; 1 unit = 8g pure ethanol. Significant effects ($p < .05$) are in boldface.

Table D8. Summary of the mediation analysis examining whether lack of premeditation moderates the mediated relation between cumulative lifetime stressor exposure, emotional dysregulation and lifetime alcohol use (N = 279).

		Consequent							
		***M* (DERS-SF)**					***Y* (Alcohol Use)**		
Antecedent		**B**	**SE**	**LL**	**UL**		**B**	**SE**	**LL**	**UL**
Constant	i_M	**31.43**	**6.46**	**19.30**	**44.48**	i_Y	**69.05**	**34.66**	**5.96**	**140.47**
X (STRAIN)	*a*	0.003	0.08	-0.14	0.16	*c'*	0.37	0.27	-0.24	0.84
M (DERS-SF)		-	-	-	-	b_1	-0.79	0.60	-1.85	0.53
W (PREMED)		**1.24**	**0.55**	**0.16**	**2.32**	b_2	-5.38	3.67	-12.29	2.20
W x *X*		0.01	0.01	-0.01	0.03	b_3	-0.04	0.04	-0.10	0.05
W x *M*		-	-	-	-	b_4	0.17	0.09	-0.03	0.34
Age		**-0.29**	**0.06**	**-0.41**	**-0.17**		-0.06	0.17	-0.40	0.25
Sex = Male		0.17	1.38	-2.53	2.93		6.73	3.96	-1.25	14.43
Education										
GCSE & below		Ref.					Ref.			
A-levels & equivalent		4.48	3.07	-1.63	10.48		-16.24	10.13	-37.54	2.34
Undergraduate & higher		3.21	2.77	-2.31	8.58		-18.22	9.86	-39.06	-0.09
Employment										
Unemployed		Ref.					Ref.			
Student		**8.67**	**3.33**	**1.82**	**14.88**		-5.24	16.08	-39.65	22.28
Employed		**7.74**	**2.56**	**2.51**	**12.64**		-11.80	14.69	-43.97	11.88
Household Income										
Low		Ref.					Ref.			
Medium		-1.86	2.35	-6.30	2.91		-0.71	5.20	-10.54	9.84
High		-2.49	1.70	-5.71	0.96		0.24	5.17	-9.47	10.88
Subjective Social Status										
Low		Ref.					Ref.			

Medium	-1.06	2.05	-5.15	2.86	3.17	6.69	-8.82	17.54
High	-1.05	1.80	-4.55	2.45	0.64	4.54	-8.06	9.74
		$R^2 = 0.32$				$R^2 = 0.13$		
		$F(13, 265) = 9.79$, $p < .001$				$F(15, 263) = 2.73$, $p < .001$		

Note. Models were adjusted for age, sex, highest level of education achieved, employment status, and household income. LL and UL represent the lower and upper limit of the bootstrapped 95% CI (10,000 bootstraps), respectively. STRAIN = Stress and Adversity Inventory for Adults Stressor Severity Index; DERS-SF = Difficulties in Emotional Regulation Scale Short Form; PREMED = Premeditation subscale of the Shortened Urgency, Premeditation, Perseverance, Sensation Seeking, Positive Urgency, Impulsive Behaviour Scale; 1 unit = 8g pure ethanol. Significant effects ($p < .05$) are in boldface.

Table D9. Summary of the mediation analysis examining whether sensation seeking moderates the mediated relation between cumulative lifetime stressor exposure, emotional dysregulation and lifetime alcohol use (N = 279).

		Consequent							
		***M* (DERS-SF)**					***Y* (Alcohol Use)**		
Antecedent		**B**	**SE**	**LL**	**UL**		**B**	**SE**	**LL**	**UL**
Constant	i_M	**42.83**	**7.50**	**27.78**	**57.37**	i_Y	5.59	30.99	-49.11	72.03
X (STRAIN)	*a*	0.11	0.10	-0.09	0.31	*c′*	0.49	0.26	-0.02	0.99
M (DERS-SF)		-	-	-	-	b_1	1.02	0.64	-0.35	2.16
W (SENSAT)		0.05	0.58	-1.03	1.23	b_2	3.30	2.54	-2.00	7.90
W x *X*		0.004	0.01	-0.02	0.02	b_3	-0.04	0.02	-0.08	0.01
W x *M*		-	-	-	-	b_4	-0.05	0.06	-0.16	0.07
Age		**-0.32**	**0.06**	**-0.44**	**-0.20**		-0.11	0.17	-0.46	0.21
Sex = Male		-0.76	1.55	-3.89	2.25		6.55	3.99	-1.73	14.16
Education										
GCSE & below		Ref.					Ref.			
A-levels & equivalent		4.85	3.28	-1.69	11.32		**-20.17**	**9.37**	**-39.18**	**-2.44**
Undergraduate & higher		2.89	3.03	-3.14	8.78		**-22.03**	**9.22**	**-40.97**	**-4.63**
Employment										
Unemployed		Ref.					Ref.			
Student		5.60	3.84	-2.16	12.99		-7.52	17.14	-45.23	19.29
Employed		5.16	3.06	-1.09	10.95		-14.54	15.67	-50.02	8.11
Household Income										
Low		Ref.					Ref.			
Medium		-1.24	2.27	-5.45	3.36		-2.74	5.15	-12.39	7.72
High		-1.63	1.84	-5.15	2.12		-1.25	5.35	-11.24	9.80
Subjective Social Status										
Low		Ref.					Ref.			

Medium	-0.99	2.19	-5.43	3.16	3.02	6.72	-9.03	17.25
High	-2.15	1.91	-5.94	1.63	0.63	4.68	-8.46	9.78
			$R^2 = 0.23$				$R^2 = 0.13$	
			$F(13, 265) = 6.11$, $p < .001$				$F(15, 263) = 2.54$, $p < .001$	

Note. Models were adjusted for age, sex, highest level of education achieved, employment status, and household income. LL and UL represent the lower and upper limit of the bootstrapped 95% CI (10,000 bootstraps), respectively. STRAIN = Stress and Adversity Inventory for Adults Stressor Severity Index; DERS-SF = Difficulties in Emotional Regulation Scale Short Form; SENSAT = Sensation Seeking subscale of the Shortened Urgency, Premeditation, Perseverance, Sensation Seeking, Positive Urgency, Impulsive Behaviour Scale; 1 unit = 8g pure ethanol. Significant effects ($p < .05$) are in boldface.

Table D10. Summary of the mediation analysis examining whether positive urgency moderates the mediated relation between cumulative lifetime stressor exposure, emotional dysregulation and lifetime alcohol use (N = 279).

		Consequent								
		***M* (DERS-SF)**					***Y* (Alcohol Use)**			
Antecedent		**B**	**SE**	**LL**	**UL**		**B**	**SE**	**LL**	**UL**
Constant	i_M	**29.21**	**6.49**	**16.79**	**42.27**	i_Y	26.48	23.14	-15.12	75.17
X (STRAIN)	*a*	0.08	0.07	-0.07	0.22	*c'*	**0.54**	**0.22**	**0.12**	**1.00**
M (DERS-SF)		-	-	-	-	b_1	0.14	0.48	-0.82	1.04
W (POSURG)		**1.67**	**0.52**	**0.66**	**2.72**	b_2	1.19	2.38	-3.62	5.77
W x *X*		0.004	0.01	-0.01	0.02	b_3	**-0.05**	**0.03**	**-0.10**	**-0.001**
W x *M*		-	-	-	-	b_4	0.04	0.05	-0.06	0.15
Age		**-0.23**	**0.06**	**-0.35**	**-0.12**		-0.05	0.17	-0.38	0.28
Sex = Male		-0.74	1.32	-3.34	1.84		6.75	3.97	-1.30	14.39
Education										
GCSE & below		Ref.					Ref.			
A-levels & equivalent		4.02	3.05	-1.91	10.03		**-17.86**	**9.51**	**-37.89**	**-0.24**
Undergraduate & higher		3.44	2.75	-1.98	8.87		**-19.19**	**9.32**	**-38.87**	**-2.06**
Employment										
Unemployed		Ref.					Ref.			
Student		4.68	3.76	-2.96	11.74		-9.50	17.90	-47.93	19.59
Employed		4.40	3.01	-1.91	9.94		-15.33	16.43	-51.50	9.22
Household Income										
Low		Ref.					Ref.			
Medium		-0.16	2.24	-4.43	4.37		-0.58	4.83	-9.79	9.16
High		-2.12	1.58	-5.12	1.11		-0.74	5.20	-10.46	9.92
Subjective Social Status										
Low		Ref.					Ref.			

Medium	-1.12	2.07	-5.25	2.86	2.69	6.64	-9.19	16.68
High	-1.73	1.81	-5.25	1.89	-0.38	4.66	-9.34	8.78
			$R^2 = 0.36$			$R^2 = 0.12$		
			$F(13, 265) = 11.29$, $p < .001$			$F(15, 263) = 2.40$, $p < .001$		

Note. Models were adjusted for age, sex, highest level of education achieved, employment status, and household income. LL and UL represent the lower and upper limit of the bootstrapped 95% CI (10,000 bootstraps), respectively. STRAIN = Stress and Adversity Inventory for Adults Stressor Severity Index; DERS-SF = Difficulties in Emotional Regulation Scale Short Form; POSURG = Positive Urgency subscale of the Shortened Urgency, Premeditation, Perseverance, Sensation Seeking, Positive Urgency, Impulsive Behaviour Scale; 1 unit = 8g pure ethanol. Significant effects ($p < .05$) are in boldface.

Table D11. Summary of the mediation analysis examining whether risk-taking (BART scores) moderates the mediated relation between cumulative lifetime stressor exposure, emotional dysregulation and lifetime alcohol use (N = 279).

		Consequent							
		***M* (DERS-SF)**					***Y* (Alcohol Use)**		
Antecedent		**B**	**SE**	**LL**	**UL**		**B**	**SE**	**LL**	**UL**
Constant	i_M	**47.63**	**6.61**	**34.79**	**60.88**	i_Y	30.07	25.63	-16.26	82.75
X (STRAIN)	*a*	0.12	0.07	-0.03	0.26	*c'*	0.07	0.21	-0.36	0.46
M (DERS-SF)		-	-	-	-	b_1	0.57	0.39	-0.25	1.30
W (BART)		-0.10	0.11	-0.33	0.11	b_2	0.07	0.55	-1.09	1.08
W x *X*		0.001	0.002	-0.003	0.01	b_3	0.002	0.01	-0.01	0.02
W x *M*		-	-	-	-	b_4	-0.003	0.01	-0.02	0.02
Age		**-0.33**	**0.06**	**-0.44**	**-0.21**		-0.06	0.17	-0.40	0.27
Sex = Male		-0.73	1.46	-3.62	2.14		6.93	3.95	-0.79	14.58
Education										
GCSE & below		Ref.					Ref.			
A-levels & equivalent		3.66	3.33	-2.84	10.19		-17.53	9.80	-37.77	0.73
Undergraduate & higher		1.87	3.12	-4.37	7.97		**-19.16**	**9.42**	**-39.01**	**-1.93**
Employment										
Unemployed		Ref.					Ref.			
Student		5.83	3.83	-2.05	13.06		-5.96	17.74	-43.93	23.58
Employed		4.89	2.98	-1.29	10.38		-12.12	15.99	-47.38	12.02
Household Income										
Low		Ref.					Ref.			
Medium		-1.01	2.17	-5.29	3.25		-2.22	4.94	-11.78	7.84
High		-1.23	1.81	-4.75	2.37		-0.08	5.04	-9.25	10.60
Subjective Social Status										
Low		Ref.					Ref.			

Medium	-1.31	2.14	-5.63	2.85	2.58	6.61	-9.72	16.43
High	-2.32	1.94	-6.02	1.44	0.10	4.80	-9.26	9.71
		$R^2 = 0.23$				$R^2 = 0.11$		
		$F(13, 264) = 6.11$, $p < .001$				$F(15, 262) = 2.08$, $p = 0.011$		

Note. Models were adjusted for age, sex, highest level of education achieved, employment status, and household income. LL and UL represent the lower and upper limit of the bootstrapped 95% CI (10,000 bootstraps), respectively. STRAIN = Stress and Adversity Inventory for Adults Stressor Severity Index; DERS-SF = Difficulties in Emotional Regulation Scale Short Form; BART = average number of space bar presses for unburst balloons during the Balloon Analogue Risk Task; 1 unit = 8g pure ethanol. Significant effects ($p < .05$) are in boldface.

Table D12. Summary of the mediation analysis examining whether delay discounting (1 - AUC) moderates the mediated relation between cumulative lifetime stressor exposure, emotional dysregulation and lifetime alcohol use (*N* = 279).

		Consequent							
		***M* (DERS-SF)**					***Y* (Alcohol Use)**		
Antecedent		**B**	**SE**	**LL**	**UL**		**B**	**SE**	**LL**	**UL**
Constant	i_M	**49.42**	**11.09**	**26.83**	**70.46**	i_Y	17.30	56.74	-94.50	131.25
X (STRAIN)	*a*	-0.02	0.18	-0.35	0.36	*c'*	0.32	0.49	-0.59	1.37
M (DERS-SF)		-	-	-	-	b_1	0.25	1.13	-1.92	2.52
W (1 - AUC)		-6.29	12.23	-28.91	19.59	b_2	16.30	66.99	-112.67	150.60
W x *X*		0.20	0.227	-0.29	0.62	b_3	-0.25	0.61	-1.53	0.90
W x *M*		-	-	-	-	b_4	0.30	1.50	-2.74	3.17
Age		**-0.34**	**0.06**	**-0.45**	**-0.22**		-0.02	0.18	-0.38	0.32
Sex = Male		-0.75	1.47	-3.60	2.10		7.01	3.95	-0.78	14.75
Education										
GCSE & below		Ref.					Ref.			
A-levels & equivalent		3.96	3.41	-2.72	10.77		-17.04	9.93	-37.95	1.35
Undergraduate & higher		1.70	3.19	-4.62	7.93		**-18.57**	**9.56**	**-38.90**	**-0.89**
Employment										
Unemployed		Ref.					Ref.			
Student		6.10	3.90	-2.07	13.21		-6.02	18.62	-47.06	23.78
Employed		5.68	3.08	-0.83	11.24		-12.02	16.93	-50.33	12.93
Household Income										
Low		Ref.					Ref.			
Medium		-1.23	2.22	-5.59	3.25		-1.97	5.02	-11.40	8.36
High		-0.98	1.80	-4.40	2.59		0.32	5.09	-9.37	10.70
Subjective Social Status										
Low		Ref.					Ref.			

Medium	-0.91	2.24	-5.33	3.40	2.12	6.84	-10.42	16.47
High	-1.86	2.00	-5.85	1.98	-0.31	5.22	-10.38	9.89
			$R^2 = 0.22$			$R^2 = 0.11$		
			$F(13, 262) = 5.71$, $p < .001$			$F(15, 260) = 2.05$, $p = 0.013$		

Note. Models were adjusted for age, sex, highest level of education achieved, employment status, and household income. LL and UL represent the lower and upper limit of the bootstrapped 95% CI (10,000 bootstraps), respectively. STRAIN = Stress and Adversity Inventory for Adults Stressor Severity Index; DERS-SF = Difficulties in Emotional Regulation Scale Short Form; 1 – AUC = 1 minus the area under the curve scores (greater scores reflect greater delay discounting) for the Titrating Alternatives Delay Discounting Task; 1 unit = 8g pure ethanol. Significant effects ($p < .05$) are in boldface.

Appendix E: UPR16 Form

FORM UPR16

Research Ethics Review Checklist

Please include this completed form as an appendix to your thesis (see the Research Degrees Operational Handbook for more information

UNIVERSITY OF PORTSMOUTH

Postgraduate Research Student (PGRS) Information				**Student ID:**	735470
PGRS Name:	James Michael Clay				
Department:	Psychology	**First Supervisor:**	Lorenzo Stafford		
Start Date: (or progression date for Prof Doc students)	**September 2019**				
Study Mode and Route:	Part-time ☐	MPhil ☐	MD ☐		
	Full-time ☒	PhD ☒	Professional Doctorate ☐		

Title of Thesis:	The Impact of Impulse Control on Alcohol Use in the Context of Acute, Chronic, and Cumulative Lifetime Stress
Thesis Word Count: (excluding ancillary data)	40,224

If you are unsure about any of the following, please contact the local representative on your Faculty Ethics Committee for advice. Please note that it is your responsibility to follow the University's Ethics Policy and any relevant University, academic or professional guidelines in the conduct of your study

Although the Ethics Committee may have given your study a favourable opinion, the final responsibility for the ethical conduct of this work lies with the researcher(s).

UKRIO Finished Research Checklist:
(If you would like to know more about the checklist, please see your Faculty or Departmental Ethics Committee rep or see the online ve
the full checklist at: https://ukrio.org/publications/code-of-practice-for-research

a) Have all of your research and findings been reported accurately, honestly and within a reasonable time frame?	YES ☒ NO ☐
b) Have all contributions to knowledge been acknowledged?	YES ☒ NO ☐
c) Have you complied with all agreements relating to intellectual property, publication and authorship?	YES ☒ NO ☐
d) Has your research data been retained in a secure and accessible form and will it remain so for the required duration?	YES ☒ NO ☐
e) Does your research comply with all legal, ethical, and contractual requirements?	YES ☒ NO ☐

Candidate Statement:	
I have considered the ethical dimensions of the above named research project, and have successfully obtained the necessary ethical approval(s)	
Ethical review number(s) from Faculty Ethics Committee (or from NRES/SCREC):	SHFEC 2019-123A ETHICS-10155 SFEC 2020-030 SHFEC 2021-022A
If you have *not* submitted your work for ethical review, and/or you have answered 'No' to one or more of questions a) to e), please explain below why this is so:	
N/A	
Signed *(PGRS)*: 	**Date:** 20 June 2023

UPR16 – April 2018