The Effect of Stress and Locus of Control on Continued Substance Use during Opioid Substitution Treatment for Opioid Dependency.

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Masters of Research Thesis
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Statement of Authentication

The work presented in this thesis is, to the best of my knowledge and belief, original except as acknowledged in the text. I hereby declare that I have not submitted this material either in full or in part, or a degree at this or any other institution.

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L. C. Fitzgerald
Table of Contents

1.0 Abstract .................................................................................................................................. 9

2.0 Introduction .............................................................................................................................. 10
  2.1 Diagnostic Criteria of Opioid Use Disorder .............................................................................. 12
  2.2 Opioids .................................................................................................................................. 12
  2.3 Opioid Dependency Treatment ............................................................................................... 17
    2.3.1 Detoxification .................................................................................................................... 17
    2.3.2 Opioid Substitution Therapy .............................................................................................. 19
  2.4 Stress .................................................................................................................................... 21
    2.4.1 Psychological model ........................................................................................................... 25
    2.4.2 Neurological model ............................................................................................................ 27
  2.5 Individual Factors .................................................................................................................... 31
    2.5.1 Demographics .................................................................................................................... 31
    2.5.2 Locus of Control ................................................................................................................ 32

3.0 Aims and Hypotheses ................................................................................................................. 33

4.0 Method .................................................................................................................................... 34
  4.1 Participants ............................................................................................................................... 34
  4.2 Materials .................................................................................................................................. 35
    4.2.1 Demographic variables ....................................................................................................... 35
    4.2.2 Perceived Stress Scale (PSS) .............................................................................................. 35
    4.2.3 Locus of Control (LOC) ...................................................................................................... 36
    4.2.4 Inventory of Drug Taking Situations (IDTS) ..................................................................... 36
    4.2.5 Substance use ..................................................................................................................... 37
  4.3 Procedure ................................................................................................................................ 37

5.0 Results ..................................................................................................................................... 39
  5.1 Demographics .......................................................................................................................... 39
# LIST OF TABLES

<table>
<thead>
<tr>
<th>Table</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1.</td>
<td>Frequencies, percentages and adjusted standardised residuals for positive and negative toxicology results for male and female genders</td>
<td>40</td>
</tr>
<tr>
<td>1.2.</td>
<td>Frequencies, percentages and adjusted standardised residuals for positive and negative toxicology results for last opioid use timeframe</td>
<td>49</td>
</tr>
<tr>
<td>5.1.</td>
<td>Mean and Standard Deviation scores for all drug class users and nonusers for IDTS subscales</td>
<td>60</td>
</tr>
<tr>
<td>5.2.</td>
<td>Mean and Standard Deviation scores for all drug class users and nonusers for subscale profiles</td>
<td>60</td>
</tr>
<tr>
<td>5.3.</td>
<td>Mean and Standard Deviation scores for Opiate and Cannabinoid users and nonusers for IDTS subscales</td>
<td>62</td>
</tr>
<tr>
<td>5.4.</td>
<td>Mean and Standard Deviation scores for Opiate and Cannabinoid users and nonusers for subscale profiles</td>
<td>63</td>
</tr>
<tr>
<td>5.5.</td>
<td>Mean and Standard Deviation scores for Amphetamine and Benzodiazepine users and nonusers for IDTS subscales</td>
<td>65</td>
</tr>
<tr>
<td>5.6.</td>
<td>Mean and Standard Deviation scores for Amphetamine and Benzodiazepine users and non-users for subscale profiles</td>
<td>65</td>
</tr>
</tbody>
</table>
## LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figures</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1.</td>
<td>Gender and positive toxicology results</td>
<td>39</td>
</tr>
<tr>
<td>1.2.</td>
<td>Age group and positive toxicology result</td>
<td>40</td>
</tr>
<tr>
<td>1.3.</td>
<td>Combined age groups and positive toxicology result</td>
<td>42</td>
</tr>
<tr>
<td>1.4.</td>
<td>Employment status and positive toxicology results</td>
<td>43</td>
</tr>
<tr>
<td>1.5.</td>
<td>Education and positive toxicology results</td>
<td>44</td>
</tr>
<tr>
<td>1.6.</td>
<td>Martial status and positive toxicology result</td>
<td>45</td>
</tr>
<tr>
<td>1.7.</td>
<td>Timeframe of last opioid use and positive toxicology result</td>
<td>48</td>
</tr>
<tr>
<td>1.8.</td>
<td>Opiate lifetime use of all drug class use group and no-drug class use group</td>
<td>49</td>
</tr>
<tr>
<td>1.9.</td>
<td>Mean opiate lifetime use of opiate use group and no-opiate use</td>
<td>50</td>
</tr>
<tr>
<td>1.10.</td>
<td>Mean opiate lifetime use of benzodiazepine use group and no-benzodiazepine use group</td>
<td>50</td>
</tr>
<tr>
<td>1.11.</td>
<td>Mean opiate lifetime use of cannabinoid use group and no-cannabinoid use group</td>
<td>51</td>
</tr>
<tr>
<td>1.12.</td>
<td>Mean opiate lifetime use of amphetamine use group and no-amphetamine use group</td>
<td>51</td>
</tr>
<tr>
<td>2.1.</td>
<td>Participants with positive toxicology results</td>
<td>52</td>
</tr>
<tr>
<td>3.1.</td>
<td>Mean stress scores of all drug class user group and no-drug use group</td>
<td>53</td>
</tr>
<tr>
<td>3.2.</td>
<td>Mean stress scores of opiate use group and no-opiate use group</td>
<td>53</td>
</tr>
<tr>
<td>3.3.</td>
<td>Mean stress scores of benzodiazepine use group and no-benzodiazepine use group</td>
<td>54</td>
</tr>
<tr>
<td>3.4.</td>
<td>Mean stress scores of cannabinoid use group and no-cannabinoid use group</td>
<td>55</td>
</tr>
<tr>
<td>3.5.</td>
<td>Mean stress scores of benzodiazepine use group and no-benzodiazepine use group</td>
<td>55</td>
</tr>
<tr>
<td>4.1.</td>
<td>Mean LOC score for all drug class use and no-drug class use</td>
<td>56</td>
</tr>
<tr>
<td>4.2.</td>
<td>Mean LOC score for opiate use and no-opiate use</td>
<td>57</td>
</tr>
<tr>
<td>4.3.</td>
<td>Mean LOC score for amphetamine use and no-amphetamine use</td>
<td>57</td>
</tr>
</tbody>
</table>
4.4. Mean LOC score for cannabinoid use and no-cannabinoid use .......... 58
4.5. Mean LOC score for benzodiazepine use and no-benzodiazepine use ................................................................................. 59
5.1. Mean subscale profile scores for all drug classes, opiates, cannabinoids, amphetamine and benzodiazepine toxicology results .... 60
1.0 Abstract

In Australia, it is estimated that $55.4 billion is spent annually due to the direct and indirect costs of problematic drug use. Worldwide, it is estimated that 32 million individuals misuse opioids, with 16 million misusing heroin. To date, the Methadone Maintenance Treatment (MMT) programs are the most common treatment of opioid addiction at a global scale. Although MMT diminishes withdrawal symptoms, continued substance and polydrug use remains a salient issue within treatment settings. As relapse remains problematic, current literature implies that stress, Locus of Control (LOC) and exposure to high-risk situations are key contributors to relapse. Fifty opioid dependent participants, aged between 19-64 years old, who currently receive opioid substitution treatment at the Royal Prince Alfred Hospital (RPAH) in Camperdown, Sydney, were recruited for the present study. Independent t-tests, chi square and logistic regression were conducted on demographic, stress, LOC and high-risk context variants in the aim of establishing a predictor model for continued substance use. No significant results were found all analyses, however, data trends highlight that higher stress scores indicated greater use for all drug use classes, cannabinoids and benzodiazepines. Higher LOC score trends also suggested greater substance use for all drug classes. The results support literature identifying continued drug use within medically assisted treatment programs in an Australian sample. Future research should continue to identify individual variants that can predict continued substance use and adapt these findings in creating a holistic, multimodal approach to addiction treatment, as medically assisted interventions alone appear to be ineffective for many opioid dependent patients.

Keywords: opioids, opiates, heroin, addiction, dependency, stressors.
The Effect of Stress and Locus of Control on Continued Substance Use during Opioid Substitution Treatment.

2.0 Introduction

The key issue of Substance Use Disorders (SUDs) involves the compulsive drug seeking and use, even in the face of negative adverse physical, psychological and social consequences (Leshner, 1997; Goldstein & Volkow, 2011). Substance dependent individuals often display impulsive decision making choices that yield instant gratification and immediate gain, but with higher future losses (Zhang et al., 2011). In 2011, it was estimated that 32 million individuals globally misused opioids, 17 million used cocaine, 34 million used amphetamine-type stimulants and 181 million used cannabis (The Lancet, 2013). In 2016, the National Drug Strategy Household Survey identified that roughly 43% of the Australian population had used an illicit substance in their lifetime and 15.6% had done so in the past 12 months. In Australia, the top four most commonly used illicit substances within the past 12 months include: cannabis (10.4%), the non-medical use of prescription opioids (NMPR) (3.6%), cocaine (2.5%) and 400,000 used ecstasy (2.2%) (AIHW, 2016).

Globally, NMPR use is an increasingly tremendous burden on health care systems, resulting in an increase in emergency department visits, treatment admission, overdoses (Muhuri, Gfroerer & Davies, 2013) and serious morbidity from the high consumption of combination products containing paracetamol or ibuprofen (Degenhardt et al., 2016). The current epidemic of prescription opioid abuse is noted as an exclusive American disease, as Americans represent 5% of the population but consume 80% of the world’s opioid analgesics (Ling, 2017). In 2014, 1.9 million Americans aged over 12 were diagnosed with a SUD involving prescription opioids and 18,893 prescription medication related deaths were recorded (Eitan, Emery, Bates & Horraz, 2017). However, in Australia prescription opioids represent the most substantial increase in problematic drug use (Haber & Day, 2014) and it
was estimated in 2015 that 1% of the Australian population were dependent on opioids (Lintzeris, 2015).

Additionally, despite efforts to reduce opium production, misuse and dependency, the United Nations Office on Drugs and Crime (UNODC) estimated in 2007 that 11 million individuals were using heroin globally (UNODC, 2007). In 2000, it was estimated that there were approximately 74,000 heroin dependent individuals in Australia alone (Warner-Smith, Lynskey, Darke & Hall, 2000). Opiates account for more than 60% of treatment demand in Central and South East Asia (Brown & Lawrence, 2009) as it is estimated half of all global users live in Asia (Pecoraro et al., 2012). Heroin dependency also remains a prominent issue in Europe, with increasing heroin problems arising in surrounding countries such as Afghanistan, Russia, India and Africa (Brown & Lawrence, 2009).

Collectively, addiction to illicit and licit substances poses direct costs to the individual and indirect secondary costs to society through increased mortality, morbidity, marginalisation, criminal behaviour, medical events, social issues and loss of productivity (Koob, & Moal, 2006; Lobmaier, Gossop, Waal & Bramness, 2010). The Office of National Drug Control Policy (2001) estimated that illicit drug use in the United States alone costs $161 billion and hospitalisation rates for overdoses in the United States increased by 55% between 1999 to 2008, costing $737 million in 2008 (Martins et al., 2015). In Australia, reports from 2004-2005 estimated tangible (labour in the workforce, labour in the household, healthcare, crime and resources used in abusive consumption) and intangible (loss of life, pain and suffering) social costs due to drug use equalling $55.2 billion due to alcohol (27.3%), tobacco (56.2%) and illicit substances (14.6%) (Collins & Lapsey, 2008).
2.1 Diagnostic Criteria of Opioid Use Disorder

With regular and prolonged use to substances, the human body will experience neuroadaptation and physiological dependence characterised by tolerance and withdrawal (Lintzeris, 2015; Ling et al., 2011). Tolerance is defined as the need for increased amounts to achieve intoxication and occurs when brain cells containing opioid receptors become less responsive to opioid stimulation (Kosten, & George, 2002). Contrastingly, withdrawal is defined as symptoms of dysphoric mood, nausea, muscle aches, lacrimation, sweating, pupillary dilation, diarrhoea, fever, yawning and insomnia due to the cessation of substance use (American Psychiatric Association, 2013). Withdrawal is also identified when the drug is taken in order to maintain normal physiological functioning by suppressing withdrawal symptoms (West, 2006).

In regards to the diagnosis of an Opioid Use Disorder, the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-V) (American Psychiatric Association, 2013) details that tolerance and withdrawal symptoms represent two of the eleven diagnostic criteria. The complete diagnostic criteria for Opioid Use Disorder is shown in Appendix A.

2.2 Opioids

Opioid is a term that groups all drugs, natural and synthetic, with morphine-like actions. Currently they are the most powerful and effective drug class for pain relief known to humans (Koob & Moal, 2006). Opioid class medications include agonists (morphine, heroin, oxycodone, methadone, fentanyl and codeine), partial agonists that partially activate opiate receptors (buprenorphine) and antagonists that block opiate receptors (naloxone and naltrexone) (Lintzeris, 2015).

Opioids and heroin belong to the same opioid drug class as they bind with the mu opioid receptor (MOR) in the brain to produce a euphoric effect (National Institute of Drug
Abuse, 2015). The MOR is one of the three G protein-coupled receptors that the opioid system is comprised of: mu, delta and kappa. All three receptors are stimulated by endogenous opioid peptides and exogenously by alkaloid opiates (Contet, Kieffer & Befort, 2004). MOR are largely distributed along reward circuit pathways as well as alternate brain areas belonging to circuits of addiction including mesolimbic dopaminergic neurons, locus coeruleus, hypothalamus and some brainstem nuclei (Contet et al., 2004). These findings are critical in identifying areas of the brain responsible for the initiation and maintenance of dependence.

Dependency to opioids results in changes in the mesolimbic dopaminergic system affecting tolerance for euphoric effects and sensitisation for the incentive to take substances. Repeated exposure to opioids results in receptor desensitisation and down-regulation, meaning opioid tolerance increases, and the incentive to take drugs is sensitised for a much longer period (Lobmaier, Gossop, Waal & Bramness, 2010).

In Australia, eight opioids are subsidised by the Government’s Pharmaceutical Benefit Scheme (PBS): buprenorphine, codeine, fentanyl, hydromorphone, methadone, morphine, oxycodone and tramadol. Between 1992 and 2012, there was a 15-fold increase in government subsidised prescribed opioid use within Australia, with oxycodone being the main contributor for this increase. From this data, the most recent trend highlighted the escalating use of buprenorphine and fentanyl for the treatment of pain. Additionally, the increase in subsidised prescription opioids from 1992 to 2012 showcase a corresponding financial upsurge as evident by the Pharmaceutical Benefit Scheme (PBS) increasing government costing by 32-fold ($8.5 million to $271 million) (Blanch, Pearson & Haber, 2014; Degenhardt et al., 2015).

The family of opioid pain relievers include medications comprising of fentanyl, hydrocodone, oxycodone, propoxyphene, oxymorphone, hydromorphone, meperidine and
The International Narcotics Control Board (INCB) and World Health Organisation population data highlighted that Australia was the 5th top country of hydrocodone consumption (mg/capital), 13th for fentanyl and 2nd for oxycodone behind the United States for 2015 (University of Wisconsin, 2015).

The 2013 National Drug Strategy Household Survey indicated that 900,000 (4.7%) Australians aged 14 years and above, have used a pharmaceutical substance for non-medical purposes within the last 12 months. In comparison to 2011 statistics, the current figure entails a rise from 4.2% to 4.7% of NMPR in the Australian population (Australian Institute of Health and Welfare, 2016). Across 1998 to 2009, opioid-related hospitalisations in Australia increased from 605 to 1,464 cases (Blanch, Pearson & Haber, 2014). Between 2002 to 2011, accidental overdose deaths due to opioids have also increased from 32.3 to 49.5 per million persons between ages 15-49 (Australian Institute of Health and Welfare, 2016). More specifically, a study conducted in Victoria, Australia, showed that the detection of oxycodone in deaths increased from four cases in 2000 to 97 in 2009 (21-fold increase) (Rintoul et al., 2011). Globally, the trend of increasing deaths from NMPR and decreasing deaths from illicit substances is being noted widely throughout literature (Martins, Sampson, Cerda & Galea, 2015).

The use of prescription opioids for the treatment of physical pain, the extensive availability of prescription pain medication and the public misconception of the safety and addictive properties of these medications in comparison to illicit counterparts, are all intertwined key factors contributing to the rise in NMPR misuse, abuse and dependency Ling, Mooney & Hillhouse, 2011). Research has identified common motives for engaging in NMPR for alternate demographics. The most common motives for NMPR use for poly-illicit drug users were to get high, sleep or to relieve anxiety and stress. Amongst college students,
NMPR was associated with pain relief, getting high, experimental reasons and experiential awareness (Hartwell et al., 2012).

It is estimated that 3.27-11.5% of clients who are prescribed opioids for pain management will lead to an opioid substance use disorder (SUD) (Eitan, Emery, Bates & Horrax, 2017; Ling et al., 2011) and less than 25% of opioid dependent individuals receive addiction treatment (Alford et al., 2011). For individuals that do seek treatment, treatment admissions in the USA increased by 400% between 1998 and 2008 as the data escalated from 52,840 admissions in 2003 to 120,887 in 2008 (Ling et al., 2011).

Currently, there is a global concern that NMPR usage can also progress into using illicit substances. For example, within Australia, 39% of NMPR users have reported using illicit substances (AIHW, 2016). Further, research conducted in the United States has noted an increase of 82.6% heroin users between 2007 to 2013 and 6.2-fold increase in heroin deaths from 2002 to 2015 (National Institute of Drug Abuse, 2017). This increase is noted to be influenced by the initiation of heroin amongst NMPR users (Votaw et al., 2016) as four in five heroin users initially misused prescription opioids (Eitan et al., 2017). Further, 80% of new heroin users have previously misused prescription painkillers (Gostin, Hodge & Noe, 2017).

Heroin, known as diacetylmorphine, was resynthesised from morphine by Bayer Pharmaceuticals in Germany in 1897. The name is derived from the German “heroisch” which translates to heroic. Initially it was marketed as an over the counter non-addictive cough suppressant medication, as well as a cure to morphine addiction. However, research soon found that heroin was more addictive than morphine and by 1910 it became the primary illicit drug in the United States and was widely used throughout Europe (Pecoraro, Ma & Woody, 2012).
When administered either by snorting intra-nasally, smoking or intravenous injection, heroin produces a rapid onset of euphoria. Heroin penetrates the blood-brain barrier and is metabolised to the active compound 6-monoacetylmorphine (6-MAM) within 5-15 minutes. This conversion to 6-MAM and then to morphine characterises heroin as a pro-drug (Koob & Moal, 2006). As opiates are respiratory depressants (Strang, 2015), effects of heroin include bradycardia and hypoventilation. Other effects include flushing of the skin, heaviness in the extremities, dry mouth, constriction of the pupils, hypotension and itching. Once the euphoria passes, the user fluctuates between drowsy and wakeful states (Seidler, 2001).

Withdrawal from heroin occurs rapidly due to the short half-life of 6-MAM (30 minutes) and its metabolite morphine has an elimination half-life of 2-4 hours (Lobmaier et al., 2010). Withdrawal typically commences within four to 12 hours post dosage and peaks after two to four days. Acute withdrawals can occur for as long as seven to ten days post dosage. Withdrawal symptoms may include anxiety, muscle cramps, sweating, dilated pupils, hypertension, insomnia, tachycardia, tremors, emesis, restlessness, rhinorrhea and lacrimation (Seidler, 2001; Koob & Moal, 2006). Due to the short half-life of heroin and the timeframe of experiencing withdrawal symptoms, the frequency of heroin usage is much higher than other illicit substances as 49% of users have documented using heroin as often as daily or weekly (AIHW, 2016).

In addition of withdrawal symptoms, the most prominent opiate-related health problems recorded include infections, gastrointestinal disorders, musculoskeletal and connective-tissue disorders, general disorders, surgical complications and mortality (Schroeder, Schmittner, Epstein & Preston, 2005). Overdose is the most prominent cause of death within an opiate dependent demographic, with violence and suicide adding to mortality (Bell, 2012a). For example, throughout 2014 a total of 47,055 drug overdose deaths were
recorded in the United States and more than three out of five deaths involved opioid usage (United Nations Office on Drugs and Crime, 2016).

Additionally, as heroin is commonly injected, health implications such as HIV, hepatitis B, hepatitis C and other injection related illnesses have been found (National Institute on Drug Abuse, 2015). In 2011, out of the 14 million people globally who inject substances, 1.6 million were HIV positive, 1.2 million had hepatitis B infection and 7.2 million people had hepatitis C infection (The Lancet, 2013). It has been found that 90% of new hepatitis C infections are a direct result of unsafe injection practices by injection drug users (Pecoraro, Ma & Woody, 2012)

2.3 Opioid Dependency Treatment.

2.3.1 Detoxification.

The goal of detoxification is to provide supervised withdrawal from a drug of dependence in an attempt to reduce withdrawal symptoms and potential medical complications (Mattick & Hall, 1996). Opioid detoxification programs, both inpatient and outpatient, have reported varied successful completion rates between 4-100%. Inpatient programs have typically reported higher levels of treatment completion as well as lower opiate relapse rates post detoxification treatment. However, inpatient detoxification programs are typically 20 times more expensive than community detoxification programs (Day & Strang, 2011).

As detoxification alone has been found to lead to high rates of relapse, literature details that detoxification as a stand alone treatment for opioid dependence is ineffective (Chalana et al., 2016; Lobmaier et al., 2010; Nosyk et al., 2014). Research shows that only 20-30% of individuals remain abstinent one month after detoxification (Stein, Anderson &
Running Head: STRESS AND LOC ON OPIOID DEPENDENCY

Bailey, 2015), with relapse rates after 12-36 months post opioid detoxification ranging between 72-88% (Chalana et al., 2016).

Early relapse after inpatient detoxification has been predicted by factors such as younger age, greater heroin use, history of injecting and failure to attend aftercare while relapse in an outpatient detoxification program has been attributed to interpersonal variants, drug-related cues (meeting other drug users, being offered substances) and negative affect (Chalana et al., 2016). Research has also found outcomes for detoxification are lower than for individuals who received other treatments including methadone maintenance, therapeutic community or outpatient residential treatment (Lobmaier et al., 2010). Although detoxification still plays an essential step in recovery, it is recommended that medically assisted detoxification should act rather as a bridging treatment between maintenance and abstinence, taking into consideration vocational, family, medical and psychiatric issues of the individual (Day & Strang, 2011).

Research has identified that the length of remaining in alternate treatment services once discharged from a detoxification setting has been found to be statistically significant for males with alcohol use disorders. The after care variant included appointments with a counsellor, nurse or physician at an outpatient facility (Romelsjo et al., 2005). Growing international literature supports the notion that continuous long-term care is pivotal in clients remaining abstinent from substances and is a sufficient “turning point” in developmental recovery trajectory (Manning et al., 2017). Therefore, it is suggested that once a detoxification program has been completed, individuals should be consulted about care options (e.g medication-assisted, residential, intensive supportive counselling) to reduce the “revolving door” of serial detoxification admissions (Stein et al., 2015), with appointment scheduling and timeframe of aftercare attendance being important factors.
2.3.2 Opioid Substitution Therapy

From the 1960s onward, Opioid Substitution Treatment (OST) was implemented to suppress withdrawal symptoms due to opioid addiction (Bell, 2012a). Currently, the pharmacotherapy for opioid addiction involves three maintenance medications: methadone, buprenorphine and suboxone. The goal of OST is not to achieve drug abstinence; rather its purpose is to improve quality of life by enabling the individual to live without illicit substance use and to reduce harms and cost for society (Lobmaier et al., 2010).

Methadone is a full $\mu$ opioid receptor agonist with an average half-life of 24 hours, ranging between 13-50 hours (Lobmaier et al., 2010). It has an oral bioavailability of approximately 80% and the initial effects occur within 30 minutes of oral ingestion. Due to the long half-life of methadone, it is a highly effective pharmacologic treatment for opioid dependency as patients only require a once a day dosage, between 80-120mg per day, to diminish withdrawal symptoms (Saxon et al., 2013). Methadone was developed in 1943 by I. G. Farben and an appropriate dose to create a stable plateau between withdrawal and sedation was discovered in 1964 by Dole and Nyswander (Dole & Nyswander, 1965; Pecoraro et al., 2012).

Contrastingly, buprenorphine is a partial $\mu$ opioid receptor agonist and a $\kappa$ receptor antagonist (Ling et al., 2011). When compared to methadone, buprenorphine has a poor oral bioavailability (16%) and is rather ingested through sublingual formulations (Lobmaier et al., 2010). Sublingual buprenorphine tablets have a bioavailability average of 35% and have an average half-life of 32 hours. Stabilisation doses of buprenorphine range between 2-32mg per day and typically the initial effects of buprenorphine in sublingual form appear within 30 minutes (Saxon et al., 2013).

Buprenorphine has a greater margin of safety than methadone due to its less severe withdrawal symptoms in comparison to full agonists (Pecoraro et al., 2012). Additionally,
buprenorphine showcases a ceiling effect on $\mu$ opioid receptor activity as further doses after ingesting the initial treatment amount do not increase the effects which therefore significantly reduces the risk of respiratory depression and overdose (Saxon et al., 2013).

Lastly, the compound suboxone containing a $\mu$ opioid partial agonist (buprenorphine) and an antagonist (naloxone) was developed due to misuse of methadone and buprenorphine. Naloxone alone has a poor bioavailability in sublingual form (1%), but a high bioavailability if injected (70%) which makes suboxone less attractive for diversion. Naloxone alone competes with agonists at opioid receptors and displaces them, resulting in effectively reversing opioid intoxication (Lobmaier et al., 2010).

To date, the Methadone Maintenance Treatment (MMT) program is the most common pharmacological treatment of heroin addiction at a global scale (Bell, 2012b). However, the OST program as a whole has been criticised as it ultimately offers rehabilitation without a cure (Walsh, 1970). This inability to cure opioid addiction is evident as 60% of heroin users relapse approximately three months post treatment, while 75-85% of users relapse after 12 months of abstinence (Brown & Lawrence, 2009). Another criticism of the OST program is that by continuing the consumption of an opiate substitute, the OST program may impede and extend the recovery process as patients remain drug-dependent (McKeganey, Russell, & Cockayne, 2013) and therefore never learn how to live a drug free lifestyle.

Further, although the OST program aims to reduce opioid withdrawal symptoms and inhibit drug behaviours, continued drug use appears to remain a major concern during the treatment program (Iguchi et al., 1988). For example, in a sample of 209 participants completing buprenorphine treatment for opioid dependency, 25% tested positive for a single substance while 18% indicated two or more substances. Toxicology results for the 89 participants who tested positive showed 57% used opiates, 36% cannabis, 28% benzodiazepine, 15% cocaine and 7% amphetamine (Campbell et al., 2016). Additionally, a
pre-assessment for a detoxification treatment found that out of 73 participants who were receiving methadone treatment, 28 individuals (41.2%) had used one illicit substance in addition to heroin, 20 (29.4%) had used two more substances, eight (11.8%) had used three substances and one (1.5%) had used four substances in addition to heroin (Day & Strang, 2011).

In conclusion, contemporary research identifies a literature gap in understanding why substance dependent individuals continue to engage in drug activity despite receiving SUD treatment. Therefore, the present document will explore the influence of uprising stress models and alternate individual variants that may be predictive of continued relapse behaviour. However, it should be noted that biological factors of personality and genetics that have previously been found to predispose an individual in developing SUDs (Neilsen & Kreek, 2012; Levran et al. 2014; Eitan et al., 2017) will not be explored.

2.4 Stress

As relapse remains problematic, contemporary addiction literature suggests that stress conditions are a vital contributor to relapse (Sinha, Shaham & Heilig, 2011; Goldstein & Volkow, 2011). The term “stress” refers to the processes involving perception, appraisal and response to fearful, threatening or challenging events or stimuli (Sinha, 2008). In humans, the stress response commences with the HPA axis “with the release of corticotropin-releasing factor (CRF) from the hypothalamus, stimulating the release of adrenocorticotropic hormone (ACTH) from the pituitary gland. ACTH travels through the bloodstream to stimulate the release of cortisol from the adrenal cortex” (Jackson, Knight, & Rafferty, 2010, p. 934).

Stress-induced relapse has been modeled in animal laboratory experiments using a reinstatement model whereby drug-seeking behaviours are established, extinguished and then reinstated with acute exposure to certain stressors (Sinha, Shaham & Heilig, 2011). It was
found in rats that stressors such as food deprivation (Shalev, Highfield, Yap & Shaham, 2000), footshock (Stewart, 2003), tailpinch, social isolation (Sinha, 2008), social stress and administration of the stress neurohormone corticotrophin-releasing factor (CRF) (Brown & Lawrence, 2009) reinstated self-administered opiate seeking after drug-seeking behaviour was extinguished. However, the degree to which stressors and environmental determinants reinstate drug seeking is not fully known in humans (Shalev et al., 2000; Sinha, Shaham & Heilig, 2011).

Literature has identified that vulnerable populations exposed to greater stressors including immigrants (Savage & Mezuk, 2014) and homeless individuals (Galaif, Nyamathi & Stein, 1999) experience higher rates of drug use behaviour than control populations. For example, in a Latino and Asian American population within the United States of America, psychosocial and contextual stress factors of acculturation, acculturative stress, neighborhood characteristics, family characteristics and discrimination were analysed in relation to developing alcohol use and drug use disorders. Results found acculturation, family conflict and discrimination were positively associated with alcohol and drug use disorders (Savage & Mezuk, 2014).

Between 1994 to 1996, a sample of 1,179 homeless women were surveyed to examine the relationship between psychosocial factors and three different drug use constructs (drug use, drug problems and physical drug dependence). Results found that drug use was predicted by negative social support, depression and less positive coping. Drug problems were predicted by more negative coping, depression and less positive coping. Lastly, physical drug dependence was predicted by more negative social support, depression and less positive social support. Collectively these results highlight the importance of investigating the positive and negative psychosocial factors of individuals presenting with drug use dependency (Galaif, Nyamathi, & Stein, 1999).
The need for treatment plans to identify psychosocial stressors is further strengthened as social networks also play a role in developing drug-associated behaviours. Based on social learning theory, whereby substance using peers increase the risk of an individual engaging in drug related activity, has detailed that individuals who belong to social networks with increased concentrations of substance users are more likely to use substances themselves (Gibson et al., 2014). Partaking in drug associated activity as a means of social interaction was also found to be highly correlated with continued drug use (Galea, Rudenstine & Vlahov, 2005).

The quality of the social network has been found to be associated with substance use within particular activity spaces of a community. Network quality was determined by the calculation of substance use, with positive influence and negative influence scores for each network member listed at every activity space. Higher network scores indicated a more positive network quality while lower scores indicated the opposite. From the data collected from 90 young males, the study concluded that engagement in risky activity spaces was associated with lower social network quality and increased substance use (Gibson et al., 2015).

Environmental stressors have also been found to be associated with engagement in drug-related activity (Galea, Rudenstine & Vlahov, 2005). For example, areas of greater disadvantage including regional and more remote communities have higher rates of opioid utilisation and problematic opioid use (Degenhardt et al., 2016) as individuals residing in disadvantaged neighbourhoods have been found to encounter more social stressors, more frequent negative life events, criminal victimisation, illness, higher levels of social strain and higher levels of psychological distress.

A study of 139 adolescents found that relative neighborhood disadvantage was associated with substance use after controlling for age, sex and race factors. Additionally,
relative neighbourhood disadvantage has been associated with stress and this result is moderated by substance use, whereby the positive effect of neighbourhood disadvantage on stress is stronger for individuals with heightened substance use (Mennis et al., 2016). Further, within a Detroit population, Boardman et al. (2001) found that neighbourhood disadvantage was highly correlated with drug use beyond the influence of socioeconomic and sociodemographic factors. The study concluded that one unit increase in the neighbourhood disadvantage score increased the odds of using illicit substances by 8.2%.

Collectively, the neighbourhood stress-based framework identified that disadvantaged neighbourhood features including poverty, crime, decreased social resources, decreased access to psychological services and increased risk of psychological distress, act as chronic risk factors whereby individuals may resort to substance use to alleviate these stressors (Gibson et al., 2015). For example, for the many black Americans who live in difficult environments involving stressful living conditions, the most readily accessible option for address stress are numerous unhealthy behaviours including smoking, drinking, drug use and overeating (Jackson et al., 2010).

Another stressor of financial instability has previously been identified as contributing stress factors in relapse. For example, Siahp push and Carlin (2006) found that smokers with higher financial stress subtypes were less likely to quit, and ex-smokers were significantly more likely to relapse. The results highlighted a casual relationship of financial stress to a reduced probability of cessation and increased probability of relapse (Siahp push & Carlin, 2006).

Additionally, a study conducted with electronic diary data found that within a methadone receiving participant group, a positive association between ratings of background stress that was not prompted by a particular negative event (saw heroin, saw other using, was offered, tempted out of the blue, wanted to see what would happen, handled more than $10
US) and opioid craving. Stress was also positively associated with negative mood and negatively associated with feelings of happiness and relaxation (Preston & Epstein, 2011).

Together, stress can present itself in many forms, with individuals being exposed to numerous stressful contexts during their lifetime. As contemporary addiction literature has demonstrated that a variety of stress determinants are highly correlated with substance recidivism, it is noted that relapse often does not follow from exposure to a singular stress factor (Del Pozo, Gomez, Fraile & Perez, 1998). Similarly, research has not identified a prominent stress factor model, inclusive of all stress variants, that is predictive of continued substance use.

2.4.1 Psychological model.

According to the psychological model of addiction, the stress reduction hypothesis identifies that human drug use is utilised as a coping mechanism in regards to stressful contexts and negative life experiences (Boardman, Finch, Ellison, Williams & Jackson, 2001; Sinha, 2008). The cognitive behavioural formulation identifies coping strategies as a class of cognitive or behavioural patterns that effectively deal with problematic situations (Gossop et al., 2002). This analogy is similar to the self-medication hypothesis whereby individuals use substances to cope with tension elicited by life stressors, or to relieve pain due to psychological distress (Goeders, 2004).

Both hypotheses depict the phenomenon of instrumental learning where addiction rises due to the operation of reward and punishment, a motivational system operating outside of conscious awareness. Operant learning is made up of two elements: positive and negative reinforcement. Drug dependence through positive reinforcement occurs when the substance acts as an award and with continuous repetition, the cue-response-reward association becomes stronger. In regards to stress relief, drug dependence through negative reinforcement involves consuming substances to escape or avoid unpleasant stimuli (West, 2006).
As an extension of the negative reinforcement model, the affective model of drug motivation (Baker et al., 2004) details that when an individual has low levels of negative affect (NA) they are unaware of the NA and rather engage in drug use due to it becoming an automatic stress coping behaviour. However, at high levels of NA, the individual has insight about the NA. Due to not being able to effectively cope and engage in drug use without cognitive control, NA therefore increases the incentive value of drug use (al'Absi, 2006).

These models of addiction have demonstrated that drug activity is associated with maladaptive coping in response to an array of stressful situations including conflict, daily hassles (ElGeili & Bashir, 2005) and major negative life events (Slopen et al., 2013). For example, research has found that adolescent and adult drug dependent populations are predominantly found to use avoidant coping strategies (Sinha, 2001). A study conducted with participants from 23 residential programs, all of who reported heroin usage three months before entering treatment, highlighted the relationship between coping strategies (avoidance, cognitive and distraction techniques) and relapse. Participants that remained abstinent after completing treatment reported statistically significant use of all three categories of coping techniques than at intake. In contrast, relapse participants reported no change in initiating the three types of coping responses (Gossop et al., 2002). The inability to effectively cope with stress and high rates of drug use was also identified by Skalski et al. (2013) in a HIV-positive adult sample. Results showed that participants who use self-destructive avoidant strategies (e.g. “I started an argument to get my anger out”) were significantly more likely to use drugs (Skalski et al., 2013).

Other emotional coping mechanisms such as denial, disengagement and withdrawal from the stressful situation have been found to correlate positively with on-going substance use. For example, in a younger high-risk population, coping strategies such as disengagement were found to perpetuate substance use, while proactive task-oriented coping deter substance
use (Wong et al., 2013). Specifically, individuals engaging in emotional suppressor strategies were significantly more likely to engage in drug use activity and report more severe problems in drug engagement than individuals that use adaptive coping strategies at an earlier age.

As avoidant strategies are usually utilised when the individual has limited ability to resolve, or lacks appropriate resources to solve the present stressors, treatment models need to focus on educating and teaching drug dependent persons adaptive coping skills and effective strategies to manage drug cues, craving and stress (Sinha, 2001).

2.4.2 Neurological model.

Alternatively, the neurological model of addiction highlights that substance abuse leads to various dysfunctions of cognitive and stress circuits that drive relapse behaviour. For humans, stress responses are elicited by the corticotrophin-releasing hormone (CRH), the locus coeruleus (LC)-noradrenaline autonomic system, the pituitary-adrenal axis and the autonomic systems. The stress system triggers the release of glucorticoids (cortisol) from the adrenal cortex, adrenaline and noradrenaline from the sympathetic nerves and adrenal medulla (al’Absi, 2006).

In the last two decades, functional neuroimaging techniques have highlighted that the key brain regions underlying addiction are made up of a network of interdependent and overlapping circuits. Research has previously documented dysfunction in reward circuits involving the nucleus accumbens and ventral pallidum as well as memory and learning circuits involving the amygdala and hippocampus. Cognitive control located in the dorsal anterior cingulate cortex and motivation located in the orbital frontal cortex have also been found to be involved in the initiation and maintenance of addiction (Jiang et al., 2011; Sinha, 2009).

Stress responses are mediated by the hypothalamic-pituitary-adrenal (HPA) axis and addiction has been found to deregulate the HPA axis resulting in the inability to adaptively
and appropriately respond to stress (Zhang et al., 2011). Additionally, alterations in stress and reward pathways such as the hypothalamic corticotrophin releasing factor (CRF) system as well as alterations in dopaminergic activity due to excessive drug use have been evident in some cases (Sinha, 2009). The importance of the CRF system in regards to engaging in stress reactive drug seeking behaviour has been noted whereby the pharmacological manipulation using CRF medications have been found to alter stress responses and block stress-induced reinstatement of drug use (Moeller, 2012).

Cognitive deficits in brain regions responsible for healthy neuropsychological functioning that are not primarily linked to stress pathways due to substance dependency, may further prompt relapse behaviour due to the inadequacy to manage stress. For example, substance use has been found to lead to disruption of the prefrontal cortex (PFC), a region fundamentally responsible for encompassing emotion, self-control, salience attribution, cognition and behaviour (Goldsetin & Volkow, 2011). Abnormalities of the PFC have been found to result in dysregulated signaling to the mesolimbic reward system, resulting in the reduced ability to use judgement, restrain impulses and a higher tendency to engage in compulsive behaviours (Kosten & George, 2002).

Other brain regions affected by chronic opioid use includes the thalamus, temporal insula and sensorimotor structures (Motlagh et al., 2016). In addition to the deregulation of neuropsychological pathways, opioids have been found to indirectly affect opiate receptors involved in dopamine modulation within areas of the brain that are rich in dopamine receptors (Myers et al., 2017). Gray matter deficits within the pre frontal and temporal cortices have also been found in opioid dependent patients. Gray matter contains most of the neuronal cells of the brain responsible for muscle control, sensory perception, memory, emotion, speech and decision making abilities (Motlagh et al., 2016).
Neuroadaptations of brain regions due to excessive substance abuse have also been found to increase in the incentive salience of drugs whereby exposure to drugs and drug-related stimuli result in an excessive craving (Sinha, 2001). Research has identified in comparison to control participants, heroin addicts were found to excessively attribute salience to drug-related cues (Goldstein & Volkow, 2011). Functional Magnetic Resonance Imaging (fMRI) studies have also highlighted enhanced neuroanatomical brain responses in the amygdala, extended limbic system and regions of the frontal cortex in response to salient heroin cues in Methadone Maintenance Treatment (MMT) patients (Langleben et al. 2008; Sinha 2009; Wang et al. 2011).

Event-related potentials (ERPs) recorded by an electroencephalogram (EEG) provide a direct measure of cortical activity with sufficient temporal precision to distinguish between rapid perceptual and cognitive processes that occur in response to specific stimuli (Lubman et al., 2007). ERPs show various frequencies of oscillations with specific spatial distribution with different states of brain functional interaction between brain regions at alternate frequency bands. EEG research has found that chronic heroin dependency results in altered ERP power, coherence and information processing ability abnormalities in various brain regions (Motlagh et al., 2016).

ERP research also details that the P300 component of the brain reflects the process of pre-motor decisional processes such as memory updating (Lubman et al., 2007) and ‘context-uploading’ whereby salient stimuli attract greater attentional resources and produce larger P300s (Lubman et al., 2008). Enhanced P300s have been repeatedly reported in response to drug-related stimuli than non-drug stimuli (Lubman et al., 2007; Lubman et al., 2008). Due to the increased activation of the P300 in response to drug-related stimuli, the P300 therefore may be an “objective index of the incentive motivational properties of drug cues in addiction” (Lubman et al., 2007, p. 1208).
Additionally, P300 amplitude has been found to be influenced by opioid usage as a significant decline in P300 amplitude had been noted in heroin-addicted participants in comparison to controls. As the P300 is found to restore during abstinence periods, the P300 result yields clinical implications as it can be used as a central nervous system recovery measurement due to the association of the P300s amplitude and abstinence duration (Motlag et al. 2016). Collectively, electrophysiology results suggest that incentive salience to drug-related stimuli in habitual users, as well the P300, may be influential factors of relapse behaviour.

In relation to length of abstinence, cognitive deficits appear to exist after varying opioid abstinence time frames. For example, decision-making deficits were found between short-term abstinence groups (3-30 days), longer-term (3-12 months) and 24 month groups. These results highlight that stress continues to disrupt decision making in the short-term and longer-term detoxified patients (Zhang et al., 2011). Even when abstinent individuals are matched to a control group, early abstinent opioid dependent persons were found to perform significantly worse in working memory and fluid intelligence than their normal control counterparts (Rapeli et al., 2006). These results suggest that executive functioning deficits may occur up into late abstinent periods and treatment programs will need to cater to reduced higher order cognition and dysregulated emotion throughout the early abstinence journey.

In summary, neuroimaging results can help explain and understand cognitive abnormalities and significant functional impairments in opioid dependent individuals in tasks such as response inhibition, decision making and working memory have repeatedly been found (Jiang et al., 2011). Neuroimaging results also demonstrate that although methadone suppresses withdrawal symptoms, it does not alter and correct neural substrates impaired due to opioid addiction (Wang et al. 2011).
2.5 Individual Factors.

2.5.1 Demographics.

Males have been found to typically report greater drug use disorders, drug abuse and drug dependence as well as a higher rate of alcohol abuse and dependence than females (Moeller 2012; Broome, Hurley & Taber, 2010). Males are also found to use all drug types, illicit and licit, more than females (Rodgers, 2015). However, women are noted to experience overall negative effect due to consequences of drug use earlier and in greater thresholds than males (Broome et al., 2010).

Young adulthood, 18-29 years, has been listed as a high-risk time for substance use and misuse behaviour development in comparison to other age groups (Quek et al., 2013) as drug use experimentation has been found to significantly increase throughout adolescence (Adlfa, Hamilton, Wu & Noh, 2009). The relationship between education and relapse rates is widely documented as relapse is significantly higher for individuals with lower levels of education in comparison to higher educated patients (Pashaei et al., 2013).

Factors such as low socioeconomic status and unemployment have also been found in being positively correlated with relapse (Del Pozo et al., 1998) while unemployed patients have been noted to have a higher risk of relapse in comparison to individuals with full-time roles (Pashaei et al., 2013). Specifically, an American study highlighted that a 1% increase in a regional unemployment rates was associated with a 3% increase in opioid overdose deaths and a 7% increase in emergency department admissions (Cantor, Stoller & Saloner, 2016). Alternate factors such as higher dosage use, greater frequency of use, longer duration of use, daily use and heightened degree of contact with other drug users also highlight positive correlations with relapse (Gossop et al., 2002).

Combinations of these demographic variants have been found to make an individual more susceptible for engaging in drug use. For example, a study conducted with a methadone
population for opioid dependency, found that patients with THC-positive urines were more likely to be male and have a longer history of cannabis use than patients with no THC-positive urines (Ghitza, Epstein & Preston, 2007). A predictive model for drug dependence also highlights the demographic factors of being younger, male, never married, less than 12 years of education and unemployed make an individual most vulnerable in developing a substance use disorder (Teesson et al., 2006). Alternatively, factors of being older in age, female, holding full time employment and no criminal justice involvement were associated with longer and successful treatment completion (Nosyk et al., 2014).

2.5.2 Locus of Control.

At an individual level the Locus of Control (LOC) concept has also been identified as an influencing factor in relapse vulnerability. Derived from social learning theory, the LOC is a paradigm that identifies how an individual perceives the connection between their actions and its consequences (Jafari & Shahidi, 2009). When an event is perceived by an individual as the result of luck, fate or chance, this is analysed as an external control belief. Alternatively, if the event is perceived as contingent upon their behaviour, this is analysed as an internal control belief. The LOC concept hypothesises that if an individual perceives an event as contingent upon their own behaviour, a positive reinforcement will strengthen the potential for that behaviour to occur in a similar situation, while a negative reinforcement would result in weakening the behaviour. Contrastingly, if perceived as being out of their control, the behaviour is less likely to be strengthened or weakened (Rotter, 1966). Individuals with a greater internal LOC orientation are confident with their own personal agency while an external locus of control orientations are characterised by feelings of powerlessness (Kao et al., 2014).

The LOC scale has previously been used to predict and explain the onset and continuous use of substances (Mokwena & Fernandes, 2014). Research notes that substance dependent
persons yield high external LOC scores and report lower internal LOC scores than non-addicted participants (Jafari & Shahidi, 2009; Fernandes & Mokwena, 2016). Current substance users were also found to exhibit higher external LOC scores than methadone and former user groups (Kao et al., 2014). Alcohol-dependent individuals were also found to have greater external scores than abstinent individuals recovering from an alcohol-use disorder (Ersche et al., 2012). Results indicate that through the LOC model, the more an individual feels controlled by external forces, the less likely they are to take responsibility and be proactive about their continued substance use behaviours (Booth-Butterfield, Anderson & Booth-Butterfield, 2000).

Other findings unrelated to substance use derived from the LOC model include that low self-esteem, stress as a result of feeling powerless, poor school achievement and ineffective stress management have also been found to be associated with an external LOC score (Mokwena & Fernandes, 2014). Contrasting, individuals showcasing an internal locus of control profile are more likely to adhere to health regimes and engage in preventative health behaviours (pap screenings, breast exams, healthy diets, wearing seatbelts) and report a greater quality of life than externally orientated persons (Booth-Butterfield et al., 2000).

3.0 Aims and Hypotheses

As drug dependency research is primarily conducted in the United States, the current study aims to expand our understanding of the relationship between numerous variables associated with developing SUDs in an Australia population. The study is also guided by research demonstrating that Australians are more likely to have alcohol or drug dependence than American counterparts after controlling for demographic variables (Teesson et al., 2006). The goal of the report is to assist in closing the literature gap in highlighting predictor variables for continued substance use during OST services. Together, the report aims to identify key health and economic considerations for SUD treatment within the public health
sector. This study also aims to be the first to collect data implementing the PSS-10, LOC and the Inventory of Drug Taking Situations (IDTS) measures to explore the relationships between stress, LOC, high-risk contexts and demographic profiles on relapse vulnerability within an Australian OST program.

The present study hypothesises that firstly, the demographic factors of the male gender, aged between 18-29 years, lower education level, unemployed, single relationship status and long-term opioid use will be associated with higher levels of drug use than their counterparts. Secondly, it is hypothesised that higher stress scores will predict continued drug use. Thirdly, external LOC scores will predict continued drug use. Fourthly, it is hypothesised that IDTS high problem scores for the negative situations profile will be significantly associated with continued drug use.

Lastly, hypothesis five depicts that if hypothesis one, two, three and four are supported, a model of demographic, stress, LOC and IDTS factors will significantly predict continued drug use during the OST program.

4.0 Method

4.1 Participants

Fifty participants, 30 males and 20 females, were initially recruited for the present study. Inclusion criteria included currently receiving OST for opioid dependency at RPAH Drug Services, Camperdown, Australia. Exclusion criteria included being under the influence of substances at the time of survey completion. A total of sixteen participants were excluded from the study for either not providing urine samples for routine toxicology screening or not completing all questionnaire materials.

The final participant group included thirty-four participants, 19 males and 15 females, aged between 19 and 64 ($M = 43.36, SD = 10.11$). Participants recorded employment status
was 71% unemployed, self-employed (24.4%) and casual (0.06%). The majority of participants were listed as single (69.69%), while others detailed living with a partner (24.24%), separated (3.03%) or divorced (3.03%). Three participants (8.8%) disclosed they were referred to the OST program due to opioid dependency, while the remainder 31 participants (91.2%) started treatment due to opiate (heroin) dependency.

Participants, regardless of completing all components, were reimbursed for partaking in the study with a $20 Woolworths essentials food gift voucher. The study was approved by the Ethics Review Committee (RPAH Zone) of the Sydney Local Health District (Protocol number X17-0053).

4.2 Materials.

4.2.1 Demographic variables.

Demographic factors including gender, age, education level, employment status, marital status, time of last drug self-administration and timeframe of lifetime drug use were recorded (Appendix B).

4.2.2 Perceived Stress Scale (PSS).

The 10 item Perceived Stress Scale (PSS-10) (Appendix C) was administered as it provides a universal assessment of stress, as items are sensitive to both the non-occurrence of events and to ongoing life circumstances (Ezzati et al., 2014). This scale provides a unique stress variable whereby a participant’s stress score reflects the level of stress experienced due to how unpredictable, uncontrolled and overloaded patients find their lives (Mitchell, Crane & Kim, 2008). Ultimately, it can identify the degree to which situations are perceived as excessively stressful relative to their ability to cope (Taylor, 2015).
Higher PSS scores have been associated with higher levels of negative affect, depression, anxiety (Ezzati et al., 2014) as well as physical health outcomes (Mitchell, Crane & Kim, 2008). The scale showcases high reliability scores with a Cronbach alpha ranging between 0.84 (Taylor, 2015) and 0.91 (Mitchell, Crane & Kim, 2008).

4.2.3 Locus of Control (LOC).

Rotter’s (1966) Internal-External LOC Scale (Appendix D) has 29 items that highlight the degree to which individuals think events are under their control, or the control of external forces. The higher the score, the more the external control is used by the respondent (Jafari & Shahidi, 2009). Internal consistency estimates for Rotter’s LOC Scale ranged between 0.65 and 0.79 and test-retest reliability estimates ranged between 0.49 and 0.83 (Rotter, 1966).

4.2.4 Inventory of Drug Taking Situations (IDTS).

The IDTS (Appendix E) is a 50-item self-report measure established by Annis and Martin (1985) that analyses intrapersonal environmental and interpersonal factors. The instrument comprises of 8 categories that are labelled as high-risk situations [negative emotion (NE), physical discomfort (PD), positive emotion (PE), testing personal control (TPC), urge and temptation (UT), conflict with others (CO), pleasant time with others (PT) and social pressure (SP)]. High-risk situations are defined as stressful situations that pose a threat to the individual’s sense of control and increase the risk of relapse (ElGeili & Bashir, 2005). The eight scales combine to form three global categories: negative situations (UE, PD and CO), positive situations (PE and PT) and temptation Situations (UT, SP and TPC).

The IDTS yields high levels of validity and reliability with a Cronbach coefficient alpha of .95 for the total scale, and a range of 0.7 to 0.87 for the subscales (Hartwell, Back, McRae-Clark, Shaftman & Brady, 2012).
Previous research has identified positive correlations between the IDTS subscales of NE, CO, UT and heroin recidivism within a Saudi Arabian population (El Sheikh & Bashir, 2004). Additionally, demographic factors such as financial status and education are negatively correlated with the interpersonal situations of SP, PT and CO subscales (ElGeili & Bashir, 2005). ElGeili and Bashir (2005) also state that TPC and SP situations are also significant predictors of relapse as they fuel UT.

4.2.5 Substance use.

Continued drug use was identified through routine urine drug screening tests conducted at RPAH Drug Health Services. Immunoassay urine toxicology screenings are conducted by RPAH to confirm patients are consuming the OST medication as well as identifying the consumption of alternate substances (NSW Department of Health, 2006). Participant’s electronic medical records were accessed four to six weeks after completing the measurements to identify drug use. Urine toxicology results were screened for the following drug classes: benzodiazepines, cannabinoids, cocaine & metabolites, opiates, methadone metabolite and amphetamine type substances. Positive toxicology results were indicated with cutoff values for the various drug classes: benzodiazepines (200ug/L), opiates (300ug/L), cocaine (300ug/L), amphetamines (100ug/L) and cannabinoids (50ug/L).

It should be noted that RPAH does not terminate the patient from treatment if continued substance use or polydrug use is flagged through immunoassay toxicology reports.

4.3 Procedure.

Participants were approached in person at the RPAH Drug Health Services waiting room and invited to attend an interview room within the hospital to complete a series of questionnaires. Before the commencement of the data collection, all participants were required to read an information sheet (Appendix F) and asked to voluntarily sign a consent
form (Appendix G). The questionnaires were then distributed in paper format collectively taking 15 – 30 minutes for participants to complete.

During the screening process, data was collected on a number of potential predictors of relapse: demographic variables (age, sex, employment status, educational status, relationship status, last use of opioids and life-time use of opioids); stress (Perceived Stress Scale [PSS-10]), control belief (Locus of Control [LOC]) and high-risk contexts [IDTS]).

Routine urine toxicology results conducted by RPAH staff were reviewed four to six weeks post initial questionnaire screening, which was to investigate whether participants had used opiates or other illicit substances while completing the OST program.
5.0 Results

5.1 Demographics

5.1.1 Gender.

Overall, 16 males (47%) and 11 females (32%) tested positive to either benzodiazepines, opiates, amphetamines or cannabinoids. No participant had positive readings to cocaine and metabolites. For each drug class (see Figure 1.1), seven males (21%) and six females (18%) tested positive to benzodiazepine, four males (12%) and three females (9%) for opiates, seven males (21%) and seven females (21%) to cannabinoids and seven males (21%) and four females to amphetamines (12%).

Figure 1.1. Gender and positive toxicology results

Two-way chi-square analyses revealed not significant relationships between type of gender and cannabinoid use \( \chi^2 (1, N = 34) = 0.56, p > 0.05, \) Cramer’s \( V = .09 \) and benzodiazepine use \( \chi^2 (1, N = 34) = 0.85, p > 0.05, \) Cramer’s \( V = .03 \). Frequencies are shown in Table 1.1
All drug class use, opiate use and amphetamine use could not be analysed through chisquare analyses as the assumption of expected frequency in each category was violated.

**Table 1.1**

*Frequencies, percentages and adjusted standardised residuals for positive and negative toxicology results for male and female gender.*

<table>
<thead>
<tr>
<th></th>
<th>Positive toxicology result</th>
<th>Negative toxicology result</th>
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<tr>
<td>Cannabinoids</td>
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<td>Male</td>
<td>7</td>
<td>36.8%</td>
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<tr>
<td>Female</td>
<td>7</td>
<td>46.7%</td>
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<tr>
<td>Benzodiazepines</td>
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</tr>
<tr>
<td>Male</td>
<td>7</td>
<td>36.8%</td>
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<tr>
<td>Female</td>
<td>6</td>
<td>40%</td>
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*Note: f = frequency, % = percentage within type of gender, $f_e$ = expected frequency, ASR = adjusted standardized residuals.*

**5.1.2 Age.**

**19-24 years**

One participant (3%) indicated age between 19-24 years old. Toxicology result reported benzodiazepine (100%), cannabinoid (100%) and amphetamine (100%) use for this age group.

**25-39 years**

Seventeen participants (50%) indicated age between 25-39 years old. Toxicology result reported benzodiazepine (41%), cannabinoid (23.5%), opiate (23.5%) and amphetamine (100%) use for this age group.

**40-49 years**

Eight participants (23.5%) indicated age between 40-49 years old. Toxicology result reported benzodiazepine (37.5%), cannabinoid (50%), opiate (25%) and amphetamine (37.5%) use for this age group.

**50+ years**
Eight participants (23.5%) indicated age between 50+ years old. Toxicology result reported benzodiazepine (25%), cannabinoid (62.5%), opiate (12.5%) and amphetamine (62.5%) use for this age group (see Figure 1.2).

**Figure 1.2.**

*Age group and positive toxicology result*

![Age group and positive toxicology result](image)

*Figure 1.2. Benzo = benzodiazepine, Cannab = cannabinoid, Amphet = amphetamine*

Two-way chi square analyses could not be conducted to identify whether a statistically significant difference in the number of drug use for each age category as the assumption of expected frequency in each category was violated.

To balance the number of participants in each age category, the two younger brackets and the two older brackets were combined to produce a 19-39 years group (*n* = 18) and a 40-50+ years group (*n* = 16).

**19-39 years**

Results reported benzodiazepine (44.44%), cannabinoid (27.7%), opiates (22%) and amphetamine (16.6%) use for this combined age group.
40-50+ years

Results reported over all drug use (87.5%), benzodiazepine (31.25%), cannabinoid (56.25%), opiate (18.75%) and amphetamine (50%) use for this combined age group (see Figure 1.3).

**Figure 1.3.**
Combined age groups and positive toxicology result

![Bar chart showing drug use and positive toxicology results for 19-39 years and 40-50+ years.](chart.png)

*Figure 1.3. Benzo = benzodiazepine, Cannab = cannabinoid, Amphet = amphetamine*

Two-way chi square analyses could not be conducted to identify whether a statistically significant difference in the number of drug use for each age group as the assumption of expected frequency in each category was violated.

### 5.1.3 Employment.

Two participants (6%) indicated casual employment status. Positive toxicology results for benzodiazepine (50%) and amphetamine (50%) were found for this group. No participants indicated part time or full time employment status.
Eight participants (23.5%) indicated self employed employment status. Positive toxicology results for benzodiazepine (62.5%), cannabinoids (25%), opiate (37.5%) and amphetamine (37.5%) were found for this group.

Lastly, 24 participants (70.5%) indicated unemployment status. Positive toxicology results for benzodiazepine (29%), cannabinoids (50%), opiate (16.6%) and amphetamine (29%) were found for this group (see Figure 1.4).

**Figure 1.4.** Employment status and positive toxicology results

Two-way chi square analyses could not be conducted to identify whether a statistically significant difference in the number of drug use for each employment status category as the assumption of expected frequency in each category was violated.
5.1.4 Education.

Five participants (14.7%) indicated attending primary school. Positive toxicology results indicated benzodiazepine (40%), cannabinoid (100%), opiate (20%) and amphetamine (20%) use for this group.

Seventeen participants (50%) indicated attending high school. Positive toxicology results indicated benzodiazepine (53%), cannabinoid (29%), opiate (29%) and amphetamine (53%) use for this group.

Five participants (14.7%) indicated attending TAFE/private college. Positive toxicology results indicated benzodiazepine (40%), cannabinoid (80%), opiate (20%) and amphetamine (20%) use for this group (see Figure 1.5).

Figure 1.5.
Education and positive toxicology results

Figure 1.5. Benzo = benzodiazepine, Cannab = cannabinoid, Amphet = amphetamine

Two-way chi square analyses could not be conducted to identify whether a statistically significant difference in the number of drug use for each education status category as the assumption of expected frequency in each category was violated.
5.1.5 Relationship Status.

For this demographic, 23 participants (67%) indicated single relationship status. Twenty participants (83%) tested positive for drug usage with benzodiazepine (41.6%), cannabinoid (41.6%), opiate (16.6%) and amphetamine (33.3%).

Eight participants (23.5%) indicated cohabiting relationship status. Five participants (62.5%) tested positive for drug usage with benzodiazepine (37.5%), cannabinoid (25%), opiate (25%) and amphetamine (25%).

One participant indicated a separated martial status. The toxicology result found a positive cannabinoid (100%) and opiate (100%) result. One participant indicated a divorced martial status. The toxicology result found a positive cannabinoid (100%) and amphetamine (100%) result (see figure 1.6).

Figure 1.6. Martial status and positive toxicology result

Two-way chi square analyses could not be conducted to identify whether a statistically significant difference in the number of drug use for each relationship status category as the assumption of expected frequency in each category was violated.
5.1.6 Last Use.

1-3 days

One participant indicated opiate use 1-3 days prior to questionnaire completion. Results highlighted positive cannabinoid (100%) and opiate (100%) toxicology results for this group.

3 days-1 week

Three participants indicated opiate use 3 days - 1 week prior to questionnaire completion. Results indicated drug use for 100% of the sample including positive benzodiazepine (33%), cannabinoid (33%), opiates (67%) and amphetamine (100%) toxicology results for this group.

1 month

Two participants indicated opiate use one month prior to questionnaire completion. Results indicated drug use for 50% of the sample including a positive opiate (50%) toxicology result for this group.

1-3 month

Two participants indicated opiate use 1-3 months prior to questionnaire completion. Results indicated drug use for 50% of the sample including a positive opiate (50%) and benzodiazepine (50%) toxicology result for this group.

3-6 month

Five participants indicated opiate use 3-6 months prior to questionnaire completion. Results indicated drug use for 40% of the sample including a positive amphetamine (20%) and benzodiazepine (40%) toxicology result for this group.

6-12 month

Six participants indicated opiate use 6-12 months prior to questionnaire completion. Results indicated drug use for 100% of the sample including a positive benzodiazepine
(50%), cannabinoid (50%), opiates (17%) and amphetamine (33%) toxicology results for this group.

1-3 years

Four participants indicated opiate use 1-3 years prior to questionnaire completion. Results indicated drug use for 75% of the sample including a positive cannabinoid (50%) and amphetamine (50%) toxicology results for this group.

3-5 years

Four participants indicated opiate use 3-5 years prior to questionnaire completion. Results indicated drug use for 100% of the sample including a positive benzodiazepine (25%), cannabinoid (75%) and amphetamine (25%) toxicology results for this group.

5+ years

Seven participants indicated opiate use 5+ years prior to questionnaire completion. Results indicated drug use for 86% of the sample including a positive benzodiazepine (83%), cannabinoid (43%), opiate (14%) and amphetamine (14%) toxicology results for this group (see Figure 1.7)
Figure 1.7.

Timeframe of last opioid use and positive toxicology result

![Graph showing participants with positive toxicology over timeframes](image)

Figure 1.7. Benzo = benzodiazepine, Cannab = cannabinoid, Amphet = amphetamine

After combining groups in 1 day-1 year and 1 year-5+ year groups, two-way chi-square analyses revealed a not significant relationship between timeframe of last opioid use and benzodiazepine use \[ \chi^2 (1, N = 34) = 0.85, p > 0.05, \text{Cramer’s } V = .03 \] and cannabinoid use \[ \chi^2 (1, N = 34) = 0.20, p > 0.05, \text{Cramer’s } V = .22 \]. Frequencies are shown in Table 1.2.

All drug class use, opiate use and amphetamine use could not be analysed through chisquare analyses as the assumption of expected frequency in each category was violated.
Table 1.2
*Frequencies, percentages and adjusted standardised residuals for positive and negative toxicology results for last opioid use timeframe.*

<table>
<thead>
<tr>
<th></th>
<th>Positive toxicity result</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>Negative toxicity result</th>
<th></th>
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<th></th>
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<tbody>
<tr>
<td></td>
<td>f</td>
<td>%</td>
<td>f_e</td>
<td>ASR</td>
<td></td>
<td>f</td>
<td>%</td>
<td>f_e</td>
<td>ASR</td>
</tr>
<tr>
<td><strong>Benzodiazepine</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1day-1year</td>
<td>7</td>
<td>36.8%</td>
<td>7.3</td>
<td>-.2</td>
<td></td>
<td>12</td>
<td>62.3%</td>
<td>11.7</td>
<td>.2</td>
</tr>
<tr>
<td>1year-5+years</td>
<td>6</td>
<td>40.0%</td>
<td>5.7</td>
<td>.2</td>
<td></td>
<td>9</td>
<td>60%</td>
<td>9.3</td>
<td>.2</td>
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<tr>
<td><strong>Cannabinoids</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1day-1year</td>
<td>6</td>
<td>31.6%</td>
<td>7.8</td>
<td>-1.3</td>
<td></td>
<td>13</td>
<td>68.4%</td>
<td>11.2</td>
<td>1.3</td>
</tr>
<tr>
<td>1year-5+years</td>
<td>8</td>
<td>53.3%</td>
<td>6.2</td>
<td>1.3</td>
<td></td>
<td>7</td>
<td>46.7%</td>
<td>8.8</td>
<td>-1.3</td>
</tr>
</tbody>
</table>

*Note: f = frequency, % = percentage within type of gender, f_e = expected frequency, ASR = adjusted standardized residuals.*

5.1.7 *Lifetime Use.*

The mean life-time use of opioids was 17 years (SD = 11.24). Independent t-tests showed that amongst participants that had a positive toxicology result for all drug classes, a not significant difference between drug use and non-drug use groups was found, \(t(32) = -0.486, p < 0.05\). Life-time use was longer for all drug classes use group (\(M = 17.63, SD = 11.85\)) than the non-drug use group (\(M = 15.29, SD = 9.05\)) (see Figure 1.8).

**Figure 1.8.**

*Opiate lifetime use of all drug class use group and no-drug class use group*

*Figure 1.8. Pos = positive toxicology result, Neg = negative toxicology result*
Opiate life-time use indicated a not significant difference between opiate relapse and non-relapse groups, $t(32) = -.148, p < 0.05$. Life-time use was longer for relapse group ($M = 17.71, SD = 12.04$) than the non-relapse group ($M = 17.00, SD = 11.26$) (see Figure 1.9).

**Figure 1.9.**
*Mean opiate lifetime use of opiate use group and no-opiate use group*

![Figure 1.9](image1)

*Figure 1.9. Pos = positive toxicology result, Neg = negative toxicology result*

Opiate life-time use indicated no significant differences between benzodiazepine use and non-use groups, $t(32) = .152, p < 0.05$. Life-time use was shorter for relapse group ($M = 16.77, SD = 9.12$) than the non-relapse group ($M = 17.38, SD = 12.59$) (see Figure 1.10).

**Figure 1.10.**
*Mean opiate lifetime use of benzodiazepine use group and no-benzodiazepine use group*

![Figure 1.10](image2)

*Figure 1.10. Pos = positive toxicology result, Neg = negative toxicology result*
Opiate life-time use indicated a not significant difference between cannabinoid use and non-use groups, \( t(32) = -0.831, p < 0.05 \). Life-time use was longer for relapse group (\( M = 19.07, SD = 14.04 \)) than the non-relapse group (\( M = 15.80, SD = 8.95 \)) (see Figure 1.11).

**Figure 1.11.**
Mean opiate lifetime use of cannabinoid use group and no-cannabinoid use group

![Figure 1.11](pos-cannabinoid_neg-cannabinoid.png)

\( \text{Pos} = \text{positive toxicology result, Neg} = \text{negative toxicology result} \)

Opiate life-time use indicated an significant difference between amphetamine use and non-use groups, \( t(32) = -2.102, p = 0.043 \). Life-time use was longer for relapse group (\( M = 22.73, SD = 13.57 \)) than the non-relapse group (\( M = 14.48, SD = 9.105 \)) (see Figure 1.12).

**Figure 1.12.**
Mean opiate lifetime use of amphetamine use group and no-amphetamine use group

![Figure 1.12](pos-amphetamine_neg-amphetamine.png)

\( \text{*p < .05.} \)

\( \text{Pos} = \text{positive toxicology result, Neg} = \text{negative toxicology result} \)
5.2 Drug classes.

Seven participants tested positive (20.6% of participants) for opiate, 11 (32.4%) for amphetamine, 14 (41.2%) cannabinoid, 13 (38%) benzodiazepine. Twenty seven (79%) participants tested positive to either individual or combined drug classes (see Figure 2.1)

Figure 2.1. Participants with positive toxicology results

![Bar chart showing drug classes with positive results](image)

*Figure 2.1.* Benzo = benzodiazepine, Cannab = cannabinoid, Amphet = amphetamine

5.3 Stress.

A series of independent t-tests were conducted to compare differences between all drug class usage and stress scores, with assumptions of normality and homogeneity of variance met. Relapse levels as indicated by positive toxicology results for all drug classes and PSS scores found there was not a significant difference between relapse and non-relapse groups, $t(32) = .478, p < 0.05$. Mean stress scores was higher for relapse group ($M = 21.53, SD = 5.62$) than the non-relapse group ($M = 20.57, SD = 5.62$) (Figure 3.1.).
The individual t-test for PSS score and opiate use indicated a not significant difference between relapse and non-relapse groups, $t(32) = .24, p < 0.05$. Mean stress scores was lower for relapse group ($M = 20.57$, $SD = 8.50$) than the non-relapse group ($M = 21.33$, $SD = 7.41$) (Figure 3.2).

*Figure 3.2.* Positive = Positive toxicology result for opiate use. Neg = negative toxicology result.
A statistically significant difference between PSS scores was between amphetamine users and no-amphetamine group $t(32) = 2.52, p = 0.017$. Mean stress scores was lower for amphetamine group ($M = 23.26, SD = 6.88$) than the no-amphetamine group ($M = 16.82, SD = 7.17$) (Figure 3.3).

**Figure 3.3.**
Mean stress scores of amphetamine use group and no-amphetamine use group

![Figure 3.3](image)

*Figure 3.3. *$p < .05$. Positive = positive toxicology result for amphetamine. Neg = negative toxicology result.

No significant difference between PSS scores for cannabinoid users and no-cannabinoid group $t(32) = -1.85, p = 0.073$ was found. Mean stress scores was higher for cannabinoid group ($M = 23.93, SD = 2.26$) than the no-cannabinoid group ($M = 19.25, SD = 6.29$) (Figure 3.4.).
A not significant difference between PSS scores for benzodiazepine users and non-benzodiazepine group $t(32) = -0.49, p < 0.05$. Mean stress scores was higher for relapse group ($M = 22.00, SD = 1.13$) than the non-relapse group ($M = 20.67, SD = 1.98$) (Figure 3.5.).
5.4 LOC.

An independent t-test was conducted to compare differences between all drug class usage and LOC scores, with assumptions of normality and homogeneity of variance met. Relapse as indicated by positive toxicology results from any drug class and LOC scores, found a not significant difference between relapse and non-relapse groups, \( t(32) = 1.08, p > 0.05 \). Mean stress scores was higher for relapse group (\( M = 11.44, SD = 3.04 \)) than the non-relapse group (\( M = 10.71, SD = 1.11 \)) (see Figure 4.1).

![Figure 4.1](image_url)

Mean LOC score for all drug class use and no-drug class use

For individual drug classes, the t-test for LOC score and opiate use indicated a not significant difference between relapse and non-relapse groups, \( t(32) = -1.07, p > 0.05 \). Mean LOC scores was higher for the relapse group (\( M = 12.29, SD = 2.29 \)) than the non-relapse group (\( M = 11.04, SD = 2.85 \)) (see Figure 4.2).
Figure 4.2. Mean LOC score for opiate use and no-opiate use

![Diagram showing mean LOC scores for opiate use and no-opiate use](image)

Figure 4.2. Positive = positive toxicology result, Neg = negative toxicology result

Amphetamine use indicated a not significant difference between LOC, amphetamine and no-amphetamine groups, \( t(32) = -3.63, p > 0.05 \). Mean LOC scores was higher for amphetamine use group \( (M = 11.55, SD = 4.20) \) than the no-amphetamine group \( (M = 11.17, SD = 1.83) \) (see Figure 4.3).

Figure 4.3. Mean LOC score for amphetamine use and no-amphetamine use

![Diagram showing mean LOC scores for amphetamine use and no-amphetamine use](image)

Figure 4.3. Positive = positive toxicology result, Neg = negative toxicology result
Cannabinoi d use indicated a not significant difference between LOC, cannabinoid use and no-cannabinoid use groups, $t(32) = 8.69 \ p > 0.05$. Mean LOC scores was higher for cannabinoid group ($M = 12.00, SD = 2.94$) than the no-cannabinoid group ($M = 10.80, SD = 2.59$) (see Figure 4.4).

**Figure 4.4.** Mean LOC score for cannabinoid use and no-cannabinoid use

Benzodiazepine use indicated a not significant difference between LOC, benzodiazepine use and no-benzodiazepine groups, $t(32) = .730, p > 0.05$. Mean LOC scores was higher for the benzodiazepine group ($M = 11.38, SD = 3.09$) than the no-benzodiazepine group ($M = 11.24, SD = 2.61$) (see Figure 4.5).
Figure 4.5. Positive = positive toxicology result, Neg = negative toxicology result

5.5 IDTS.

Thirteen participants indicated they had used opioids within 12 months of partaking in the study and qualified to complete the IDTS.

Drug Use.

Average group scores for participants who tested positive to any drug class had a greater mean score for UE, PD, PE, TPC, UT, SP and PT than negative all drug class group (see Table 5.1). Independent t-tests did not indicate significant differences between all drug class use and the UE subscale $t(11) = -0.29, p > 0.05$, PD subscale $t(11) = -0.77, p > 0.05$, PE subscale $t(11) = -1.16, p > 0.05$, TPC subscale $t(11) = -1.34, p > 0.05$, UT subscale $t(11) = -1.02, p > 0.05$, CO subscale $t(11) = 0.06, p > 0.05$, SP subscale $t(11) = -0.75, p > 0.05$, or the PT subscale $t(11) = -1.32, p > 0.05$.

Average mean scores for the negative situations (NS) profile were greater for non-drug users, higher for positive situations (PS) profile for all drug class users and higher for temptation situations (TS) profile for non-drug users. However, independent t-tests did not
indicate significant difference between all drug class use and the NS profile $t(11) = .27, p > 0.05$, PS profile $t(11) = -1.64, p > 0.05$ and TS profile $t(11) = 1.31, p > 0.05$ (Table 5.2.).

Table 5.1
Mean and Standard Deviation scores for all drug class users and non-users for IDTS subscales

<table>
<thead>
<tr>
<th>Subscale</th>
<th>All Drug Use</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Positive</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td></td>
<td>$M (SD)$</td>
<td>$M (SD)$</td>
<td>$p$</td>
</tr>
<tr>
<td>UE</td>
<td>63.78 (19.56)</td>
<td>59.00 (40.64)</td>
<td>.29</td>
</tr>
<tr>
<td>PD</td>
<td>56.78 (13.34)</td>
<td>46.25 (37.97)</td>
<td>.77</td>
</tr>
<tr>
<td>PE</td>
<td>48.56 (25.39)</td>
<td>27.75 (38.97)</td>
<td>-1.16</td>
</tr>
<tr>
<td>TPC</td>
<td>47.89 (22.75)</td>
<td>26.50 (34.68)</td>
<td>-1.34</td>
</tr>
<tr>
<td>UT</td>
<td>56.00 (19.69)</td>
<td>42.75 (25.86)</td>
<td>-1.02</td>
</tr>
<tr>
<td>CO</td>
<td>43.00 (19.54)</td>
<td>43.75 (28.89)</td>
<td>.06</td>
</tr>
<tr>
<td>SP</td>
<td>59.11 (19.33)</td>
<td>49.75 (24.66)</td>
<td>-.75</td>
</tr>
<tr>
<td>PT</td>
<td>59.78 (15.62)</td>
<td>44.75 (25.79)</td>
<td>-1.32</td>
</tr>
</tbody>
</table>

Note: $M = \text{mean}, SD = \text{standard deviation}, UE = \text{unpleasant emotion}, PD = \text{physical discomfort}, PE = \text{pleasant emotion}, TPC = \text{testing personal control}, UT = \text{urges and temptations}, CO = \text{conflict with others}, SP = \text{social pressure}, PT = \text{pleasant time with others}, Positive = \text{positive toxicology result}, Negative = \text{negative toxicology result}.$

Table 5.2.
Mean and Standard Deviation scores for all drug class users and nonusers for subscale profiles

<table>
<thead>
<tr>
<th>Subscale</th>
<th>All Drug Use</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Positive</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td></td>
<td>$M (SD)$</td>
<td>$M (SD)$</td>
<td>$p$</td>
</tr>
<tr>
<td>NS</td>
<td>159.11 (75.55)</td>
<td>169.50 (6.56)</td>
<td>.79</td>
</tr>
<tr>
<td>PS</td>
<td>110.44 (43.23)</td>
<td>67.50 (44.67)</td>
<td>.13</td>
</tr>
<tr>
<td>TS</td>
<td>132.56 (50.53)</td>
<td>167.50 (18.95)</td>
<td>.21</td>
</tr>
</tbody>
</table>

Note: $M = \text{mean}, SD = \text{standard deviation}, NS = \text{negative situations}, PE = \text{pleasant situations}, TS = \text{temptation situations}, Positive = \text{positive toxicology result}, Negative = \text{negative toxicology result}.$
Opiate.

Average group scores for participants who tested positive to opiates had a greater mean score for UE, PE, TPC, UT and SP than negative-opiate group (see Table 5.3.). Independent t-tests did not indicate significant differences between opiate use and any of the UE subscale $t(11) = -.43, p > 0.05$, PD subscale $t(11) = .21, p > 0.05$, PE subscale $t(11) = -.22, p > 0.05$, TPC subscale $t(11) = -.81, p > 0.05$, UT subscale $t(11) = -.65, p > 0.05$, CO subscale $t(11) = .54, p > 0.05$, SP subscale $t(11) = -.13, p > 0.05$, or the PT subscale $t(11) = .27, p > 0.05$.

Average mean scores for the Negative situations (NS) profile were greater for non-drug users, higher for Positive situations (PS) profile and higher for temptation situations (TS) profile. However, independent t-tests did not indicate significant difference between opiate use and the NS profile $t(11) = .49, p > 0.05$, PS profile $t(11) = -.86, p > 0.05$ and TS profile $t(11) = 1.61, p > 0.05$ (Table 5.4.).

Cannabinoid.

Average group scores for participants who tested positive to cannabinoids had a greater mean score for UE, PD, TPC, UT and SP than negative-cannabis group (see Table 5.3.). Independent t-tests did not indicate significant differences between cannabinoid use and the UE subscale $t(11) = -.44, p > 0.05$, PD subscale $t(11) = -.12, p > 0.05$, PE subscale $t(11) = -.18, p > 0.05$, TPC subscale $t(11) = -.59, p > 0.05$, UT subscale $t(11) = -.82, p > 0.05$, CO subscale $t(11) = .13, p > 0.05$, SP subscale $t(11) = -1.23, p > 0.05$, or the PT subscale $t(11) = .26, p > 0.05$.

Average mean scores for the negative situations (NS) profile were greater for non-drug users, higher for positive situations (PS) profile and higher for temptation situations (TS) profile than cannabinoid users. However, independent t-tests did not indicate a significant
difference between cannabinoid use and the NS profile \( t(11) = 2.09, p > 0.05 \), PS profile \( t(11) = .73, p > 0.05 \) and TS profile \( t(11) = 2.01, p > 0.05 \) (Table 5.4).

**Table 5.3.**

*Mean and Standard Deviation scores for Opiate and Cannabinoid users and nonusers for IDTS subscales*

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Opiates</th>
<th></th>
<th></th>
<th>Cannabinoid</th>
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<tr>
<td></td>
<td>Positive</td>
<td>Negative</td>
<td></td>
<td>Positive</td>
<td>Negative</td>
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<tr>
<td>UE</td>
<td>66.40 (26.88)</td>
<td>59.75 (26.86)</td>
<td>.67</td>
<td>67.25 (11.79)</td>
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<tr>
<td>PD</td>
<td>51.80 (16.65)</td>
<td>54.63 (26.58)</td>
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<td>PE</td>
<td>42.40 (29.26)</td>
<td>42.00 (32.70)</td>
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<td>39.75 (33.09)</td>
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<td>TPC</td>
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<td>UT</td>
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<td>59.75 (23.98)</td>
<td>48.44 (20.97)</td>
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<td>CO</td>
<td>39.00 (22.45)</td>
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<td>SP</td>
<td>57.20 (26.17)</td>
<td>55.63 (18.17)</td>
<td>.90</td>
<td>66.50 (26.13)</td>
<td>51.67 (17.31)</td>
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<tr>
<td>PT</td>
<td>53.20 (16.33)</td>
<td>56.38 (22.28)</td>
<td>.79</td>
<td>53.25 (18.86)</td>
<td>56.00 (20.87)</td>
<td>.83</td>
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</table>

*Note: M = mean, SD = standard deviation, UE = unpleasant emotion, PD = physical discomfort, PE = pleasant emotion, TPC = testing personal control, UT = urges and temptations, CO = conflict with others, SP = social pressure, PT = pleasant time with others, Positive = positive toxicology result, Negative = negative toxicology result.*
Table 5.4.
Mean and Standard Deviation scores for Opiate and Cannabinoid users and nonusers for subscale profiles

<table>
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<tr>
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<td>Negative</td>
<td>Positive</td>
<td>Negative</td>
<td>Positive</td>
<td>Negative</td>
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<td>Profile</td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>p</td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>p</td>
</tr>
<tr>
<td>NS</td>
<td>148.00 (103.86)</td>
<td>171.25 (13.54)</td>
<td>.64</td>
<td>114.75 (81.95)</td>
<td>183.44 (40.13)</td>
<td>.06</td>
</tr>
<tr>
<td>PS</td>
<td>111.40 (56.61)</td>
<td>88.38 (40.71)</td>
<td>.41</td>
<td>82.75 (43.69)</td>
<td>103.67 (48.87)</td>
<td>.47</td>
</tr>
<tr>
<td>TS</td>
<td>119.20 (59.87)</td>
<td>158.38 (28.85)</td>
<td>.18</td>
<td>109.25 (66.09)</td>
<td>158.44 (25.20)</td>
<td>.07</td>
</tr>
</tbody>
</table>

Note: M = mean, SD = standard deviation, NS = negative situations, PE = pleasant situations, TS = temptation situations, Positive = positive toxicology result, Negative = negative toxicology result.

Amphetamine.

Average group scores for participants who tested positive to amphetamine had a greater mean score for UE, PD, PE, TPC, UT, CO than negative-amphetamine group (see Table 5.5). Independent t-tests did not indicate significant differences between amphetamine use and the UE subscale t(11) = -.22, p > 0.05, PD subscale t(11) = -.97, p > 0.05, PE subscale t(11) = -1.47, p > 0.05, TPC subscale t(11) = -.11, p > 0.05, UT subscale t(11) = -.65, p > 0.05, CO subscale t(11) = -.67, p > 0.05, SP subscale t(11) = .06, p > 0.05, or the PT subscale t(11) = -2.09, p > 0.05.

Average mean scores for the negative situations (NS) profile were greater for amphetamine users, higher for positive situations (PS) profile and marginally higher for temptation situations (TS) profile than non-amphetamine users. However, independent t-tests did not indicate significant difference between amphetamine use and the NS profile t(11) = -.57, p > 0.05, PS profile t(11) = -.95, p > 0.05 and TS profile t(11) = .58, p > 0.05 (Table 5.6).
Benzodiazepines.

Average group scores for participants who tested positive to opiates had a greater mean score for PE, TPC PT than negative-benzodiazepine group (see Table 5.5.). Independent t-tests did not indicate significant differences between benzodiazepine use and the UE subscale $t(11) = 1.03, p > 0.05$, PD subscale $t(11) = .05, p > 0.05$, PE subscale $t(11) = -.57, p > 0.05$, TPC subscale $t(11) = -.70, p > 0.05$, UT subscale $t(11) = .62, p > 0.05$, CO subscale $t(11) = .37, p > 0.05$, SP subscale $t(11) = .54, p > 0.05$, or the PT subscale $t(11) = -.74, p > 0.05$.

Average mean scores for the negative situations (NS) profile were greater for benzodiazepine users, higher for positive situations (PS) profile and marginally higher for temptation situations (TS) profile than non-benzodiazepine users. However, independent t-tests did not indicate significant difference between benzodiazepine use and the NS profile $t(11) = -.80, p > 0.05$, PS profile $t(11) = -1.56, p > 0.05$ and TS profile $t(11) = -.04, p > 0.05$ (Table 5.6).
Table 5.5.
Mean and Standard Deviation scores for Amphetamine and Benzodiazepine users and nonusers for IDTS subscales

| Subscale | Amphetamine | | | Benzodiazepine | | |
|----------|-------------|-------------|---------------|-------------|-------------|---------------|-------------|
|          | Positive    | Negative    | p             | Positive    | Negative    | p             |              |
| UE       | 64.40 (20.96) | 61.00 (29.96) | .83           | 51.25 (14.77) | 67.22 (28.97) | .33           |
| PD       | 61.20 (14.39) | 48.75 (26.05) | .35           | 53.00 (12.06) | 53.78 (26.50) | .96           |
| PE       | 57.00 (21.66) | 32.88 (32.17) | .17           | 49.50 (13.77) | 38.89 (35.41) | .58           |
| TPC      | 42.40 (21.36) | 40.63 (32.03) | .92           | 49.50 (16.82) | 37.67 (31.18) | .50           |
| UT       | 57.00 (21.66) | 48.75 (22.39) | .53           | 46.25 (11.03) | 54.44 (25.11) | .55           |
| CO       | 48.40 (14.67) | 40.00 (25.30) | .52           | 39.75 (18.12) | 44.78 (23.73) | .72           |
| SP       | 55.80 (14.65) | 56.50 (24.48) | .96           | 51.50 (12.77) | 58.33 (23.55) | .60           |
| PT       | 67.80 (13.81) | 47.25 (18.93) | .06           | 61.25 (9.95)  | 52.44 (22.53) | .35           |

Note: M = Mean, SD = Standard deviation, UE = unpleasant emotion, PD = Physical discomfort, PE = Pleasant emotion, TPC = Testing personal control, UT = Urges and temptations, CO = Conflict with others, SP = Social pressure, PT = Pleasant time with others. Positive = Positive toxicology result, Negative = Negative toxicology result.

Table 5.6.
Mean and Standard Deviation scores for Amphetamine and Benzodiazepine users and nonusers for subscale profiles

| Profile | Amphetamine | | | Benzodiazepine | | |
|---------|-------------|-------------|---------------|-------------|-------------|---------------|-------------|
|          | Positive    | Negative    | p             | Positive    | Negative    | p             |              |
|          | M (SD)      | M (SD)      |               | M (SD)      | M (SD)      |               |              |
| NS       | 175.20 (40.63) | 154.25 (73.81) | .57           | 183.25 (54.08) | 153.00 (65.94) | .44           |
| PS       | 112.80 (32.49) | 87.50 (53.26) | .36           | 125.75 (30.36) | 84.56 (48.20)  | .15           |
| TS       | 144.60 (31.09) | 142.50 (54.78) | .94           | 144.00 (28.14) | 143.00 (53.04) | .97           |

Note: M = Mean, SD = Standard deviation, NS = Negative situations, PE = Pleasant situations, TS = Temptation situations, Positive = Positive toxicology result, Negative = Negative toxicology result.
Figure 5.1.
Mean subscale profile scores for all drug classes, opiate, cannabinoid, amphetamine and benzodiazepine toxicology results

Note: NS = negative situations, PE = pleasant situations, TS = temptation situations, Pos = positive toxicology result, Neg = negative toxicology result, Druguse = all drug classes, Cannab = cannabinoids, Amphet = amphetamine, Benzo = benzodiazepine.

5.6 Logistic regression.

Only variables in the previous result sections one, two, three, four and five that had indicated a significant difference in mean scores were selected for regression analyses. These variants include PSS mean scores and opiate-lifetime use for positive amphetamine toxicology results.

Logistic regression analysis was conducted to determine whether PSS and opiate lifetime use scores would predict amphetamines use or not. A test of the full model against a constant only model was statistically significant, indicating that predictors as a set reliably
distinguished between amphetamine users and amphetamine non-users ($X^2 (8) = 9.771, p < 0.05$).

Prediction success overall was 67.6% (73.9% for amphetamine non-users and 54.5% for amphetamine users). PSS added significantly to the predictor ($p = 0.35$), while opiate lifetime use did not ($p = 0.74$). ExpB value highlights that when the PSS score is raised by one unit, the odds ratio is 0.86 times as large and therefore individuals are 0.86 times less likely to use amphetamines.

6.0 Discussion

Hypothesis One: Demographic

Due to the small sample size, chi square analyses for all variables within the demographic category could not be completed. For the chi square analyses completed (gender and cannabinoid use, gender and benzodiazepine use, timeframe of last opioid use and cannabinoid use, timeframe of last opioid use and benzodiazepine) all identified no significant results for each demographic variant. Although hypothesis one was not supported and the null hypothesis retained, the trends of the mean scores for each demographic variant are explored below.

Although not significant, the trend from drug class usage shows that cannabinoids were the main drug consumed, followed by benzodiazepine, amphetamines and opiates last. Despite literature indicating the rapid increase in individuals using cocaine since 2004 (AIHW, 2016), no participants in the present study tested positive for cocaine toxicology. As cocaine has been found to be highest amongst individuals who are employed with high socioeconomic status (AIHW, 2016), this justifies the lack of positive toxicology results in the present study, as the demographic variables of participants indicated that the majority was unemployed with suspected low economic status.
The significance of cannabinoid usage amongst participants is concurrent with research identifying the most commonly used drug in Australia is cannabis with 9.8% of Australian reporting cannabis use on at least five occasions within the past 12 months (Teesson et al., 2006). Increased cannabis use within the Australian populations may be influenced by current societal perception of cannabis in comparison with previous years. For example, the National Drug Strategy Household Survey conducted in 2016 has found that a great portion of individuals support legalisation and a lower proportion of the Australian community support penalties for sale and supply. Additionally, more people (85%) are supporting the use of cannabis in clinical trials in comparison to 2013 (75%). Further, cannabis was found to be the mostly commonly used substance in combination with other illicit substances in the previous 12 months. Interestingly, individuals who were listed as NMPR users were most likely to only co-use opioids and cannabis within the same 12 month period (AIHW, 2016).

As research has noted that opioids are commonly abused in combination with benzodiazepine substances (Darke & Hall, 1995; Iguchi et al., 1988; Jones et al., 2012), the finding of benzodiazepines being the second most consumed substance in the present study adds to this literature. Although benzodiazepines are typically prescribed for the treatment of anxiety disorders, insomnia (Schuman-Olivier et al., 2013) and other psychiatric and neurological illnesses, benzodiazepines are typically abused non-medically for the purpose of becoming intoxicated, and co-used with opioids in order to exemplify the u agonist effects of opioids (Jones et al., 2012). This prevalence of benzodiazepine use within methadone and buprenorphine maintenance programs is estimated to range between 51% to 70% (Jones et al., 2012). Literature states that between 2004 and 2011, there was a significant rise (11.0 to 34.2 per 100,000 population) of NMPR related emergency department visits due to the combined use of opioids and benzodiazepines and an 18% increase of opioid deaths due to benzodiazepine co-use (Jones, & McAninch, 2015). Although the present study only found
that 38% tested positive for benzodiazepine, the lowered score in comparison to contemporary literature findings may have been influenced by the small sample size.

The co-use of amphetamine and opioids identified is not surprising as the rise in amphetamine use is prominent in Australia, as daily and weekly use of amphetamines, including meth-amphetamines, has increased from 9.3% in 2010 to 20% in 2016. Specifically, amphetamine users detailing “ice” as their main amphetamine substance increased from 22% in 2013 to 57% in 2016. This also resulted in “ice” users indicating a greater increase in daily or weekly use from 12.4% in 2010 to 32% in 2016 (AIHW, 2016).

The gender trend highlighted that males indicated greater use of all drug classes, benzodiazepines, opiates and amphetamines than females. This result is consistent with current literature indicating that males are at a greater risk of developing substance use disorders (Gibson et al., 2015) as they typically report greater use of a variety of substances than their female counterparts.

The age group that indicated greater overall drug use, benzodiazepine use and opiate use was the 25-39 year bracket. However, this age group also had the greater amount of participants (n = 17). Interestingly, the age group of 50+ years, although only consisting of five participants, indicated greater use for cannabinoids and amphetamines. When the two older population groups are combined (n = 16) to produce a 40-50+ years age bracket, this group indicates greater drug use of all classes, cannabis and amphetamine than the younger population groups. This appears consistent with current results showing that continued amphetamine use is more prominent in older age groups than the younger demographic (AIHW, 2016). Additionally, as the Australian population is ageing and the baby boomer populace has been found to more likely consume alcohol and illicit substances than the younger generation (Johnco, Whithall & Draper, 2015), this factor can explain why the age
group trend identified in the present Australian sample does not adhere to concurrent literature findings that younger populations exhibit higher substance use behaviour.

The trend of unemployment status indicated heightened use of all drug classes combined, opiate use, cannabinoid use, benzodiazepine use and amphetamine use in comparison to the casual and self-employed groups. This finding is consistent with current literature indicating that individuals who are unemployed are significantly associated with increased susceptibility of developing substance use disorders as well as engaging in polydrug use (Darke & Hall, 1995)

However, based on these trends explored, a type II error may have occurred for independent t-test and chi square analyses due to the small sample size. A more powerful test with greater participant samples would need to be conducted to confirm the occurrence of a type II error in this participant sample.

Hypothesis two: Stress

Although the majority of the results also highlighted not significant differences between stress scores and substance use, indicating that the null hypothesis was retained, the average stress score trends highlight that greater stress levels were evident for those who used all drug classes, benzodiazepines and cannabinoids. These trends give support to the ideology that stress is a predictor of continued substance use within the OST program.

Contrastingly, the significant difference between the mean score of stress and amphetamine use does not support the hypothesis that heightened stress, resulting in negative affect, predicts drug use. However, an insight into this relationship may be derived from the IDTS subscale results as amphetamine users detailed higher mean scores for the pleasant emotions subscale and positive situations profile than non-amphetamine users. The present
data does not allow for further analysis so we cannot infer the reasons as to why lower stress scores were significantly associated with greater amphetamine use.

**Hypothesis three: LOC**

All drug use classes collectively and independently indicated the trend that drug use groups had higher LOC mean scores than non-users. However, the null hypothesis was also retained, as significance was not achieved for these relationships. The trend identified in the present study is consistent with literature noting that substance dependent individuals yield greater LOC scores than abstinent individuals (Ersche et al., 2012) and non-users within a methadone program (Kao et al., 2014).

**Hypothesis four: IDTS profile scores**

The null hypothesis was also retained for hypothesis four as significance was not achieved for mean differences for IDTS subscales, profiles and substance use. Mean scores for the IDTS subscales UE, PD, PE, TPC, UT, SP and PT were higher for user groups than non-user groups. However, due to the low number of participants completing the IDTS (13 participants), it is difficult to identify further key trends from this data for individual drug class groups as individuals who completed the scale had to have previously used opioids within the past year, and answered the questions based on prior opioid consumption.

Interestingly, the data from the NS, PS and TS profiles for overall drug use, opiates and cannabinoids do not appear to support the hypothesis that substance use is sought for to eliminate negative contexts or emotions. Rather, the NS profile for amphetamine and benzodiazepine use highlight that the user groups would use these two drug classes within NS contexts. The results from these two substance user groups support the notion depicted by the
NDSHS whereby the increase of continued illicit drug use was due to attempts to “improve mood” or “stop feeling unhappy” (AIHW, 2016).

Collectively, the IDTS subscale and profiles still provide important information for clinicians and future researchers to consider when developing new treatment plans as it appears that alternate high-risk situations can impact individuals differently, and alternate substances may be utilised more frequently within specific subscales or profiles than other substances.

**Hypothesis five: Predictive model**

As the null hypotheses for hypothesis one, two, three and four were retained, the null hypothesis for hypothesis five was also retained as a significant predictive model of demographic, stress, LOC and IDTS profiles could not be established.

**6.1 Implications and Future Research**

Implications of the present study highlight the importance of continuing research in the field of SUDs within an Australian demographic, to assist in providing current data on continued substance use within OST programs to healthcare providers and government bodies. This information may be pivotal in influencing policy changes and the development of new treatment modalities. Although results did not display significant effects, trends indicate that within the small population sample, stress may be associated with increased engagement in drug seeking behaviour within a maintenance treatment program. Additionally, results indicated that participants with higher LOC scores engage in greater drug use than those who did not, highlighting the importance of accountability and behaviour control beliefs in the first and early stages of recovery. Future research should expand the present study to a
A state wide and national wide project to identify Australian trends and key predictor factors of continued substance use within OST programs.

Further, these trends indicate that the materials used for the present study (PSS-10, LOC and IDTS) can be adopted within treatment services to screen for potential relapse factors that the individual and professional body can target by developing a multivariable prevention plan. For example, alcohol dependent individuals who shifted from an external LOC to an internal LOC throughout treatment, elicited greater treatment outcomes than individuals who maintained an external LOC orientation (Ersche et al., 2012). Therefore, the LOC scale could be used as a quantitative measure for treatment progress. Additionally, it could be advised that rehabilitation programs cater toward an individual’s LOC orientation to improve treatment outcomes as conflict between an individual’s control belief (LOC) and treatment approach has a significantly lower success probability (Fernandes & Mokwena, 2016).

Although the results found that older populations engaged in overall greater drug use, the results still indicate continued substance use for younger age populations within treatment services. As substance use within younger populations remains a public health concern, the results support the notion that early intervention strategies should be developed and strengthened, targeting young individuals who are at high-risk for developing substance use disorders. For example, early prevention strategies and support groups that detail the importance of obtaining a higher school education, accessing counseling services, appropriate stress management styles and effective communication, as well as access to other health services may provide additional support to those who are particularly vulnerable in developing substance use disorders. It is also suggested that preventative programs should be instituted prior to the higher grades of high school to reduce substance use within younger demographics (Quek et al., 2013).
This study also poses implications for current council bodies to identify the impact of environmental protective factors on diminishing drug related activity within disadvantaged communities. For example, access to areas for recreation or spiritual purposes and social support can aid those within disadvantaged communities as engagement in these areas may act as stress-buffering mechanisms (Gibson et al., 2015). Government bodies should also continue reducing environmentally produced and mediated stressors by improving living conditions, creating realistic employment opportunities and diminishing the detrimental impact of poverty (Jackson et al., 2010). Unless environmental stressors are attended to, the cycle of engaging in unhealthy coping behaviours to deplete psychological stress for individuals living within disadvantaged communities will likely continue to occur.

Implications of the mean trends identified also call for focus on providing multimodal interventions for disadvantaged, vulnerable populations, extending to rural and regional areas (Degenhardt et al., 2016) as well as prison settings. Within prison populations, rates of drug use amongst adult male prisoners exceed the general public and reports have found that more than half of state and federal prisoners in the United States are drug dependent (NIDA, 2012). A smaller study also found that within a sample of incoming prisoners, half met the criteria for a SUD (Strang et al., 2006). Reports have also found that former inmates are especially vulnerable to drug overdoses within a fortnight post release from prison (Rowell et al., 2012).

It is argued that if an addicted person has committed a drug related crime, imprisonment without treatment is counterproductive. Without treatment, re-offending once released in drug related activity will continue to be significantly linked (Leshner, 1997). Therefore, as prisons may be the first opportunity for disadvantaged individuals to receive a psychiatric diagnosis and appropriate treatment for SUDs (Rowell et al., 2012), this may result in significant health outcomes by reducing drug related crime and drug use that ultimately achieves significant savings in direct and indirect societal costs (NIDA, 2012).
Within these disadvantaged communities, interventions need to acknowledge that for populations living under chronically stressful conditions, engaging in unhealthy behaviours have an adaptive, neurological effect that alleviate immediate negative psychological and physiological states whereby the short-term benefits outweigh the risk of long-term physical health and mortality (Jackson et al., 2010). This further strengthens the importance of establishing new procedures and policies that target reducing stressors and overall substance use within vulnerable populations across Australia.

In regards to the dangers of polydrug use, particularly the popularity of opioid and benzodiazepine co-use, the present study’s findings highlight that within a singular OST program, healthcare providers and services need to continue working closely with clients in preventing the potentially lethal combination of benzodiazepines and medically-assisted opioids. Risk information should be provided to those who receive benzodiazepines through prescription, as patients who were receiving buprenorphine for opioid substance use disorder and had a benzodiazepine prescription have been found to be more than at three-fold increase of experiencing accidental injury, increased emergency department visits for accidental injury and other medical causes in comparison to control subjects without benzodiazepine prescription (Schuman-Olivier et al., 2013). For individuals who are receiving treatment for anxiety disorders, it is recommended that selective-serotonin reuptake inhibitors and serotonin-norepinephrine reuptake inhibitors be prescribed instead and that benzodiazepine should only be consumed short-term and only when necessary (Schuman-Oliver et al., 2013).

As continued illicit substance use and polydrug use within OST programs are a continuing issue, rather than adhere to punitive measures of termination of treatment or administrative discharge, in line with earlier research, health organisations should adopt alternative strategies, such as contingency management, to promote remaining abstinent from illicit substances while continuing medical-assisted treatment programs (Iguchi et al., 1998). Drug
services should also provide information to polydrug users of the detrimental effects of drug combinations including diminished general health, overdose lethality and lowered treatment outcome (Jones et al., 2012).

In tackling opioid dependency due to restoring pain relief, the implications of the current study highlight the importance for treatment providers to aim at providing alternate resources so clients can learn how to cope with the pain without having to suffer, with or without the prescription of opioids (Ling, 2017) or other illicit substances. Other evidence-based interventions currently available for pain relief and for treating somatoform pain disorder include cognitive behavioural therapy (CBT) and acceptance and commitment therapy (ACT) (APS, 2010).

The salient implication of the present study indicates that future research should further continue identifying individual vulnerabilities in developing and maintaining substance use disorders. Additionally, different treatments for different vulnerability profiles should be explored as substance use disorder should not be clinically treated as a population as a whole, rather treatment should identify vulnerabilities triggered within the individual and treatment should be addressed to the specific combination of vulnerabilities presented by each client (Redish et al., 2008). Due to the complex biological, psychological and social aspects of addiction, pharmacotherapy treatment for substance use disorders should be combined with appropriate psychosocial treatments depending on the individual’s needs (Kosten & George, 2002). Although a costly approach, a collaborative approach to facilitate long-term behavioural change may be required to effectively treat opioid dependency (Lobmaier et al., 2010). The addition of psychosocial treatments including individual, group and family counselling, psychotherapy, psychoeducation, behavioral contingency interventions and expressive art therapies, have been found to play an important role in improving the efficacy of medically-assisted treatment programs for opioid dependency (Pecoraro et al., 2012).
In a recent study, 240 opiate dependent participants were divided into either Cognitive Behavioural Therapy (CBT)+MMT or MMT alone conditions (Pan et al., 2015). The results highlighted that the CBT+MMT condition showed greater opiate-negative urine tests collected during the 12th and 26th weeks. Therefore, the study concluded that employing CBT into the MMT programs was effective in further reducing opiate use and relapse (Pan et al., 2015).

Programs tailored to assist with added stressors have resulted in increased treatment gains when these psychosocial issues are attended to as well as substance abuse issues (Broome et al., 2012). Supplemental social support services involving trained clinical case managers to assist patients with education, employment, housing, recreation and parenting issues was implemented in extension to an outpatient public sector program. Results found that enhanced program participants produced 25-40% better treatment outcomes than control groups (McLellan et al., 1998).

As a recognized program, the Safeport three-phase residential treatment program catered for assisting the needs of women with substance use disorders, living in public housing. The program developed a facility including seven public housing units, transforming them into one, two and three bedroom units with shared living quarters. Onsite childcare centre, recreational areas and counselling offices were also created. The first phase of the program involved abstinence and stabilisation where the patient and family developed skills to live a drug-free lifestyle, including therapeutic interventions for all family members. Phase two focused on building self-sufficiency skills whereby employment skills were emphasised. Phase three prepared patients for a drug-free life where they continued attending counselling, relapse prevention sessions and could seek employment outside the facility. The additional support services of Safeport resulted in a 62% completion rate of the program in comparison of 42% for other outpatient rehabilitation programs (Alexandre et al., 2003).
An office-based opioid treatment (OBOT) program in primary care that operated at Boston Medical Center in 2003, treated 408 patients across a five year period with buprenorphine (Alford et al., 2011). The OBOT program used a collaborative care model that included a full-time nurse program director, nurse care managers, a program coordinator and generalist physicians. The program consisted of three stages: assessment by physicians and nurse care managers to determine appropriateness for the program, supervised induction and stabilisation by nurse care managers including buprenorphine dose adjustments and maintenance. The maintenance stage included buprenorphine treatment with illicit drug use screening and weekly counselling as well as monitoring involuntary and voluntary discharge criteria.

At 12 month follow up, 196 (51.3%) patients showcased a successful outcome of either treatment retention or buprenorphine taper after treatment adherence and absence of illicit drug use for at least six months. However, 162 (42.4%) patients demonstrated unsuccessful outcomes due to either being discharged from the study or not returning to follow up appointments. Alford et al. (2011) identified key characteristics associated with successful outcomes in comparison to unsuccessful outcomes. The results indicated through bivariate analyses characteristics of being female, white race, being employed, self-maintaining with illicit buprenorphine, prescription opioid abuse or methadone maintenance and no self-report of past year cocaine use were significantly associated with treatment success. Although a successful treatment program, unfortunately the main barrier to OBOT program offerings was the lack of clinical support needed for the continuing monitoring of patients (Alford et al., 2011).

Financial challenges and public funding constraints also make it difficult for addiction treatment programs to recruit and retain clinicians as staff turnover in the United States for substance dependency treatment centres are estimated between 18.5% and 49% (Pecoraro et
Publicly funded treatment programs are influenced by declining public reimbursement rates, which links to providing strong services and profiting less, or providing lower quality services with lower quality clinicians investing fewer resources, treating more patients and cutting costs to create a greater profit (Pecoraro et al., 2012). Additionally, public treatment providers are generally limited in treatment options, have longer waiting times, have lower staff quality, are less likely to offer specialised treatment and have delays in the adoption of technological advances to improve treatment efficacy (Cantor, Stoller & Saloner, 2016; Edmond, Aletraris & Roman, 2015).

In New South Wales, Australia, the majority of opioid treatment services and other substance use treatment programs are based in public hospitals, which can come at the expense of allied health and community care resources. Residential rehabilitation programs are mostly no-for-profit non government organisations that receive government funding but many also charge client fees to cover unfunded costs. Privately funded hospitals also provide addiction services (Haber & Day, 2014), where the client is expected to pay for the service independently. The advantage of private clinics includes lower client to staff ratios allowing for more individualised treatment programs (O’Grady et al., 2014), however access to private services are limited for substance dependent persons as they typically have low socioeconomic status.

For example, there are distinct demographic characteristics found for individuals who seek public versus private treatment for substance use disorders. Trends identified in the United States found individuals who accessed public treatment services reported lower incomes, low education level, no medical insurance coverage and were more likely to identify as Latino or African-American than their private treatment counterparts (O’Grady et al., 2014). Therefore, treatment providers within rural and urban populations are more likely to be
non-profit and publicly funded as higher poverty levels in these disadvantaged communities are financially problematic for for-profit or privately funded centres (Edmond et al., 2015).

Health expenditure on alcohol and drug treatments between 2012-2013 in Australia was estimated to cost $1,213 million, where state and territory governments accounted for 50.7% of this expenditure funding services in public hospitals and other substance treatment programs (Chalmers et al., 2015). Although resources are invested annually into public substance dependency treatment services, there is not a lot of research conducted on the economic merits of these various services (Alexandre et al., 2003). Future research should evaluate economic costs of available treatment services within the public sector to assist state governments and the Commonwealth in allocating limited public funding to the more effective and efficient addiction treatment facilities (French, Popovici, & Tapsell, 2008). This research should further extend to exploring the economic worth of funding the delivery of public services providing high quality, long-term specialised treatment services within disadvantaged communities such as rural areas and incarcerated populations. Ultimately, additional funding to healthcare systems catering for substance dependent clients may result in decreased morbidity, mortality and incremental costs to the government due to continued substance use, repeated detoxification and cyclic readmissions to acute settings.

Lastly, alternate factors for developing and maintaining substance use disorder not explored in this study include alternate individual factors such as personality and genetics. Personality traits including sensation seeking, impulsivity, risk-taking behaviours, tolerance of deviance and rebelliousness have been linked with developing a substance use disorder (Eitan et al., 2017). In regards to genetics, currently it is known that the mu opioid receptor gene *OPRM1* plays a central role in opioid dependency and tolerance (Nielsen & Kreek, 2012). Additionally, cannabinoid receptor type 1 (*CNR1*) is found within various brain regions, including areas responsible for drug reward. Proudnikov et al. (2010) genotyped six
polymorphisms of \textit{CNR1} and found that the genotype of 1359GG was significantly associated with heroin dependency vulnerability, while the genotype of 1359A that is also located on the \textit{CNR1} gene was associated with protection from developing substance use disorders.

Genetics research has also identified several other genes associated with heroin dependency including delta and kappa opioid receptors (OPRD1 and OPRK1), dopamine receptors D2 and D4, serotonin receptors (HTR1B), serotonin transporter (SLC6A4), gamma-aminobutyric acid receptor (GABRG2), catechol-O-methyltransferase (COMT), period circadian protein (PER3), proopiomelanocortin (POMC), proenkaphalin (PENK), tryptophan hydroxylase (TPH1 and TPH2), brain-deprived neurotrophic factor (BDNF) and melanocortin receptor type 2 (MC2R) (Nielsen & Kreek, 2012). Variants in the stress related gene FKBP5 have also been found to increase vulnerability to heroin relapse as results showed increased sensitivity to both stress and heroin conditions (Levran et al. 2014). As a whole, expanding on genetics research will allow treatment providers to screen and identify predisposing gene variants associated with opioid dependence and implement this vulnerability factor into treatment development.

6.2 Limitations

The main limitation of the present study is the lack of significance due to the small sample size. An increase in participant numbers would have resulted in the key trends identified as significant findings through the chi square, t-test and logistic regression analyses. As the initial aim of the study was to develop a predictive model of continued drug use behaviour within a medically assisted maintenance program, the lack of participants recruited and maintained for the final analyses did not facilitate this. The only significant predictor for the model was a lower mean PSS score for amphetamine use for individuals which resulted in the null hypothesis being retained.
Secondly, the timing of questionnaire completion and medication administration was not consistent for all participants. As individuals were approached in the waiting room, they let the principal researcher know if they wanted to complete the surveys before or after their medication administration numbers. On days where there was a long waiting time, participants typically completed the surveys before administration. Contrastingly, for shorter waiting periods, participants requested to wait until after administration to avoid missing their turn and needing to wait for another number. As methadone has numerous side effects on the body including increased daytime tiredness, attention and memory problems (Dursteler-MacFarland et al., 2010), those who completed the surveys afterward may have performed worse than those beforehand. Therefore, future research should take this into consideration or keep this consistent in case this variable had a significant impact on result outcomes.

Another limitation encountered due to methodology design was that further analysis of urine toxicology results could not be completed as this was outside of the scope of routine drug testing conducted at RPAH. Additional resources would have needed to be recruited and approved through the ethics application. Time constraints also prohibited the further analysis to identify specified substances that resulted in the positive toxicology results. Therefore, the results of the current study can only be labeled for the drug-classes as it is not known whether positive results were due to consumption of specific substances under the umbrella of each drug class.

Additionally, the results for the individual drug classes should be considered with less weight as numerous routinely prescribed and nonprescription medications have been found to trigger false positive urine drug screening results. Medication including brompheniramine, bupropion, chlorpromazine, clomipramine, dextromethorphan, diphenhydramine, doxylamine, ibuprofen, naproxen, promethazine, quetiapine, quinolones, ranitidine, sertraline, thioridazine, trazodone, venlafaxine, verapamil, and a nonprescription nasal inhaler were found to trigger
false-positive results for amphetamines the most, followed by opioids, barbiturates, cannabinoids and benzodiazepine (Brahm et al., 2010). If the present study were to be replicated, it would be advised that additional analytic methods (gas chromatography-mass spectrometry) be considered to rule out false-positive results.

Furthermore, the current study did not screen for comorbid psychopathology. Comorbidity refers to co-occurrence of a substance use disorder with one or more mental health disorders. Amongst substance dependent individuals receiving treatment, there is a high prevalence of comorbid mental health disorders range between 47-100% (Kingston, Marel & Mills, 2016). In Australia, the most common psychiatric disorders including mood, depression, anxiety and personality disorders (Dore, 2015; Zhuang, An & Zhao, 2014). Increased psychotic symptoms have also been noted in patients presenting with methamphetamine use. The development of an SUD may occur due to self-medication for symptom relief of an alternate mental disorder, or the symptoms of mental illness are a direct consequence of excessive substance use (Dore, 2015; Kingston et al., 2016). Regardless of the comorbidity nature, mental disorders may interfere and produce significant challenges for the client and the professional body. Therefore, treatment facilities should assess for and adapt rehabilitation programs in light of the occurrence of comorbidity presentations.

Other psychiatric problems such as conduct disorder have previously been found to be key risk factors for substance use in adolescence (Cerda et al., 2016). Research highlighted that in a sample of 503 adolescents aged between 13-19 years, those who experienced a 1-unit increase in their conduct problems scores, their marijuana use and alcohol quantity consumption scores also increased by approximately one unit. Interestingly, when increased quantity of alcohol use was evident, anxiety problems scores increased, highlighting a reserve association between substance use and anxiety symptoms (Cerda et al., 2016). Additionally, in a study of 769 individuals with a substance use disorder, antisocial personality disorder and
a lower socioeconomic status were significantly associated with early mortality (Cornelius et al., 2008).

Lastly, as data collection was based on qualitative self-report measures, participants may have responded with heightened, exaggerated or falsified responses. Self-report responses may also be affected by the social desirability effect whereby participants provide “acceptable” rather than true responses (De Vaus, 2002). If the study was to be replicated on a greater scale, it is recommended that a validity scale be included to identify results that may distort the data.

In regards to the IDTS, individuals completed the survey based on previous opioid use within the specific subscale contexts. As the present study could only evaluate the drug use classes based on prior opiate consumption, we cannot be confident in the contexts that individuals may use alternate substances within alternate contexts. In future research, individuals should complete an IDTS subscale for every substance they have used within the past 12 months so these scores can be compared to the positive toxicology results for each drug class.

6.3 Conclusion

Collectively, the trends identified in the present study add to current literature indicating that substance use is shaped by multiple risk factors working together (Cerda et al., 2016). Although no significant results supporting the hypotheses were found, future research should replicate the current project on a state wide and national wide scale to further identify key vulnerabilities for continued substance use within OST treatment facilities in Australia.

As current public treatment organisations do not have the appropriate resources to attend to individual needs of substance dependent clients, the prominent implication of the present study highlight the need for government bodies within the public sector to investigate the
economic value of funding the development and maintenance of long-term multimodal treatment facilities for SUDs. These facilities will need to be derived from a continuing care model, whereby continuous monitoring and coordination between alternate services are maintained long-term. These approaches have previously been found to be more cost-effective than acute treatment episodes in the United States, with positive abstinent outcomes up to nine years later (Manning et al., 2017).

To conclude, the results of the present study contribute to research that aims to shift the management of a chronic relapsing condition using conventional OST, into a new paradigm involving alternate treatment modalities as current research highlights that no single pharmacological approach to the treatment of SUDs can be completely effective (Stewart, 2003).
7.0 References


model in opioid-dependent patients participating in the methadone maintenance
treatment in Iran. *Iranian Journal of Public Health, 42*(8), 896-902.

Pecoraro, A., Ma, M., & Woody, G. E. (2012). The science and practice of medication-
assisted treatments for opioid dependence. *Substance Use & Misuse, 47*, 1026-1040.


episodes among adolescents: A longitudinal investigation. *Journal of Substance Abuse
Treatment, 43*, 44-52.

Rapeli, P., Kivisaari, R., Autti, T., Kahkonen, S., Puuskari, V., Jokela, O., & Kalska, H.
(2006). Cognitive function during early abstinence from opioid dependence; a
comparison to age, gender, and verbal intelligence matched controls. *6*(9), 1-10.

Vulnerabilities in the decision process. *Behavioral and Brain Sciences, 31*, 415-487.


Principles and Practice*, (pp. 375-378). Australia: IP Communications.


Availability:


Appendix A

Diagnostic criteria for Opioid Use Disorder

A. A problematic pattern of opioid use leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:
1. Opioids are often taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control opioid use.
3. A great deal of time is spent in activities necessary to obtain the opioid, use the opioid, or recover from its effects.
4. Craving, or a strong desire or urge to use opioids.
5. Recurrent opioid use resulting in a failure to fulfill major role obligations at work, school, or home.
6. Continued opioid use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of opioids.
7. Important social, occupational, or recreational activities are given up or reduced because of opioid use.
8. Recurrent opioid use in situations in which it is physically hazardous.
9. Continued opioid use despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance.
10. Tolerance, as defined by either of the following:
   a. A need for markedly increased amounts of opioids to achieve intoxication or desired effect.
   b. A markedly diminished effect with continued use of the same amount of an opioid.
11. Withdrawal, as manifested by either of the following:
   a. The characteristic opioid withdrawal syndrome (refer to Criteria A and B of the criteria set for opioid withdrawal, pp. 547-548).
   b. Opioids (or a closely related substance) are taken to relieve or avoid withdrawal symptoms.
Appendix B

Demographic Sheet

**DEMOGRAPHIC INFORMATION**

Name: _______________________

Date: _______________________

*Please put an [X] in the box next to the answer of your choice, or write in the space provided.*

**Age:**
- Less than 18 [ ]
- 19 – 24 [ ]
- 25 – 39 [ ]
- 40 – 49 [ ]
- 50+ [ ]

**Gender:**
- Male [ ]
- Female [ ]

**Employment status:**
- Casual [ ]
- Part time [ ]
- Full time [ ]
- Self employed [ ]
- Unemployed [ ]
- If other please specify: ___

**Highest level of education completed:**
- Primary School [ ]
- High School [ ]
- Tafe/ Private College [ ]
- Bachelor Degree [ ]
- Postgraduate Degree [ ]
- If other please specify: ___

**Marital status:**
- Single [ ]
- Cohabiting (living together) [ ]
- Married [ ]
- Separated [ ]
- Divorced [ ]
- Widowed [ ]
- If other please specify: ___

The Relationship of Stress and Locus of Control on Opioid Dependency Demographic Survey, Version 1, 15/02/2017
What situational context do you believe caused you to use opioids for the first time?:
- Negative emotion
- Positive emotion
- Physical discomfort
- Personal control
- Urge and temptation
- Conflict with others
- Pleasant time with others
- Social pressure

Approximately when was the last time you used opioids?:
- 1-3 days
- 1 week
- 1 month
- 1-3 months
- 3-6 months
- 1 year
- If other please specify:

"During your lifetime, approximately how many months/years have you used opioids?:

The Relationship of Stress and Locus of Control on Opioid Dependency Demographic Survey, Version 1, 15/02/2017
Appendix C

PSS-10 scale

Perceived Stress Scale (PSS-10)

Name: __________________________
Date: __________________________

The questions in this scale ask you about your feelings and thoughts during the last month. In each case, you will be asked to indicate by circling how often you felt or thought a certain way.

0 = Never 1 = Almost Never 2 = Sometimes 3 = Fairly Often 4 = Very Often

1. In the last month, how often have you been upset because of something that happened unexpectedly?
   0 1 2 3 4

2. In the last month, how often have you felt that you were unable to control the important things in your life?
   0 1 2 3 4

3. In the last month, how often have you felt nervous and "stressed"?
   0 1 2 3 4

4. In the last month, how often have you felt confident about your ability to handle your personal problems?
   0 1 2 3 4

5. In the last month, how often have you felt that things were going your way?
   0 1 2 3 4

6. In the last month, how often have you found that you could not cope with all the things that you had to do?
   0 1 2 3 4

7. In the last month, how often have you been able to control irritations in your life?
   0 1 2 3 4

8. In the last month, how often have you felt that you were on top of things?
   0 1 2 3 4

9. In the last month, how often have you been angered because of things that were outside of your control?
   0 1 2 3 4

10. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?
    0 1 2 3 4
Appendix D

Locus of Control Scale

Locus Of Control Scale

Name: __________________________
Date: __________________________

For questions 1 to 29, please put an [X] in the box next to the answer (a. or b.) of your choice.

1. a. Children get into trouble because their parents punish them too much. [ ]
   b. The trouble with most children nowadays is that their parents are too easy with them. [ ]

2. a. Many of the unhappy things in people's lives are partly due to bad luck. [ ]
   b. People's misfortunes result from the mistakes they make. [ ]

3. a. One of the major reasons why we have wars is because people don't take enough interest in politics. [ ]
   b. There will always be wars, no matter how hard people try to prevent them. [ ]

4. a. In the long run people get the respect they deserve in this world. [ ]
   b. Unfortunately, an individual's worth often passes unrecognized no matter how hard he tries. [ ]

5. a. The idea that teachers are unfair to students is nonsense. [ ]
   b. Most students don't realize the extent to which their grades are influenced by accidental happenings. [ ]

6. a. Without the right breaks one cannot be an effective leader. [ ]
   b. Capable people who fail to become leaders have not taken advantage of their opportunities. [ ]

7. a. No matter how hard you try some people just don't like you. [ ]
   b. People who can't get others to like them don't understand how to get along with others. [ ]

8. a. Heredity plays the major role in determining one's personality. [ ]
   b. It is one's experiences in life which determine what they're like. [ ]

9. a. I have often found that what is going to happen will happen. [ ]
   b. Trusting to fate has never turned out as well for me as making a decision to take a definite course of action. [ ]
10. a. In the case of the well prepared student there is rarely if ever such a thing as an unfair test. [ ]
b. Many times exam questions tend to be so unrelated to course work that studying in really useless. [ ]

11. a. Becoming a success is a matter of hard work, luck has little or nothing to do with it. [ ]
b. Getting a good job depends mainly on being in the right place at the right time. [ ]

12. a. The average citizen can have an influence in government decisions. [ ]
b. This world is run by the few people in power, and there is not much the little guy can do about it. [ ]

13. a. When I make plans, I am almost certain that I can make them work. [ ]
b. It is not always wise to plan too far ahead because many things turn out to be a matter of good or bad fortune anyhow. [ ]

14. a. There are certain people who are just no good. [ ]
b. There is some good in everybody. [ ]

15. a. In my case getting what I want has little or nothing to do with luck. [ ]
b. Many times we might just as well decide what to do by flipping a coin. [ ]

16. a. Who gets to be the boss often depends on who was lucky enough to be in the right place first. [ ]
b. Getting people to do the right thing depends upon ability, luck has little or nothing to do with it. [ ]

17. a. As far as world affairs are concerned, most of us are the victims of forces we can neither understand, nor control. [ ]
b. By taking an active part in political and social affairs the people can control world events. [ ]

18. a. Most people don't realize the extent to which their lives are controlled by accidental happenings. [ ]
b. There really is no such thing as "luck." [ ]

19. a. One should always be willing to admit mistakes. [ ]
b. It is usually best to cover up one's mistakes. [ ]

20. a. It is hard to know whether or not a person really likes you. [ ]
b. How many friends you have depends upon how nice a person you are. [ ]

21. a. In the long run the bad things that happen to us are balanced by the good ones. [ ]
b. Most misfortunes are the result of lack of ability, ignorance, laziness, or all three. [ ]

The Relationship of Stress and Locus of Control on Opioid Dependency LOC,
Version 1, 15/02/2017
22. a. With enough effort we can wipe out political corruption.  
   b. It is difficult for people to have much control over the things politicians do in office.

23. a. Sometimes I can't understand how teachers arrive at the grades they give.  
   b. There is a direct connection between how hard I study and the grades I get.

24. a. A good leader expects people to decide for themselves what they should do.  
   b. A good leader makes it clear to everybody what their jobs are.

25. a. Many times I feel that I have little influence over the things that happen to me.  
   b. It is impossible for me to believe that chance or luck plays an important role in my life.

26. a. People are lonely because they don't try to be friendly.  
   b. There's not much use in trying too hard to please people, if they like you, they like you.

27. a. There is too much emphasis on athletics in high school.  
   b. Team sports are an excellent way to build character.

28. a. What happens to me is my own doing.  
   b. Sometimes I feel that I don't have enough control over the direction my life is taking.

29. a. Most of the time I can't understand why politicians behave the way they do.  
   b. In the long run the people are responsible for bad government on a national as well as on a local level.
Appendix E

**IDTS scale**

**INVENTORY OF DRUG-TAKING SITUATIONS (IDTS)**

*To be completed by the client:*

1. Type of drug: ______________________

*(Check one)*

- Is this your MAIN substance of abuse? □
- or
- your SECOND substance of abuse? □
- or
- your THIRD substance of abuse? □

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Listed below are a number of situations or events in which some people use ________, ________, ________, ________. Read each item carefully, and answer in terms of your own use of ________ over the past year.

- If you “NEVER” used these drugs in that situation, circle “1”
- If you “RARELY” used these drugs in that situation, circle “2”
- If you “FREQUENTLY” used these drugs in that situation, circle “3”
- If you “ALMOST ALWAYS” used these drugs in that situation, circle “4”

<table>
<thead>
<tr>
<th>I USED</th>
<th>Never</th>
<th>Rarely</th>
<th>Frequently</th>
<th>Almost Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. When I was depressed about things in general</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2. When I felt shaky, sick or nauseous</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3. When I was happy</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4. When I felt there was nowhere left to turn</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5. When I wanted to see whether I could use these drugs in moderation</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6. When I was in a place where I had used or bought these drugs before</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7. When I felt tense or uneasy in the presence of someone</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8. When I was invited to someone’s home and felt awkward about refusing when they offered me these drugs</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

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<table>
<thead>
<tr>
<th>IDTS (Drugs) continued . . .</th>
<th>I USED</th>
<th>Never</th>
<th>Rarely</th>
<th>Frequently</th>
<th>Almost Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>9. When I met some old friends and we wanted to have a good time</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10. When I was unable to express my feelings to someone</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
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<tr>
<td>11. When I felt that I had let myself down</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>12. When I had trouble sleeping</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>13. When I felt confident and relaxed</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>14. When I was bored</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>15. When I wanted to prove to myself that these drugs were not a problem for me</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>16. When I unexpectedly found some of these drugs or happened to see something that reminded me of these drugs</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>17. When other people rejected me or didn’t seem to like me</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>18. When I was out with friends and they kept suggesting we go somewhere and use these drugs</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>19. When I was with an intimate friend and we wanted to feel even closer</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>20. When other people treated me unfairly or interfered with my plans</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>21. When I was lonely</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>22. When I wanted to stay awake, be more alert, or be more energetic</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>23. When I felt excited about something</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>24. When I felt anxious or tense about something</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>25. When I wanted to find out whether I could use these drugs occasionally without getting hooked</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>26. When I had been drinking and thought about using these drugs</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>27. When I felt that my family was putting a lot of pressure on me or that I couldn’t measure up to their expectations</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>28. When others in the same room were using these drugs and I felt that they expected me to join in</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>IDTS (Drugs) continued . . .</td>
<td>I USED</td>
<td></td>
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<td>------------------------------</td>
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<td></td>
</tr>
<tr>
<td>29. When I was with friends and wanted to increase my enjoyment</td>
<td>Never Rarely Frequently Almost Always</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30. When I was not getting along well with others at school or at work</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
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<tr>
<td>31. When I started to feel guilty about something</td>
<td>1 2 3 4</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>32. When I wanted to lose weight</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>33. When I was feeling content with my life</td>
<td>1 2 3 4</td>
<td></td>
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<tr>
<td>34. When I felt overwhelmed and wanted to escape</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
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<tr>
<td>35. When I wanted to test out whether I could be with drug-using friends without using these drugs</td>
<td>1 2 3 4</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>36. When I heard someone talking about their past experiences with these drugs</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>37. When there were fights at home</td>
<td>1 2 3 4</td>
<td></td>
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<td></td>
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<tr>
<td>38. When I was pressured to use these drugs and felt that I couldn’t refuse</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>39. When I wanted to celebrate with a friend</td>
<td>1 2 3 4</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>40. When someone was dissatisfied with my work or I felt pressured at school or on the job</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>41. When I was angry at the way things had turned out</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>42. When I had a headache or was in physical pain</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>43. When I remembered something good that had happened</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>44. When I felt confused about what I should do</td>
<td>1 2 3 4</td>
<td></td>
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<tr>
<td>45. When I wanted to test out whether I could be in places where these drugs were being used without using any</td>
<td>1 2 3 4</td>
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</tr>
<tr>
<td>46. When I began to think how good a rush or a high had felt</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>47. When I felt that I needed courage to face up to someone</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>48. When I was with a group of people and everyone was using these drugs</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>49. When I was having a good time and wanted to increase my sexual enjoyment</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50. When I felt that someone was trying to control me and I wanted to feel more independent</td>
<td>1 2 3 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Appendix F

Participant information Sheet

INFORMATION FOR PARTICIPANTS

The Relationship of Stress and Locus of Control on Opioid Dependency

Introduction

You are invited to take part in a research study that is looking at select variables that may influence recidivism in adults. The objective is to investigate a series of demographic and stress variables as well as an individual’s Locus of Control in persons who are currently receiving Opioid Substitution Treatment (OST) for opioid dependency. The study involves the completion of questionnaires and a follow up urine screening two months after completing the distributed surveys. Participation in the study will take up to a maximum of 30 minutes to complete.

The study is being conducted within this institution by Louise Fitzgerald from Western Sydney University, and Dr Ahmed Moustafa from Western Sydney University.

The study is being supported by a research grant from Western Sydney University.

Study Procedures

If you agree to participate in this study, you will be asked to sign the Participant Consent Form. You will then be asked to complete a series of questionnaires including a demographic survey, the Inventory of Drug Taking Situations (IDTS), Perceived Stress score (PSS-10) and Locus of Control (LOC) scales. These will seek information on demographic factors, high risk intrapersonal environmental and interpersonal factors (IDTS), a universal assessment of stress (PSS-10) and the degree to which events are perceived as under control, or due to external factors (LOC). The completion of all scales will take about 20 to 30 minutes to do. In addition, the researchers would like to access your electronic medical record to obtain urine screening results relevant to this study.

Risks

The risks of participating in this study are:

The study requires you conducting some questionnaires which will take approximately 30 minutes. Participants will be compensated financially for their participation in the study and there will not be any kind of risk or harm for their participation.

The Relationship of Stress and Locus of Control on Opioid Dependency Participant Information Form, Version 1, 15/02/2017
Benefits

While we intend that this research study furthers medical knowledge and may improve the treatment of addiction problems in the future, it will not be of direct benefit to you.

Costs

Participation in this study will not cost you anything. You will be reimbursed $20 for your participation in our study.

Voluntary Participation

Participation in this study is entirely voluntary. You do not have to take part in it. If you do take part, you can withdraw at any time without having to give a reason. Whatever your decision, please be assured that it will not affect your medical treatment or your relationship with the staff who are caring for you.

Confidentiality

All the information collected from you for the study will be treated confidentially, and only the researchers named above (or other authorised personal as appropriate) will have access to it. The study results may be presented at a conference or in a scientific publication, but individual participants will not be identifiable in such a presentation.

It is also possible that the results of this study may be used in other like projects conducted by

Further Information

When you have read this information, Miss Fitzgerald will discuss it with you further and answer any questions you may have. If you would like to know more at any stage, please feel free to contact Dr Ahmed Moustafa (Phone: 9772 6847; email: a.moustafa@uws.edu.au), or Louise Fitzgerald (email: 17220763@student.westernsydney.edu.au)

This information sheet is for you to keep.

Ethics Approval and Complaints

This study has been approved by the Ethics Review Committee (RPAH Zone) of the Sydney Local Health District. Any person with concerns or complaints about the conduct of this study should contact the Executive Officer on 02 9515 6766 and quote protocol number … … (to be added once protocol number has been received)
PARTICIPANT CONSENT FORM

The Relationship of Stress and Locus of Control on Opioid Dependency

I, ............................................................................................................. [name] of ...............................................................................................................................
[address] have read and understood the Information for Participants on the above named research study and have discussed the study with ..................................................................................

I have been made aware of the procedures involved in the study, including any known or expected inconvenience, risk, discomfort or potential side effect and of their implications as far as they are currently known by the researchers. I understand that my participation in this study will allow the researchers and others, as described in the Information for Participants, to have access to my electronic medical record, and I agree to this.

I understand that the results of this study may be used in other like projects conducted by Dr Ahmed Moustafa and colleagues but will not be used in any way that reveals my identity.

I freely choose to participate in this study and understand that I can withdraw at any time.

I also understand that the research study is strictly confidential.

I hereby agree to participate in this research study.

NAME: .............................................................................................................

SIGNATURE: .............................................................................................................

DATE: .............................................................................................................

NAME OF WITNESS: .............................................................................................................

SIGNATURE OF WITNESS: .............................................................................................................