Amy Louise Hodgson

A study of emotion regulation in individuals with schizophrenia and comorbid substance misuse.

Submitted in part fulfilment of the degree of doctorate in Clinical Psychology at the University of Edinburgh

August 2005
DClinPsychol Declaration of own work

This sheet must be filled in (each box ticked to show that the condition has been met), signed and dated, and included with all assessments - work will not be marked unless this is done

Name: **AMY HODGSON**

Assessed work: CS SSR Professional Issues **Thesis**
(please circle)

Title of work: **A STUDY OF EMOTION REGULATION IN INDIVIDUALS WITH SCHIZOPHRENIA AND COMORBID SUBSTANCE MISUSE.**

I confirm that all this work is my own except where indicated, and that I have:

- Clearly referenced/listed all sources as appropriate
- Referenced and put in inverted commas any quoted text of more than three words (from books, web, etc)
- Given the sources of all pictures, data etc. that are not my own
- Not made undue use of essay(s) of any other student(s) either past or present (or where used, this has been referenced appropriately)
- Not sought or used the help of any external professional agencies for the work (or where used, this has been referenced appropriately)
- Acknowledged in appropriate places any help that I have received from others (e.g. fellow students, technicians, statisticians, external sources)

I understand that any false claim for this work will be penalised in accordance with the University regulations

Please note:

a) If you need further guidance on plagiarism, you can:
   i/ Speak to your director of studies or supervisor
   ii/ View university regulations at [http://www.aaps.ed.ac.uk/regulations/Plagiarism/Intro.htm](http://www.aaps.ed.ac.uk/regulations/Plagiarism/Intro.htm)

b) Referencing for all assessed work should be in the format of the BPS style guide, which is freely available from the BPS web site
Acknowledgements

Thanks to Sean Harper for supervision and advice and to Arthur Still for statistical support. Thanks to Dr David Kavanagh of The University of Queensland for making the DrugCheck measure available and to Mark Trembath, also of The University of Queensland for providing details of the validation study for this measure. Thanks also to Katie Phillips for use of the Child and Adolescent Emotion Regulation Questionnaire in this study.

On the practical side thanks to all those involved in facilitating the study; Gordon Mitchell, Dr Paul Cavanagh and all staff of the Community Outreach Team, Hillview Day Hospital and the Continuing Care Clinic. Particular thanks go to all those individuals who took the time to take part.

No thanks to my noisy neighbours, lots of thanks to a spell in peaceful Yorkshire and particular thanks to not having a TV for the last 3 months.

Thanks to all who have provided support and distraction when needed, and especially to Matt for being calm, helpful, cooking my tea and delaying your renovation ambitions to allow me to get this done.
Objective: To evaluate an emotion regulation model of vulnerability to substance misuse in schizophrenia.

Background: Excess vulnerability to substance misuse in schizophrenia is a significant clinical problem associated with a range of negative outcomes. Self-reported motives for substance misuse and traits associated with the severity of misuse indicate that the behaviour may serve an emotion regulation function. This study sought to evaluate whether there are differences in either emotion experience or emotion regulation between schizophrenia only and comorbid groups that are in keeping with an emotion regulation model.

Methods: Participants were 40 psychiatric outpatients who met ICD-10 criteria for schizophrenia or a related disorder. Participants were screened for substance misuse using the DrugCheck and initially allocated to two groups based on the presence or absence of current substance misuse. Between-group comparisons were made for measures of affect (Positive and Negative Affect Scale) and two measures of emotion regulation (Emotion Regulation Questionnaire and the Child and Adolescent Emotion Regulation Questionnaire).

Results: No significant between-group differences were identified on any measures, although the analyses had insufficient power. Indications were that grouping criteria may have obscured any differences. Reclassification on the basis of lifetime substance misuse status (presence or absence over lifetime) was followed by reanalysis. Significant between-group differences were then found for measures of positive affect (comorbid group lower), the emotion regulation strategy expressive suppression (comorbid group higher) and the use of Internal-Functional strategies for the regulation of emotion (comorbid group lower).

Conclusions: Findings offer some initial support for an emotion regulation model of comorbidity, between-group differences having been identified on both a measure of affective experience and emotion regulation strategies. Replication of these findings is essential and possibilities for further research developments are discussed.
1. INTRODUCTION

1.1 Overview

1.2 Comorbidity of substance misuse and schizophrenia
   1.2.1 Excess vulnerability to substance misuse in schizophrenia
   1.2.2 Defining substance misuse

1.3 Aetiological models of comorbid substance misuse and severe and enduring mental illness
   1.3.1 Common factor models
   1.3.2 Secondary psychopathology models
   1.3.3 Secondary substance use models
     1.3.3.1 The supersensitivity model
     1.3.3.2 Psychosocial vulnerability models
   1.3.4 Bi-directional models
   1.3.5 Summary of key points from the review of aetiological theories of comorbidity

1.4 Emotion Regulation
   1.4.1 Defining emotion regulation
   1.4.2 Models of emotion regulation
   1.4.3 Functional emotion regulation
   1.4.4 Substance use as an emotion regulation strategy
   1.4.5 Emotion regulation or affect regulation?

1.5 Emotional Functioning and Schizophrenia
   1.5.1 Historical perspective
   1.5.2 Current knowledge of emotional functioning in schizophrenia
   1.5.3 Emotion regulation and schizophrenia
   1.5.4 Emotional functioning and regulation in schizophrenia with comorbid substance misuse
   1.5.5 How might difficulties in emotion regulation have developed in schizophrenia and dual diagnosis?

1.6. Rationale for the Present Study

1.7 Research Question and Hypotheses

2. METHODOLOGY

2.1 Design

2.2 Power analysis

2.3 Participants

2.4 Measures and rationale for their selection
   2.4.1 Reliability of using self-report measures of emotional functioning with individuals diagnosed with schizophrenia
   2.4.2 The DrugCheck
   2.4.3 The Positive And Negative Affect Scales (PANAS)
   2.4.4 Measures of Emotion Regulation
     2.4.4.1 The Emotion Regulation Questionnaire (ERQ)
     2.4.4.2 The Child and Adolescent Emotion Regulation Questionnaire (CA-ERQ)

2.5 Procedure

2.6 Ethical Approval
1. INTRODUCTION.

1.1 Overview.

The clinical significance of comorbid severe and enduring mental illness (SEMI) and substance misuse will first be illustrated followed by a review of aetiological theories of comorbidity and relevant research findings. This review will demonstrate the grounds for investigating an emotion regulation model of comorbidity. Theories and models of emotion regulation shall then be outlined followed by research and theory relevant to emotional functioning in schizophrenia and comorbid substance misuse to provide a broader theoretical context to the study. The aims and rationale for the study will then be laid out together with specific research hypothesis.

1.2 Comorbidity of substance misuse and schizophrenia.

1.2.1 Excess vulnerability to substance misuse in schizophrenia.

It is well established that individuals who experience serious mental health difficulties, particularly psychotic illnesses such as schizophrenia and bipolar disorder, are at considerably greater risk of substance misuse when compared to the general population (Mueser et al., 1998; Graham et al., 2004). Moreover, research conducted in the UK (Menezes et al. as cited in Williams, 2002) examining the one-year prevalence rate for substance and alcohol use problems amongst patients with psychoses found rates of 36.3 per cent. Within this, there was considerably more alcohol misuse (31.6%) compared to
misuse of other drugs (15.8%) and a substantial overlap, indicating poly-substance use. By way of comparison to the UK general population, figures for current alcohol and drug dependence show considerably lower prevalence rates of 5 and 2 per cent respectively (Farrell et al. as cited in Williams, 2002), although figures for misuse are likely to be larger than those for dependence.

However, it is necessary to be cautious with prevalence rates based on clinical populations as they may be inflated as a result of the fact that either disorder increases the chances of being in treatment and therefore the presence of both disorders means individuals with comorbid problems are more likely to be found in treatment populations, a phenomenon known as the Berkson Bias (Blanchard et al., 2000). Therefore it is important to examine prevalence rates for non-treatment samples.

Data from the Epidemiological Catchment Area study conducted in the USA provides one source of such information. Lifetime prevalence rates for any substance use disorder (including alcohol) were 47 per cent amongst individuals diagnosed with schizophrenia, 60.7 per cent for those with a diagnosis of bipolar disorder and 16.7 per cent for the general population (Reiger et al. as cited in Williams, 2002). Although these data are not based on UK populations and are somewhat dated, they clearly demonstrate that rates of comorbidity in non-treatment samples are also considerably greater than those for substance use disorders in the general population.
Whilst the rates found in different prevalence studies will vary to some degree depending on the definitions, samples and methodologies used, findings do consistently demonstrate the significantly greater risk of substance use problems in individuals with diagnoses of SEMI (Graham et al., 2004). Blanchard et al. (2000) concluded, following a review of the literature, that amongst individuals with a diagnosis of schizophrenia comorbid substance misuse is consistently present in 40 to 50 per cent of cases at some stage in their lifetime and that poly-substance use is common within this group.

Substance misuse in the context of SEMI, often referred to as dual diagnosis, is associated with increased negative outcomes for the individual and their treatment. Increased rates of relapse and hospitalisation, homelessness, violence and associated legal problems, family stress, physical ill health and treatment non-compliance are consistently found (Drake & Brunette as cited in Mueser et al., 1998). This group is generally more difficult to engage with services and treat effectively and recommendations to develop integrated treatment packages which address both mental health needs and substance use problems in a co-ordinated manner are not yet commonly reflected in available services in the UK (Graham et al., 2004).

Given then that the rates of comorbid substance misuse and SEMI are substantial and are associated with various adverse consequences both for the individual and the systems which aim to help them, there is a clear imperative to improve our understanding of this phenomenon in order to develop supports and treatment packages which maximise the
potential for positive outcomes. Before reviewing aetiological theories of dual-diagnosis, it is first necessary to clarify some of the relevant terminology.

1.2.2 Defining substance misuse.
Psychoactive substances are universally used in human societies (Hussein Rassool, 2002), serving a variety of functions. However, there is also potential for use in a way which may cause adverse outcomes for the individual or others, such as adverse effects on physical health, behaviour, psychological and social functioning. The misuse of a substance can be defined in various ways; in terms of what is socially unacceptable, legally sanctioned or potentially harmful to the individual or others (Hussein Rassool, 2002).

For the purposes of this study the term misuse will be used to refer to:

'Any taking of a drug which harms or threatens to harm the physical or mental health or social well-being of an individual, or other individuals, or of society at large, or which is illegal.'

(Royal College of Psychiatrists as cited in Hussein Rassool, 2002, p.14)

Although the emphasis in this study will not be on legality and indeed alcohol misuse will be included in substance misuse.
The World Health Organisation (WHO) recommends using terms such as unsanctioned, hazardous, dysfunctional (detrimental to psychological and social functioning), and harmful to clarify the exact nature of any misuse (Hussein Rassool, 2002). The term misuse is used within the WHO framework to describe a pattern of substance use which has the potential to cause harm to various aspects of the individual’s functioning.

The concept of substance dependence is perhaps better articulated and includes both physical and psychological aspects (Hussein Rassool, 2002). Physical dependence is associated with increased tolerance for the substance and symptoms of physical withdrawal. Narrowing of the behavioural repertoire around use and a compulsion to continue use, despite adverse consequences are also part of the dependence syndrome (Hussein Rassool, 2002). It is possible for an individual to be psychologically dependent on a given substance and experience a strong compulsion to continue use in the absence of physical dependence (Hussein Rassool, 2002).

Applying concepts of maladaptive substance use to individuals with dual diagnoses is particularly complex. This is because levels and patterns of use of substances that may not be considered particularly problematic in the general population frequently lead to adverse consequences in this sensitive group (Hussein Rassool, 2002). Therefore, using physical dependence as a mark of problematic use tends to be relatively uninformative as use of substances at levels insufficient to cause physical dependence is frequently associated with significant adverse social and psychological outcomes.
For the purposes of the current research the focus shall be on establishing the presence or absence of any misuse (including hazardous, dysfunctional and harmful misuse) rather than meeting criteria for abuse or dependence (e.g. ICD-10 criteria, DSM-IV criteria). This is considered to be more appropriate as relatively low levels of misuse have been demonstrated to have significant functional consequences in psychotic groups (Hussein Rassool, 2002). The screening method used shall be selected for its ability to identify patterns of potentially harmful misuse. The terms dual diagnosis and comorbidity shall be used for brevity to refer to cases where an individual has a severe and enduring mental health problem together with evidence of the misuse of psychoactive substances, including alcohol.

1.3 Aetiological models of comorbid substance misuse and severe and enduring mental illness.

Despite the prevalence of comorbidity and the associated adverse consequences, relatively little is understood about the factors that underlie the excess risk of substance misuse in this population (Blanchard et al., 2000; Mueser et al., 1998). The best predictors of substance misuse by an individual with psychosis remain demographic variables such as age, gender and socio-economic factors (Blanchard et al., 2000), whilst psychological vulnerability factors remain poorly understood. No one model is likely to provide a complete account of this phenomena and several models may account for some of the excess vulnerability (Mueser et al., 1998), indeed there is a role for explanatory
models which address either the initiation of substance use, it’s maintenance, or both as the relevant factors may differ.

Kushner and Mueser (as cited in Graham et al., 2004) identified four general types of aetiological models of comorbidity:

- Common factor models.
- Secondary psychopathology models.
- Secondary substance use models.
- Bi-directional models.

The empirical support for each of these shall be reviewed below.

1.3.1 Common factor models.

This approach to comorbidity proposes that one or more factors independently increase the risk of both difficulties. Research has focused on genes and anti-social personality disorder (ASPD) as common factors (Mueser et al., 1998). Whilst there is substantial evidence for a genetic contribution to both SEMI and to substance use disorders, findings do not support a genetic contribution to the excess of substance misuse in the SEMI population (Blanchard et al., 2000; Mueser et al., 1998).

There is though, evidence that ASPD could account for some of the excess vulnerability to substance misuse (Mueser et al., 1998). Traits associated with the ASPD construct, such as impulsivity or novelty-seeking, and negative affectivity are consistently found at elevated levels in individuals with dual diagnoses (Blanchard et al., 2000). However, as
these findings are correlational in nature, they cannot demonstrate causality and the association could be an outcome of substance use rather than a precursor (Blanchard et al., 2000). Longitudinal research is needed to clarify the interpretation of these associations.

Other potential common factors include aspects of cognitive functioning and socio-economic status, however there is limited research evidence available on these (Mueser et al., 1998). The relevance of socio-economic factors is well established in relation to substance misuse in the general population (Drake et al., 2002) and individuals who experience SEMI have been consistently found to experience various aspects of socio-economic adversity disproportionately. Therefore, this common-factor has face-validity and some authors urge more attention to be paid to such influences although as yet there is insufficient empirical evidence to properly assess it’s relevance (Drake et al., 2002).

1.3.2 Secondary psychopathology models.
Aetiological models of this type propose that substance use directly contributes to the development of SEMI which would not otherwise have developed (Graham et al., 2004) resulting in higher levels of substance misuse found in these groups. Three main strands of evidence are used to evaluate the secondary psychopathology model; substance use as a predictor of the later development of psychosis in long-term follow-up studies, clinical cases of individuals who develop psychoses following heavy substance use and epidemiological and cross-cultural data.
Andreasson et al.'s (as cited in Mueser et al., 1998) longitudinal study of Swedish army conscripts is often cited as one of the key sources of evidence for this theory. Various data were recorded at conscription, including cannabis use, and health service records of psychiatric illness were examined at a long-term follow up. A significant association between cannabis use at conscription and the later development of psychosis was found. However, although cannabis use was a predictive factor in this analysis, it was not the only effective predictor (psychiatric diagnosis at conscription, poor social adjustment, solvent abuse and indicators of a disadvantaged upbringing were others) and the correlational approach used is insufficient to demonstrate causality. Those who used cannabis and those who did not may have differed in some other important way which influenced their choice to use substances at the outset (Mueser et al., 1998). Indeed, the relative risk of developing schizophrenia associated with heavy cannabis use decreased substantially when psychiatric diagnoses at intake were controlled (Mueser et al., 1998).

Recent empirical reviews advise that teasing out the sequence of onset of substance use and psychosis is generally uninformative regarding aetiology (Blanchard et al., 2000; Mueser et al., 1998). Developmentally, substance use tends to have its onset at an earlier age than psychotic symptoms and the complex prodromal phase in the development of psychosis makes it extremely difficult to establish clear sequences as many prodromal signs go unrecognised at the time. Furthermore, research in this area tends to focus on substances with psychotomimetic properties, such as cannabis and amphetamines, which seriously restricts their application as models of substance use in psychosis as the most frequently misused substance in this population is alcohol (Blanchard et al., 2000).
Studies of clinical cases of psychosis which occur and persist following heavy use of psychotomimetic substances, such as LSD, have demonstrated that the clinical characteristics of such cases do not differ significantly from those which are not associated with substance misuse. However, it is generally considered more appropriate to understand substance misuse in these cases as a stressor within a stress-vulnerability framework than as a direct cause of psychosis (Mueser et al., 1998).

Hypotheses about a possible causal link between psychotomimetic substances and psychosis have been gaining attention since the increase in the use of these substances in western societies in the mid 20th century. However, observation of long-term epidemiological data and cross-cultural patterns provides a different perspective. The use of particular substances varies significantly across cohorts and over time whilst rates of psychoses remain very consistent over time, undermining claims of a significant causal link between the two phenomena (Blanchard et al., 2000). Additionally, cultures and societies differ in terms of patterns and types of substances commonly used but cross-cultural comparisons show remarkable consistency in rates of psychotic illnesses (Blanchard et al., 2000), which would suggest the latter phenomenon is not significantly influenced by patterns of the former.

Whilst it is clinically apparent that for some individuals substance misuse is associated with the onset and ongoing exacerbation of psychotic illness, it is clear from the empirical evidence that the secondary psychopathology models provide insufficient
accounts of the excess of substance-use problems in this group, particularly in terms of the use of alcohol and other non-psychotomimetic substances. Substance use seems more appropriately considered as a stressor within a stress-vulnerability framework rather than a direct causal variable (Mueser et al., 1998), the significance of which varies between individuals.

1.3.3 Secondary substance use models.

These models propose that factors associated with SEMI increase an individual's vulnerability to developing substance misuse. There has been theoretical and research exploration of several possible explanatory models within this broad approach.

1.3.3.1 The supersensitivity model.

Essentially, the supersensitivity model proposes that the actual level of substance use may not differ significantly between the general population and individuals with SEMI, but that the latter group are particularly sensitive to experiencing adverse effects from relatively low levels of substance use. This leads to their use being detected more often and classified as abuse or misuse (Mueser et al., 1998). This differs from the central premise on which other aetiological models are based, that the higher prevalence of substance-related difficulties in this group represents a greater use and that therefore the factors that contribute to motivating this elevated usage need to be identified.
Following this model, individuals experience SEMI as the result of an interaction between a psychobiological vulnerability (involving increased stress sensitivity) and environmental stressors. This same underlying sensitivity to stress increases the likelihood that the individual will experience negative consequences as a result of the use of psychoactive substances.

Several strands of research provide support for this model. Individuals with SEMI tend to use lower quantities of substances than individuals with primary substance use disorders and experience greater negative consequences as a result of this level of use than are associated with a similar level of use in the general population (Mueser et al., 1998), which may then bring their use to the attention of services. Individuals with dual diagnoses demonstrate a much lower level of physical dependence, in line with lower typical consumption, than would be the case in individuals experiencing adverse consequences from substance use in the general population. ‘Challenge tests’ have also found participants with schizophrenia to be more sensitive to the adverse effects of amphetamines (Mueser et al., 1998). The evidence then suggests that individuals with SEMI are less able to use substances at a moderate level without experiencing adverse consequences (Mueser et al., 1998).

This approach does indeed offer a useful way of reconciling the two consistent findings of a large proportion of individuals with SEMI who experience significant negative consequences associated with the use of psychoactive substances, with that of consumption of a level of substances which may not be associated with such
consequences in other populations. However, it does not address what the motivations may be for continued substance misuse in this group, although the implication is that one can expect to find that motivations reflect those found in the general population.

1.3.3.2 Psychosocial vulnerability models.

1.3.3.2.1 The self-medication hypothesis.

The self-medication hypothesis (SMH) of addictive disorders was initially developed by Khantzian (1985) based on psychodynamic research and theory and clinical experience of working with individuals in the general population who abuse substances, particularly opiates and cocaine. Khantzian (1985) proposed that such individuals were attempting to alleviate ‘painful affect states and related psychiatric disorders’ (p.1259) through their use of substances which were actively selected through ‘an interaction between the psychopharmacologic action of the drug and the dominant painful feelings with which they struggle’ (p.1259). Whilst this may be adaptive and facilitate coping in the short-term, it often leads to various negative outcomes for the individual in the longer term.

In applying the SMH to individuals with dual diagnoses, theorists have tended to focus on the self-medication of specific psychiatric symptoms, rather than of ‘painful affect states’. Support for this application of the SMH has been inconsistent, although the terminology seems to have infiltrated clinical parlance and thereby the hypothesis has gained credence whilst lacking rigorous research support.
Mueser et al. (1998) identify three strands of research which have a bearing on the veracity of the SMH as applied to comorbidity; self-reported reasons for substance misuse, patterns of substance selection by different diagnostic groups, and of patterns of use of specific substances in relation to specific psychiatric symptoms. The general consensus is that all three strands of evidence offer little support to the SMH of comorbidity as currently formulated (Blanchard et al., 2000; Mueser et al., 1998; Graham et al., 2004; Hussein Rassool, 2002). Reviews of substance choice and patterns of use amongst dually diagnosed groups have shown that these individuals tend to misuse the same substances as the general population and that their substance choice is mainly guided by availability rather than by any diagnostic or symptom-related factors as would be expected following this interpretation of the SMH (Graham et al., 2004).

Self-report studies have failed to generate consistent reports of use to cope with specific psychiatric symptoms (Mueser et al., 1998). These studies tend to identify reasons for use which are very similar to those produced by research with the general population (e.g. Cooper et al., 1995); ‘enhancement [of positive affect]’, ‘coping with unpleasant affect’, ‘social’ and ‘conformity and acceptance’ motives for example, with very limited endorsement of motives such as ‘coping with positive symptoms and side-effects’ (Spencer et al., 2002). It is notable that two of the four consistently endorsed motivations for substance use reflect attempts to modulate or cope with affects.
A recent study which sought to clarify the relevance of the SMH to comorbidity operationalized the theory in terms of several hypotheses and reported modest but consistent support for the SMH (Goswami et al., 2004). Goswami et al. (2004) found that dually diagnosed individuals expressed substance-specific reasons for use, perceived substance-specific effects and there was a match between reasons for use and perceived effects. This was interpreted as supportive to a SMH understanding, however, in terms of reasons for use being symptom orientated, there was only equivocal support, which rather undermines the relevance of the ‘match’ found between expectancies and perceived effects to a specifically self-medication model as currently conceived.

Voruganti et al. (1997) proposed a variation on the SMH of comorbidity in terms of whether individuals may develop substance misuse as a way of ameliorating neuroleptic medication induced dysphoria. They concluded from a preliminary study that those who experienced this side effect were four times more likely to develop substance misuse during the course of their treatment. However, they failed to control for other significant differences between the groups such as younger age and clinical instability which may have had a confounding effect. Blanchard et al. (2000) dismiss the effects of neuroleptic medications as a sufficient motivator for a substantial proportion of substance misuse in psychosis arguing that substance misuse generally precedes neuroleptic treatment and that there is little indication that dually diagnosed individuals preferentially use dopamine agonists, which would follow logically from this hypothesis.
1.3.3.2.2 A broader reconsideration of the self-medication hypothesis of comorbidity.

The emphasis of the SMH as applied to comorbidity of substance misuse and SEMI has been on the self-medication of specific psychiatric symptoms, often positive psychotic symptoms. However, this does not reflect the centrality of affect and its modulation in Khantzian’s (1985) original articulation of the hypothesis. Indeed, Khantzian (1985) summarizes psychodynamic theory which highlights difficulty in recognising, tolerating and modulating painful affects amongst individuals who misuse substances. A two-fold emotion regulating function is advanced for substance use of facilitating defence against aversive emotions and also allowing the experience of certain emotions, which are usually prevented by rigid defences, in a controlled and tolerable manner.

In developing the SMH Khantzian (1985) places considerably more emphasis on emotional dysfunction and deficits in emotion regulation and tolerance than has been recognised in the research into its application to comorbidity. In his 1997 rearticulation and clarification of the SMH Khantzian continues to emphasise deficits in affect tolerance and regulation as key psychological vulnerabilities for the development of problematic substance use. This psychological model of vulnerability, which Khantzian views as complementary to relevant biogenetic and sociocultural models, proposes that substance misuse is the result of a general deficit of self-regulation which includes regulation of emotions, inter-personal relationships, self-care and self-esteem. The experience and regulation of painful affects is emphasised as a core problem considered to result from the disruption of normal affective development. Emotions may be
experienced as unbearably strong, confusing, inaccessible or not amenable to expression in such cases.

According to this model, individuals will demonstrate some degree of preference for a particular type of substance. These preferences are shaped by interactions between the action of the substance, the individual’s traits and defences and the nature of their subjective distress. Khantzian (1997) makes the point that there may not be consistent associations between particular diagnoses and preferred substances as even within such groups there can be qualitative differences between individuals in the nature of subjective emotional experience. The example given is that in depression the core emotional experience for one individual may be sadness and despair whilst for another person it may be anger.

Khantzian (1997) specifically addresses the application of the SMH to individuals with schizophrenia and comorbid substance misuse and observes that such individuals often have particular difficulty tolerating and managing painful affects. Khantzian (1997) also goes on to identify negative psychotic symptoms as a source of considerable distress for individuals with schizophrenia, which he considers to be a key vulnerability for substance misuse. The appeal of substance use in this group, following this view, is largely based on the temporary relief of negative symptoms and the distress associated with them. The frequent onset of substance use prior to the individual’s schizophrenia is accounted for in terms of prodromal symptoms and associated distress.
Whilst Khantzian (1997) does continue to emphasise the role of distress, the focus on negative symptoms in his conceptualisation of substance misuse in schizophrenia is somewhat of a shift away from the view, expressed earlier in the same paper that;

\[ \text{it is not so much a psychiatric condition that one self-medicates, but a wide range of subjective symptoms and states of distress that may or may not be associated with a psychiatric disorder.} \]

Different interpretations of the SMH as applied to comorbid substance misuse and schizophrenia have led to varied research approaches, typically concentrating on the relationship between measurable positive and negative symptoms and substance use. However, following a broad consideration of Khantzian’s approach (1985; 1997), in so far as symptomatology is of relevance to substance use, it is in terms of its interaction with subjective emotional experiences and any related distress.

A narrow interpretation of the SMH applied to comorbidity has led to the neglect of it’s implications for substance misuse as means of regulating and tolerating emotions. The SMH in this broader sense may not be inconsistent with an emotion regulation approach to understanding comorbidity. Khantzian (1985) accepts that the SMH is not the only explanation for substance misuse and addiction but offers an approach that may be of clinical use and relevance in some cases. Taking on the broader meaning of the hypothesis may well increase its relevance and utility for dually diagnosed clients.
1.3.3.2.3 The alleviation of dysphoria model.

The alleviation of dysphoria model of comorbidity proposes that experiencing SEMI involves a vulnerability to dysphoric emotional experiences which motivates substance use in an attempt to alleviate these experiences. A broader approach also considers the role of substance use in enhancing positive emotional experiences. Following Kushner and Mueser's (as cited in Graham et al., 2004) classification of models of comorbidity this vulnerability to dysphoria is considered to be secondary to the experience of SEMI, although a vulnerability to dysphoria and diminished experience of positive affect may be primary vulnerabilities which could have also contributed to the development of SEMI.

These proposed motivations for substance use, of increasing positive and reducing negative emotional experiences, are essentially very similar to two of the key motivations for substance use consistently identified in the general population (Cooper, 1994; Cooper et al. 1995). Mueser et al. (1998) propose that these motives are involved in the initiation of substance use which is then maintained through physiological addictive processes. However, this fails to take account of the fact that the level at which individuals with SEMI use substances is often insufficient to lead to physiological dependence although they may well experience psychological dependence. Substance misuse may be maintained through the reinforcement of the short-term efficacy of use in achieving the desired goal of reducing negative or increasing positive emotions.

Two strands of empirical evidence clearly support the relevance of this model to understanding comorbidity. Firstly, it is well established that individuals with SEMI
experience consistently greater levels of dysphoric emotions relative to the general population (Birchwood et al., 1993). Dysphoria refers here to aversive and negative emotional experience including a range of discreet emotions such as anxiety and sadness. Secondly, studies of self-reported motives for and expectancies of substance use in comorbid groups consistently find the alleviation of negative emotions and enhancement of positive emotions amongst the primary motives expressed. For example, Spencer et al. (2002) identified four main types of motivation for substance use in this group; enhancement of positive emotions, coping with unpleasant affect, conformity and acceptance and social motives. Dixon et al. (1991) also identified key motivations which are associated with influencing affective experience; relieving depression and achieving relaxation or a ‘high’. An earlier report by the same group (Dixon et al., 1990) identified affective changes as prominent in self-reports of the effects of substance use including reduced dysphoria and anxiety together with a feeling of increased energy.

The general consensus is that research is largely consistent with the alleviation of dysphoria model of comorbidity (Mueser et al., 1998; Graham et al., 2004; Hussein Rassool, 2002). However, the empirical support is not currently as strong as that for the common-factor model of ASPD and the supersensitivity model (Mueser et al., 1998). Some theorists argue that the alleviation of dysphoria model could be subsumed into a multiple risk factor model which would cover various social, economic, cognitive and interpersonal vulnerabilities (Mueser et al., 1998; Drake et al., 2002) with dysphoria constituting another risk factor. However, one group of researchers has recently developed the alleviation of dysphoria model and proposed an as yet largely untested
integrative affect regulation model of comorbidity which incorporates links with the common-factor model of ASPD and may offer a more coherent and inclusive understanding.

1.3.3.2.4 Blanchard et al.'s (2000) integrative affect regulation model.

Blanchard and colleagues (2000) consider that the study of comorbidity has been retarded by a failure to give sufficient attention to enduring individual differences, such as personality, temperament and coping style. The tendency to focus on links between substance misuse and psychotic symptoms in research means that, due to the transient and changing nature of symptoms, it has not been possible to identify enduring individual characteristics that can elucidate the longer-term relationship between the two phenomena of psychosis and substance misuse.

Blanchard et al. (2000) highlight research into substance use with the general population those with primary substance use disorders. This body of research has demonstrated the value of examining enduring individual differences such as personality, coping style, problem solving and temperament (Blanchard et al., 2000). Research already conducted into these constructs with individuals with schizophrenia and comorbid groups is summarised and forms the basis on which the model is developed. In developing their proposed model, Blanchard et al. (2000) focus specifically on schizophrenia.

Research with the general population and those with primary substance use disorders, provides consistent support for the relevance of two particular personality traits; negative
affectivity/neuroticism and disinhibition/impulsivity (Blanchard et al., 2000). Negative affectivity/neuroticism represents a tendency to experience negative mood states, a lower threshold for experiencing negative affect and a relatively reduced tolerance of stress and negative affect (Blanchard et al., 2000). Blanchard et al. (2000) propose that the mechanism by which this trait has a bearing on vulnerability to comorbid substance misuse is in terms of the ability to effectively regulate negative affect. The individual with high trait negative affectivity/neuroticism will be both more likely to experience negative affect and less tolerant of this experience and so will seek a way to regulate and reduce the affective experience, which may be more or less effective.

The disinhibition/impulsivity construct involves poor self-regulation of behaviour and has also been linked to constructs such as sensation or novelty seeking (Blanchard et al., 2000) which confer a greater orientation to seeking stimulating and positive affective experiences. The research of Cooper and colleagues into alcohol use has demonstrated disinhibition/impulsivity to be related to the use of alcohol with the aim of enhancing positive affect (Cooper et al., 1995) in the general population.

In terms of the relevance of these traits to substance misuse by individuals with schizophrenia, there is an accumulation of research which confirms negative affectivity/neuroticism as an enduring trait in individuals with schizophrenia and impulsivity or disinhibition during childhood has been associated with the later development of schizophrenia (Blanchard et al., 2000). Blanchard et al. (1999) have also identified an association between increased negative affectivity and the severity of
substance misuse in a comorbid group and a moderate association between Disinhibition and comorbid substance use. Additionally, Dervaux et al. (2001) have found a significant difference in both impulsivity and sensation seeking traits between a group of individuals with schizophrenia and comorbid substance misuse and a comparison group of individuals with schizophrenia only. An affect or emotion regulating function of substance use is also in accordance with consistently reported motives of enhancing positive affect and coping with or reducing negative affect (Spencer et al., 2002; Dixon et al., 1991) and beliefs and expectancies that dually diagnosed individuals hold regarding the effects of substances (Dixon et al., 1990).

It is consistent with the accumulation of these research findings, which identify elevated traits that increase vulnerability to negative affect and orientation to seek positive affect, to conceptualise a key function of substance misuse in schizophrenia as being regulation of affective or emotional experience. What remains to be examined more closely is the proposed mechanism of emotion or affect regulatory processes.

Blanchard et al. (2000) propose that the relationship between traits, which shape emotional experiences and attempts to regulate affect, with substances is mediated by factors such as coping style and problem solving skills. Where an individual has poor problem solving skills and a maladaptive coping style, increased stress is likely to result from a failure to deal effectively with a problematic situation which then increases the imperative to alleviate an aversive emotional state in an alternative way. Research with the general population has supported links between avoidant coping styles and
consumption of alcohol to cope with stress (Cooper et al., 1992) as well as an association between poor problem-solving skills and substance use disorders (Blanchard et al., 2000). With respect to individuals with schizophrenia, deficits in coping and problem-solving skills have been identified (Blanchard et al., 2000). Individuals may therefore seek alternative ways to alleviate their experiences of stress having been unable to effectively modify the cause of their stress. When these difficulties coincide with positive expectancies of the effects of substances on negative affect, the likelihood of using substances as a means of coping is increased. Indeed, the presence of such ‘coping’ motives has been associated with misuse of psychoactive substances amongst individuals with schizophrenia (Spencer et al., 2002).

A key distinction drawn between the proposed integrative affect regulation model and the self-medication model is that the former focuses on enduring individual differences which can therefore improve understanding of the longer-term risk of substance use problems in this group, but this is based on the narrower interpretation of the SMH. This integrative model is not conceptualising substance use as necessarily secondary to schizophrenia or vice versa, but that the development of both disorders may be influenced by primary difficulties in the regulation of affective experiences, personality traits and coping.

At present this model awaits substantial empirical testing, although there is considerable existing research that is consistent with some of its key propositions, as outlined above.
The proposed mechanism of affect or emotion regulation in particular has been mainly examined indirectly and remains largely unexplored.

The key propositions that are embedded in an emotion or affect regulation model of comorbidity such as that proposed by Blanchard et al. (2000) are as follows:

1. Individuals with comorbid substance misuse and schizophrenia are vulnerable to experiencing elevated negative affect and lower positive affect.
2. Individuals with comorbid substance misuse and schizophrenia are more ineffective at regulating their emotional/affective experiences.
3. The degree or nature of these difficulties significantly differ between individuals with schizophrenia with and without comorbid substance misuse and increase an individual’s vulnerability to developing comorbid substance misuse.

1.3.4 Bi-directional models.

This aetiological approach essentially proposes that comorbidity results from an ongoing process of interaction between SEMI and substance misuse, each increasing an individual’s vulnerability for the other (Mueser et al., 1998). For example, an individual biologically predisposed to schizophrenia may experience an acute episode following a period of substance misuse. Some of the associated consequences of schizophrenia may then contribute to the motivations the individual has for continued substance use, which in turn contributes to a worsening course of illness. This approach is consistent with clinical experience and research findings which confirm the deleterious effect on the course of SEMI of continued substance misuse (Graham et al., 2004), at this stage they have not been fully developed or empirically tested (Mueser et al., 1998).
1.3.5 SUMMARY OF KEY POINTS FROM THE REVIEW OF AETIOLOGICAL THEORIES OF COMORBIDITY.

• There is empirical support for a common-factor of anti-social personality disorder in comorbidity. However, it is unclear what the causal relationships are proposed to be within this model and there is no clear mechanism outlined by which ASPD traits may influence the development of dual diagnosis. The relevant mechanism could well be related to the management of emotional functioning, given that disturbed emotional functioning is a primary feature of all personality disorders (Gross & Levenson, 1997).

• The empirical support for secondary psychopathology models of comorbidity is weak. The general consensus is that it is more appropriate to conceptualise substance use as a stressor within a stress-vulnerability framework, than to view it as a causal factor in the development of psychosis.

• Several features of substance use in the SEMI population are consistent with the supersensitivity model. This model suggests there is not necessarily a higher level of substance use in this group but that a level of consumption which frequently leads to negative consequences in this very sensitive group and would not be expected to do so in the general population. This model is reasonably well supported by the data but does not address the question of why, given the adverse consequences, many individuals continue to use substances. It is important to examine motivations and psychological vulnerabilities that maintain the substance use in the context of adverse effects.
The self-medication hypothesis has a somewhat different emphasis in its application to comorbidity than with non-psychotic groups. Researchers have tended to formulate and test the hypothesis in terms of specific psychiatric symptoms being 'medicated' through substance use. The empirical support for this approach is generally weak (Mueser et al., 1998; Hussein Rassool, 2002; Blanchard et al., 2000). However, if a broader interpretation of the self-medication hypothesis is applied to this group, the central proposal is not that individuals are attempting to medicate specific psychiatric symptoms but distress. This formulation of the self-medication hypothesis is not inconsistent with an emotion regulation approach to understanding comorbidity and may relate to emotional dysfunction which precedes the development of psychosis, or emotional distress more directly associated with the manifestation of the illness.

Bi-directional models offer a promising and intuitively appealing approach but are currently rather underdeveloped and lacking in empirical applications.

Research is generally consistent with an alleviation of dysphoria model (Mueser et al., 1998). However, affect or emotion regulation models seek to develop these approaches further and consider how substance misuse may relate to the enhancement of positive emotions and how emotions may be regulated in a dynamic and active way, rather than simply alleviated.

A considerable body of research is directly or indirectly supportive to an emotion regulation model of comorbidity, drawing on research into substance misuse in non-psychiatric groups (e.g. Cooper et al., 1995). However, research thus far has tended to examine related constructs of personality traits, coping style and
temperament in both psychiatric and general population samples. The proposed mechanism of the effectiveness of emotion or affect regulation processes has not been directly assessed in groups with dual diagnoses. There is a need therefore for research to begin to explore emotion regulation processes in this population and assess whether there are significant differences between dual diagnosis and schizophrenia only groups which are consistent with an emotion regulatory function for substance misuse.
1.4 Emotion Regulation.

It is important at this stage to place the integrated affect regulation model developed by Blanchard et al. (2000) in the broader context of affect or emotion regulation. The field of emotion regulation has begun to emerge and develop as a distinct domain within psychological theory and research over the past few decades (Gross, 1998). However, elements of emotion regulation theory are embedded in most psychotherapeutic approaches (Cole et al., 1994) and have been around for considerably longer. There is a limited literature addressing emotion regulation in schizophrenia to date and no specific work was identified regarding emotion regulation in dual diagnosis groups, beyond that already outlined by Blanchard et al. (2000). The general concept and models of emotion regulation shall be reviewed before moving on to consider the current state of knowledge regarding emotional functioning in schizophrenia and dual diagnosis.

1.4.1 Defining emotion regulation.

Gross (1999) defines emotion regulation as:

how individuals influence which emotions they have, when they have them, and how they experience and express these emotions. (p.552).

identifying it as a broad construct covering a range of processes.

Thompson (1994) provides the following definition of emotion regulation:
The extrinsic and intrinsic processes responsible for monitoring, evaluating and modifying emotional reactions, especially their intensive and temporal features, to accomplish one’s goals. (p27).

Describing emotion regulation as goal oriented which draws attention to the essentially functional nature of emotion regulation, both in terms of achieving desired emotional outcomes and broader goals. Thompson (1994) also observes that emotion regulation involves processes both internal and external to the individual and identifies necessary precursors to effective emotion regulation; the ability to access and evaluate their emotions accurately.

Both positively and negatively-valenced emotions are subject to regulation which may lead to changes in various aspects of the emotions ‘dynamics’ such as latency, magnitude, duration, expression and behavioural responses (Gross, 1998) Emotion regulation is not a matter of simply increasing or decreasing the experience or expression of a particular emotion.

Emotion regulation theory draws on two main theoretical antecedents; psychoanalytic theory and coping theories (Gross, 1999; 1998). A central strand in psychoanalytic theory pertains to how individuals regulate aversive emotions, generally subsumed under the term anxiety, through the use of characteristic patterns of ego defences. Whilst emotion regulation is seen as of central importance in the development of
psychopathology, the current focus of emotion regulation theory is perhaps more normative than is typically true of psychoanalytic theory (Gross, 1999).

The development of theories of emotion regulation has also drawn heavily on the stress and coping tradition, particularly the concept of emotion-focused coping (Gross, 1999). Stress and coping theory has emphasised that individuals demonstrate a consistent ‘style’ of responding to stressors, which is echoed in emotion regulation theory (Gross, 1998). Also mutual is the concept of actively managing situational variables and emotional responses as a means to facilitate preferred outcomes or minimise adverse outcomes. Coping is considered to be a broader category which overlaps with but does not ‘entirely subsume’ emotion regulation theory, the two differing in important respects (Gross, 1999, p.556), including a more specific and discreet focus on emotional functioning in the latter, which does not always involve the individual’s resources being taxed (Gross, 1999).

Becoming able to effectively self-regulate emotion is a key developmental task which progresses through the dynamic interaction of factors intrinsic to the child with environmental and caregiver factors (Calkins, 1994). In infancy the child’s emotions are largely regulated by the responses of the caregiver but over the course of development self-regulation gaining ground (Calkins, 1994). This is thought to take place through a combination of the experience of one’s own emotions being responded to and managed by caregivers and learning how others modulate their own emotion experiences (Thompson, 1994) although the processes by which these gains are made are currently
poorly understood (Cole et al., 1994). Over the course of a child’s development an emotion regulation style is considered to become a stable individual characteristic and less amenable to change (Thompson, 1994).

Emotion regulation is likely to be optimal, defined in terms of causing no disruption to functioning (Thompson, 1994), when there has been a good ‘fit’ between the child’s emotional needs at different stages and the caregiver’s sensitivity and ability to meet these needs (Calkins, 1994). Emotion dysregulation is complimentarily defined as a pattern of emotion regulation which may serve a specific function but is inflexible and interferes with one or more domains of functioning, such as cognitive or social functioning (Cole et al., 1994). Developmental circumstances which make emotion dysregulation more likely are a consistent lack of appropriate caregiver intervention in situations which make emotional demands that outstrip the child’s ability to self-regulate (Cole et al., 1994). Where emotion dysregulation becomes a stable pattern it is considered to confer vulnerability to developing psychopathology (Cole et al., 1994), and indeed constitute a ‘common dimension of most categories of psychopathology’ including schizophrenia and personality disorders (Cole et al., 1994, p.77).

An important aspect of the development of the ability to self-regulate emotions is the development of beliefs and expectancies about one’s own and caregiver’s abilities to tolerate and modulate emotions (Calkins et al., 1994). These beliefs or internal models have a role in shaping future self-regulatory behaviour and interpersonal behaviour which relates to emotion regulation. Again the parallels with attachment theory are apparent.
Several ambiguities are inherent in the term emotion regulation that arise from the dialectical nature of the concept (Campos et al., 1994); emotions may be regulated or they may have a regulating function and indeed a complex interplay between the two will usually be the case. The following clarifications made by Gross (1999) shall also be adopted for the purposes of this study. Emotion regulation shall be used to refer to:

- The regulation of emotions.
- Self-regulation (although emotion regulation often takes place in a social context and an individual may use interpersonal resources the focus is on processes in which the individual is an active agent).
- A continuum of processes from those which are fully conscious and effortful to those which are automatic and non-conscious.

1.4.2 Models of emotion regulation.

Several theorists have proposed models which attempt to delineate the processes involved in emotion regulation. Eisenberg and Fabes (1995), for example identified three types of emotion regulatory process:

- Cognitive strategies- which alter the experience of an emotion through modifying the relevant interpretations.
- Behavioural strategies- which modify the behavioural response to the emotion.
- Situational strategies- which modify the emotion arousing event or situation in some way so as to act on the initial arousal of the emotion.

Gross and colleagues have taken the approach of distinguishing regulation processes in terms of the stage in the emotion generation process at which they exert their influence.
(Gross, 1998; Gross, 1999), rather than in terms of the type of resources the strategy uses. Gross and Munoz (1995) proposed two broad types of emotion regulation processes; those that are antecedent-focused and those that are response-focused. The former are attempts to influence the factors that elicit the emotion response tendency before it is fully initiated whilst the latter are efforts to modify the emotion dynamics once it has been elicited. The authors hypothesise that antecedent-focused approaches are likely to be generally more effective as they ought to modify both the experience and expression of the relevant emotion whilst response-focused approaches act only after the emotion is elicited and are therefore less likely to have a significant impact on an individual’s subjective experience of the emotion. Research has subsequently supported this view, particularly with respect to negative emotions where response-focused strategies, specifically expressive suppression, have been found not to significantly reduce subjective experience. With respect to positive emotions, however, expressive suppression does tend to lead to a reduction in the intensity of the emotion experience (Gross & Levenson, 1997). Response-focused strategies are nonetheless essential as individuals often need to constrain expression of an emotion either where antecedent-focused strategies are not possible or ineffective, or when there is no incentive to alter subjective experience of the emotion.

Gross (1998; 1999) developed this antecedent/response distinction to a more elaborate model of emotion regulation processes. Five groups of regulatory strategy are outlined and whilst the model is perhaps not yet very sophisticated, it offers a useful framework in a relatively young field (See Table 1).
Table 1: Summary of emotion regulation strategies from Gross (1998)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Emotion Regulation Process</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antecedent-focused</td>
<td>Situation Selection</td>
<td>A decision to approach or avoid a situation on the basis of an appraisal of its likely characteristics, the individual's likely responses and any costs/benefits involved.</td>
</tr>
<tr>
<td></td>
<td>Situation Modification</td>
<td>Active efforts to modify some aspects of the situation, such as costs, consequences and emotional impact. This concept is closely related to problem-focused coping. Situation modification may also be achieved through strategic emotional expression.</td>
</tr>
<tr>
<td></td>
<td>Attentional Deployment</td>
<td>Focusing attention away from the situation or on particular aspects of the situation to achieve certain emotion regulation goals. An individual may make use of distraction concentration or rumination.</td>
</tr>
<tr>
<td></td>
<td>Cognitive Change</td>
<td>The individual concerned may have made a preliminary appraisal of the meaning of the situation and their ability to cope but either or both of these may be open to modification through cognitive processes such as reappraisal, reframing or denial.</td>
</tr>
<tr>
<td>Response-focused</td>
<td>Response modulation</td>
<td>Modification of one or more aspects of the emotional response tendency. For example, relaxation techniques can be used to modify physiological arousal associated with anxiety or anger. Drugs, both prescribed and illicit, may be used to modify physiological aspects but also for their impact on affective and cognitive elements of an emotion. Various strategies may be used to control the expression of an emotion, drawing on cognitive and behavioural resources, behavioural inhibition being perhaps the most common form of response modulation.</td>
</tr>
</tbody>
</table>

Gross (1999) raises the issues of whether emotion can ever be understood as being unregulated and whether emotion generation and regulation are really separable processes. Indeed the generation and regulation of emotion as described are very heavily interwoven processes, however, Campos et al. (1994) consider it useful to distinguish the
two and point out that the consequences of emotional expression may differ from those
the individual anticipated and thus it may prove necessary to modify the manifestations
of an emotion over time, which can meaningfully be distinguished from initial emotion
generation. Gross (1999) suggests that the original query as to whether emotion is ever
not regulated is actually misleading in that it implies that emotion regulation is an all or
nothing process whereas assessing the degree of regulation may be more appropriate.

1.4.3 Functional emotion regulation.

The issue of whether particular emotion regulation strategies or styles can be understood
to be functional or dysfunctional also presents some difficulties. Although emotion
regulation in general is framed in functional terms, with this comes the possibility for
dysfunction within the systems. Gross (1998) and others (Thompson, 1994) consider that
no emotion regulation process can be understood to be universally functional or
dysfunctional as this is context dependent. Nonetheless, Thompson (1994) suggests that
if functionality is assessed in terms of outcomes, it may be possible to make some general
distinctions.

Phillips (2003) similarly proposes that it is possible to consider individual emotion
regulation strategies as generally functional or dysfunctional in terms of established links
with various forms of psychopathology and consequences. Phillips (2003) also makes
use of a distinction between emotion regulation strategies which indicate acceptance of
the emotion and the information it conveys, and those that represent a basic rejection of
the emotion and therefore a failure to harness any of the emotion’s functional value.
These methods for establishing functionality form the basis of the distinction between functional and dysfunctional emotion regulation processes in the Child and Adolescent Emotion Regulation Questionnaire (CAERQ) (Phillips, 2003), the only measure identified which assesses functionality.

### 1.4.4 Substance use as an emotion regulation strategy.

The research outlined above concerning individual’s self-reported reasons for and expectancies of substance use, both in general populations (Cooper et al. 1995) and dual diagnosis groups (Spencer et al., 2002) is consistent with understanding substance use as an emotion regulation strategy. Although this will not be the case for all consumption of psychoactive substances, based on these findings it seems likely that in many cases the aim of using substances will be to achieve an emotion-related goal. The short and longer-term consequences of this strategy will vary as a result of various contextual factors.

Substance misuse may become a form of emotion dysregulation in many individuals with schizophrenia. Emotion dysregulation, as outlined above, does not refer to a lack of regulation but to the use of strategies to achieve a particular emotion-related goal at the cost of adversely affecting other areas of functioning. When such a strategy becomes stable and inflexible, reinforced by the importance of the function it serves despite the broader context of costs, emotion dysregulation is deemed to be present. It is certainly possible to frame psychoactive substance misuse in schizophrenia in these terms.
Whether substance use can generally be classified as either an antecedent or response-focused emotion regulation strategy is currently unclear. This distinction would seem to depend on the specific context, particularly in terms of the individual's active emotional state and their aims for change. For example, taking a necessarily simplistic approach, the alleviation of existing negative emotions is in keeping with a response-focused strategy whilst attempting to elicit positive emotional experiences in the context of active negative emotions may more appropriately be considered antecedent-focused. However, it is likely that substance-use as a method of emotion regulation involves a complex interplay between the two forms of emotion regulation process which cannot be adequately represented in a simple dichotomous model.

Phillips (2003) classifies the use of substances as a dysfunctional emotion regulation strategy which uses resources internal to the person, in that substance use acts by altering biochemistry. Substance use is classified as dysfunctional in terms of its association with adverse psychological outcomes when used consistently. This positions substance use within the two by two conceptual model of emotion regulation strategies developed by Phillips (2003) where the dimensions are functionality and the origins of the resources involved.
1.4.5 Emotion regulation or affect regulation?

The terms emotion and affect are often used interchangeably in the literature (Ellgring & Smith, 1998). This may well have the effect of obscuring understanding but Ellgring and Smith (1998) suggest it is merely an indicator of the current stage of knowledge. Nonetheless it is possible to make some working distinctions for the purposes of this research.

Gross (1998), for example, uses affect as a super-ordinate term that covers various forms of valenced subjective states, including moods, emotions and feeling states which differ mainly in terms of their duration and specificity, although there is not a simple hierarchical relationship between them. Emotion is used to refer to subjective states such as anger, happiness and sadness which are relatively discrete and tend to be related to a specific object and lead to a particular response tendency (Gross, 1998). Emotion, being a more discreet level of subjective valenced state, offers a useful level of analysis for improving understanding of how subjective states are modulated and controlled by individuals with schizophrenia and those with dual diagnoses.

The present study has drawn on literature and theory that uses both the term affect regulation and emotion regulation. From reviewing the literature, theories of emotion regulation seem to be currently better articulated and operationalized in the form of measures. This provides a basis for beginning to examine these processes in individuals who experience schizophrenia and those who have dual diagnoses. Although the
integrative model proposed by Blanchard et al. (2000) uses the term affect regulation, what is specifically meant by this is not clarified. It is proposed to make use of the relatively well-articulated models of the regulation of emotion outlined above which will enable findings to be more meaningfully interpreted in this study. The term affect will be used, as defined by Gross (1998), as a broader term and also when referring specifically to the two scales of the Positive And Negative Affect Scales.
1.5 Emotional Functioning and Schizophrenia.

1.5.1 Historical perspective.

Historically emotional functioning has been neglected in the majority of theoretical and clinical approaches to understanding schizophrenia (Bentall, 2003; Ciompi, 1998; Birchwood, 2004). The legacy of Kraeplin’s approach to classification of the psychoses has been the conceptualisation of schizophrenia as an essentially cognitive disorder, manifested primarily in disturbances of thinking, whilst mania and psychotic depression have been viewed as primarily affective disorders (Ciompi, 1998). Although Bleuler subsequently attempted to place affective disturbances at the centre of our understanding of schizophrenia, theory, research and practice have continued to be substantially influenced by the Kraeplinian approach (Bentall, 2003; Ciompi, 1998), which is strongly reflected in current diagnostic criteria.

Diagnostically, the group of negative symptoms, such as affective flattening and anhedonia, places the greatest emphasis on emotional functioning. The extent to which diagnostic frameworks such as ICD-10 (WHO, 1992) and DSM-IV-TR (APA, 2000) attend to emotional functioning is fairly superficial, describing emotional experience as generally reduced or blunted and often involving a restricted capacity to experience pleasurable emotions.

Bleuler placed considerable emphasis on the role of affect in schizophrenia and proposed that the more immediately apparent features of psychosis, such as hallucinations and
delusions, were secondary or accessory features of the condition, the fundamental elements of which he considered to be:

- A loosening of associations between thoughts.
- The experience of ambivalent and conflicting emotions and attitudes.
- Social withdrawal and dominance of fantasy.
- The experience of inappropriate or incongruent affect.

(Bentall, 2003).

Several theorists have recently sought to highlight the role of emotion in the development and maintenance of schizophrenia (Bentall, 2003; Ciompi, 1998), considering that theoretical marginalisation of emotional functioning and a false separation of emotional and cognitive processes in schizophrenia has been detrimental to developing our understanding of the disorder. Indeed, Bentall (2003) seeks to 'redress this neglect and argue that psychotic symptoms are above all emotional phenomena' (p.205).

Ciompi (1998) approaches the question of whether a disorder of affect or of cognition is primary in schizophrenia from a different angle. Using the concept of ‘affect-logic’, which is intended to convey the interdependence of affective and cognitive functioning, Ciompi (1998) proposes that in psychosis the patterns of interaction between cognitions and affects are altered in important ways. He emphasises the important energizing, organizing and integrating role of affects on cognition in normal functioning. However, in psychosis it is proposed that these connections become too loose and therefore many of the usual advantages that result from interdependence are reduced. Ciompi (1998) acknowledges that the processes by which affects and cognitions interact are currently
poorly understood but the value of his contribution is the emphasis on the complex reciprocity between affective and cognitive processes.

1.5.2 Current knowledge of emotional functioning in schizophrenia.

Power and Dalgleish (1997) observed that theories of normal emotional functioning and emotional functioning in psychological disorders have tended to develop in parallel with little interaction between the two, to the detriment of both. Whilst there is a considerable literature on emotional functioning in various psychological disorders, schizophrenia has tended to be neglected for historical reasons outlined above (Bentall, 2003). However, research examining emotional functioning in schizophrenia is growing (Kring et al., 2003; Kring & Neale, 1996; Kring & Bachorowski, 1999). There is recent empirical support for the application of the Affective Circumplex model of the structure of subjective emotional experience, which was developed with the general population and is based on two dimensions of emotion experience; valence and intensity, to groups with schizophrenia (Kring et al., 2003). Whilst this research is currently limited, it serves to increase confidence that the structure of subjective emotional experience found in the general population may be appropriately used as a framework to examine emotional functioning in schizophrenia.

Amongst individuals diagnosed with schizophrenia there is a high frequency of affective disorders such as depression and anxiety, indicative of increased negative emotion experience (Bentall, 2003). These affective disorders are often in evidence before the onset of psychotic symptoms and their presence or exacerbation has been associated with
increases in positive psychotic symptoms, although interestingly not with a similar increase in negative symptoms (Bentall, 2003; Norman et al., 1998). Similarly, there is a well established link between an individual experiencing stress and increases in positive symptoms or clinical relapses (Bentall, 2003; Neale et al., 1998; Cole et al., 1994). Overall, although interpretation is limited by the correlational nature of the findings, the indications are strong that positive psychotic symptomatology is heavily influenced by an individual’s emotional functioning both during the initial onset and longer-term course of schizophrenia.

One example of this is the link between risk of relapse and high expressed emotion (EE) within the family. Numerous studies have demonstrated a significant association between expressed emotion by significant others, in the form of high levels of criticism and verbal hostility, and higher relapse rates (Buck et al., 1998). In terms of the mechanism by which high expressed emotion influences the course of illness, individuals with schizophrenia may be highly sensitive to the negative emotional expressions of others and it is likely that this exacerbates symptoms by increasing individual’s own levels of negative emotional arousal and reducing positive emotions (Buck et al., 1998).

Emotional experience and functioning are often assessed via outward expression and behaviour and subjective emotional experience is then inferred. The assumptions involved in such inferences are problematic. Indeed, whilst empirical findings confirm significant changes in the outward expression of emotion in schizophrenia, usually in terms of reduced expressivity (Kring et al., 1993), a growing body of research
consistently identifies a significant dissociation between expression and experience of emotions in schizophrenia (Kring et al., 1993; Kring & Neale, 1996; Buck et al., 1998). These findings are based on a research paradigm where observer ratings of expressive behaviour are compared to self-reports of emotional experience when watching emotionally arousing video-clips. Experience sampling techniques provide further evidence of this dissociation. Individuals with schizophrenia often report significant emotional content in their subjective experiences even when judgements based on observable expressive behaviours have been that their affective experience is significantly flattened or blunted (Neale et al., 1998; Bentall, 2003).

These findings of dissociation between expression and experience seriously undermine the validity of inferring blunted subjective emotional experience on the basis of reduced expression. Indeed, there are many indications that far from having reduced overall experience of emotions, individuals with schizophrenia report normal levels of subjective emotions in some situations and in some instances heightened levels, particularly of negative emotions (Neale et al., 1998; Buck et al., 1998). Furthermore, measures of physiological correlates of emotions, such as skin conductance and heart rate, are largely consistent with these findings, although there are some difficulties in interpreting findings due to baseline differences in these variables and the possibility that other aspects of the testing situation may lead to heightened arousal (Salem et al., 1996; Neale et al., 1998). Research has also confirmed that self-reported emotional experiences of individuals with schizophrenia are equally reliable as those of control groups (Neale et al., 1998).
This disjuncture between expression and experience of emotions identified by research studies is more consistent with the views of Bleuler whilst Kraepelin considered reduced expressive behaviour as an accurate reflection of underlying experience (Neale et al., 1998). The dissociation between the two has been confirmed in groups free from neuroleptic medications, controlling for the possibility that reduced expressive behaviour or changes to emotional experience that has been associated with these medications distorts the findings.

The implications of difficulty executing emotional expression are substantial, particularly in terms of social communication (Ellgring & Smith, 1998) even when it does not reflect an underlying reduction of emotional experience. In groups of individuals diagnosed with schizophrenia certain characteristic differences in expressive behaviour, which don’t equate to a simple reduction of expression, have been identified. A reduction in upper facial movements and a failure to follow the normal pattern of increasing expressive behaviour when taking on the role of speaker in a social interaction are amongst the subtle differences found (Ellgring & Smith, 1998) which may have a detrimental effect on social communication (Buck et al., 1998) and make the interaction less pleasurable, and possibly aversive, for the social partner (Ellgring & Smith, 1998).

Some theorists have hypothesised that these expressive differences may represent efforts (conscious or unconscious) to compensate for cognitive deficits which make the richness of information available in social encounters overwhelming for an individual with schizophrenia (Ellgring & Smith, 1998). Another proposal is that these differences may
be aimed at protecting the individual from the social partner’s emotional expressions, to which they may be particularly sensitive (Buck et al., 1998). However, the evidence on which to assess these alternative hypotheses is currently sparse and inconsistent and so they remain speculative.

There are also questions about the extent to which differences in emotion expression reflect underlying emotional dysfunction within the individual or the impact of iatrogenic factors such as impoverished environments and the effects of typical neuroleptic medications (Bentall, 2003), the latter being associated with neuroleptic dysphoria in a substantial minority of individuals treated with them (Voruganti et al., 1997). However, studies have confirmed the presence of significant differences in the expression of emotion in groups free of neuroleptic medications at the time of assessment (Neale et al., 1998).

Research has concluded that individuals with schizophrenia provide reliable self-reports of emotion experience using scales such as The Positive and Negative Affect Schedule (PANAS-Watson et al., 1988) (Kring & Neale, 1996; Buck et al., 1998). There is some variation in findings from studies which use the film clip paradigm in terms of how the emotion experiences of individuals with schizophrenia can best be characterised relative to those of control participants. Both Kring et al. (1993) and Kring & Neale (1996) found, with groups of individuals free from neuroleptic medications at the time of assessment, that there were no significant differences between the self-reported levels of positive or negative affect in the two groups in response to emotionally arousing film
clips. However, other studies have indicated some characteristic differences. For example, Neale et al. (1998) review findings of greater negative affect in response to both positive and neutral stimuli whilst Flack et al. (1998) described a difference in terms of the ‘signal to noise ratio’ where individuals with schizophrenia experience more ambiguous ratios of appropriate to inappropriate emotional responses than control groups, although the overall levels of emotion experience are comparable. These studies have all examined responses to controlled stimuli selected to illicit particular emotions. Considerably less is known about emotional responses in more naturalistic situations, which are likely to involve greater ambiguity and therefore present increased difficulty for an individual with schizophrenia if they experience a more ambiguous ratio of appropriate and inappropriate emotional responses.

Anhedonia, which can be defined as the reduced subjective experience of pleasure, has been demonstrated to be a relatively stable trait in schizophrenia using both clinical assessments and self-report (Blanchard, 1998). Empirical investigations have established that anhedonia is associated with both reduced positive emotion experience and also an increased level of negative emotion experience (Blanchard, 1998). However, there is more than one possible route to these experiences. Anhedonia may represent a deficit in the ability to experience positive emotions but equally it could result from changes in an individual’s tendency to approach activities and situations which may contribute to pleasurable experiences (Blanchard, 1998). Similar to reduced emotional expressiveness, this could be a by-product of an effort to cope with the level of stimulation in one’s environment through a reduction in activity and interaction, at the expense of reducing
opportunities to experience pleasurable emotions. Issues regarding impoverished environments and a lack of access to pleasurable activities are also relevant here. Research findings do indicate that even the most impaired individuals retain some capacity to experience pleasure in relation to particular activities, although their ability to initiate such activities may be compromised (Blanchard, 1998).

In summary, the emotional functioning of individuals with schizophrenia has only recently begun to receive substantial theoretical and research attention. However, on the basis of currently available knowledge, it is possible to identify several features which are divergent from normative emotional functioning. Individuals with schizophrenia demonstrate qualitatively different emotional expressive behaviour, including a tendency to express less positive emotion, more negative emotion and manifest some differences in executing facial expressions. Expression may also be dissociated from role within a social interaction. Additionally, individuals with schizophrenia often demonstrate a significant disjuncture between external expression of emotion and subjective experience in situations designed to elicit positive and negative emotions, which is not demonstrated by control groups who show congruence between these aspects of emotional experience. These findings challenge the assumption that reduced emotional expression represents a 'blunting' of underlying emotional experience. Subjective emotional experience can be characterised as generally involving normal or heightened levels of response to emotion-eliciting stimuli, although this is complicated by a more ambiguous ratio of appropriate to inappropriate emotions than is the case for control groups. The substantial proportion of individuals with schizophrenia who experience anhedonia typically encounter decreased
positive and increased negative emotions in the course of their normal experiences. However, it remains unclear to what extent this reflects deficits in the capacity to experience these emotions or a reduced tendency to access positive emotion-eliciting stimuli.

1.5.3 Emotion regulation and schizophrenia.

No empirical studies were identified which assessed emotion regulation strategies or processes in schizophrenia. The research summarised above has indicated that schizophrenia is often associated with increased experience of negative emotions and reduced positive emotions (Blanchard, 1998), and that in terms of the overall functioning of emotion systems, there is frequently a dissociation between the experience and expression of emotion (Kring & Neale, 1996). These findings may be the consequences of emotion regulation systems which function less effectively than is normative or may indicate increased demands made on emotion regulation systems in the context of schizophrenia that lead the systems to struggle. It is not possible to assess the validity of these alternative possibilities based on existing data.

In Ellgring and Smith’s (1998) analysis of affect regulation in psychosis both aspects of the dialectical construct of emotion regulation, the regulation of emotion and regulation by emotion, are discussed and it is often not entirely clear to which aspect the authors are referring. Nonetheless, they make some points which clearly have relevance to understanding the regulation of emotions in schizophrenia. They hypothesise that in psychosis internally generated stimuli become dominant and regulation of these internal
processes and associated emotions absorbs most of the individual's resources. This internal dominance disrupts the balance between internal and social stimuli at the expense of the regulation of social interaction and emotion regulatory processes that occur through social interaction.

In terms of qualitative differences in emotion-expression in schizophrenia (Kring & Neale, 1996), this could be understood in some instances as an attempt to regulate emotion rather than simply an expressive deficit (Buck et al., 1998). If expressive suppression is a more frequently used emotion regulation strategy in this clinical group, this could account for both changes in expressive behaviour and improve understanding of a common emotional profile of reduced positive emotion and increased negative emotion in schizophrenia. The use of expressive suppression as an emotion regulation strategy tends to diminish the subjective experience of positive emotions but not of negative emotions (Gross & Levenson, 1997) and may therefore exacerbate the dominance of negative emotional experience. If an over-reliance on response-focused emotion regulation strategies, such as expressive suppression, is associated with schizophrenia, this style of emotion regulation is less likely to be successful in alleviating negative emotions facilitating the generation of positive emotions, perpetuating a 'dysregulated' emotional system.
1.5.4 Emotional functioning and regulation in schizophrenia with comorbid substance misuse.

Whilst existing research has provided some support for conceptualising substance use in the context of schizophrenia as having an emotion regulatory function, specifically in terms of self-reported reasons for use (e.g. Spencer et al., 2002), there is a dearth of published work specifically examining emotional functioning and emotion regulation in dual diagnosis groups. The research reviewed above has identified several ways in which emotional functioning in schizophrenia differs from that of the general population in ways that are likely to confer a greater vulnerability to using substances for emotion regulatory purposes. However, what is not currently understood is whether individuals with dual diagnoses represent a subgroup of individuals who are more severely affected in terms of aversive emotional experiences or in terms of their ability to effectively self-regulate their emotional experiences. This is essentially what the present study aims to investigate.

1.5.5 How might difficulties in emotion regulation have developed in schizophrenia and dual diagnosis?

Theorists and researchers engaged in examining the development of self-regulation of emotions propose that effective and functional emotion regulation develops through an essentially interactive process involving child and caregiver traits with many parallels to the development of attachment relationships (Cole et al., 1994; Calkins, 1994). Buck et al. (1998) propose that problems in emotion regulation in individuals with schizophrenia
are likely to have resulted from the various forms of developmental adversity which characterise this group, and therefore can be understood as a precursor to, rather than a consequence of, developing schizophrenia, although dysregulation may become more entrenched as a consequence of the effects of developing schizophrenia. Indeed Cole et al. (1994) also propose that the development of emotion dysregulation constitutes a key vulnerability to the later development of various forms of psychopathology.

Birchwood (2003) identifies three pathways to emotional dysfunction in the context of psychosis; emotional dysfunction as intrinsic to the experience of psychosis, as an emotional reaction to traumatic experiences associated with psychosis, and as a pre-existing vulnerability resulting from the excess developmental adversity associated with these groups. Birchwood (2003) suggests that the pathways are in no way mutually exclusive and may hold varying relevance for different individuals. Based on these observations, it may be that different individuals in a dual diagnosis group manifest emotional dysfunction as a result of processes associated with their illness or as the result of deficits in the development of self-regulation. There may be variation within the group in terms of routes to emotion dysfunction or dysregulation which could result in differences in the nature and pattern of emotional dysfunction and any emotion regulation difficulties.
1.6. Rationale for the Present Study.

Excess vulnerability to misusing psychoactive substances and the associated adverse consequences are well established in individuals with a diagnosis of schizophrenia although the relevant psychological vulnerability factors are poorly understood. In order to develop effective treatments in this area it is essential that the psychological vulnerabilities involved in continued substance misuse in the context of serious negative consequences are better understood.

Based on the research reviewed it is reasonable to conceptualise substance use as having an emotion regulation function, at least for some individuals with comorbid psychosis. The excess vulnerability to substance misuse in schizophrenia can then be understood as arising from the association of the condition with increased aversive emotion experiences, which it is assumed increase the compelling nature of substance use. This approach is also borne out by findings that link personality traits associated with increased negative affectivity with the severity of substance use in a dual diagnosis sample. What is currently largely unknown is whether emotion and emotion regulation variables differentiate individuals with schizophrenia who have developed patterns of substance misuse from those who have not, which shall be the focus of the present research study.

Whilst an emotion regulation model of substance misuse in schizophrenia is considered to provide a potentially valuable explanatory framework for the excess vulnerability to
substance misuse in this group, the present research takes the view, as expressed in Mueser et al. (1998) that no one model is likely to offer a comprehensive account of this excess vulnerability but that several models will have a contribution to make to our understanding of this phenomenon.

This preliminary investigation is taking place in the broader context of growing attention to emotional functioning in schizophrenia (Bentall, 2003). Research and theory pertaining to emotion in schizophrenia reviewed above have identified several ways in which emotional experience and expression in this group is qualitatively different from the general population which may be due, at least in some part, to differences in emotion regulation. However, very little is known regarding the emotion experiences of comorbid groups and whether there are significant differences from non-comorbid groups (i.e. schizophrenia with no substance misuse).

In examining an emotion regulation theory of comorbidity it is important to address the following questions:

- Are individuals with dual diagnoses experiencing more aversive emotion states than those with schizophrenia only?
- Are individuals with dual diagnoses less effective at self-regulating their emotions?
- Do they demonstrate a different style of emotion regulation?
The key propositions of an emotion regulation model of comorbidity such as the integrative model developed by Blanchard et al. (2000) include:

1. Individuals with comorbid substance misuse and schizophrenia are vulnerable to experiencing elevated negative emotions and lowered positive emotions.
2. Individuals with comorbid substance misuse and schizophrenia are relatively ineffective at regulating their emotions.
3. That the severity or pattern of these difficulties will differentiate individuals with comorbid difficulties from those diagnosed with schizophrenia who do not misuse substances.

The aim of this research study shall be to begin to assess the validity of these propositions by measuring aspects of both emotion experience and emotion regulation and comparing these for two groups of individuals with diagnoses of schizophrenia, those where there is evidence of current substance misuse, and those where there is no such indication.

With regard to the proposition of a comorbid group being relatively ineffective in regulating their emotions, this requires some consideration in terms of how relative inefficacy might be operationalized. Previous research has indicated that response-focused emotion regulation strategies, are relatively ineffective in changing an individual’s experience of aversive emotions, as they come into play once the emotion has already been generated (Gross & Levenson, 1997). Indeed, this model indicates that substance use can itself be classified as a response-focused emotion regulation strategy. As a working hypothesis, a more heavily response-focused style of emotion regulation in dual diagnosis groups shall be examined. The only available measure which
operationalizes this antecedent/response-focused distinction uses expressive suppression as an example of a response-focused strategy, which shall be used to assess whether individuals with comorbid schizophrenia and substance misuse tend to use the relatively ineffective response-focused strategies more heavily.

Substance use has also been theoretically classified as a dysfunctional emotion regulation strategy which uses internal resources (Phillips, 2003). One might therefore hypothesise that for an individual who misuses substances this may be part of a broader pattern of dysfunctional emotion regulation strategies that use resources internal to the individual, as theorists have proposed that an individual develops a stable ‘style’ of emotion regulation (Thompson, 1994). Again there is only one measure available that operationalizes the relevant constructs regarding the functional status and nature of the resources used in the processes of emotion regulation which shall be used to examine these questions.
1.7 Research Question and Hypotheses.

The primary research question of this study can be summarised as follows:

Is there a significant difference in emotion regulation between groups of individuals with schizophrenia with and without comorbid substance misuse?

The research hypotheses are as follows:

1. Individuals with comorbid substance misuse will score lower on a measure of positive emotions and higher on a measure of negative emotions than individuals who do not misuse substances.
   
   **Null hypothesis 1:** There will be no significant differences between the comorbid and comparison groups in terms of scores on measures of positive and negative emotions.

2. Individuals with comorbid substance misuse will score higher on a measure of the use of expressive suppression as an emotion regulation strategy than individuals who do not misuse substances.

   **Null hypothesis 2:** There will be no significant differences between the comorbid and comparison groups in terms of scores on a measure of expressive suppression.
3. Individuals with comorbid substance misuse will score higher on a measure of dysfunctional emotion regulation strategies than individuals who do not misuse substances.

Null hypothesis 3: There will be no significant differences between the comorbid and comparison groups in terms of scores on a measure of dysfunctional emotion regulation strategies.

4. Individuals with comorbid substance misuse will score higher on a measure of internal emotion regulation strategies than individuals who do not misuse substances.

Null hypothesis 4: There will be no significant differences between the comorbid and comparison groups in terms of scores on a measure of internal emotion regulation strategies.
2. METHODOLOGY.

2.1 Design.
A quantitative methodology was adopted as most appropriate to the research question. The research used a between-groups design and was based on an opportunity sample of psychiatric out-patients. Individuals were allocated to groups on the basis of their responses to a self-report screening measure of current substance use which provided a comparison group; individuals with a diagnosis of schizophrenia whose current substance use status was abstinence or non-problematic (henceforth referred to as the Comparison group), and an experimental group; individuals who met criteria on the screening measure for current comorbid substance misuse (henceforth referred to as the Comorbid group). The decision was taken to classify groups on the basis of current substance use as it was believed this would be most strongly associated with measures of current emotions and emotion regulation and thus provide the most powerful design. All participants also completed self-report measures of affect and emotion regulation.

2.2 Power analysis.
A prospective power analysis was carried out to establish the number of participants required in each group to achieve the recommended power level of 0.8 (Clark-Carter, 2004). There were no available data which might indicate the expected effect sizes of the between-group differences on the relevant variables. Therefore, the effect size considered to be of interest was used in these calculations. This was set at a medium effect size of $d=0.6$ as medium to large between-group differences were considered to be
of sufficient clinical import. Based on these parameters, it was established using power tables in Clark-Carter (2004) that it would be optimal to have 45 participants in each group for the use of two-tailed between-subjects t tests in the analyses.

2.3 Participants.
All participants met standardised criteria (ICD-10) for schizophrenia or a related disorder (schizoaffective, schizophreniform etc) and were adult (18 years and over) out-patients of psychiatric services in West Fife and Dunfermline. All individuals were currently prescribed anti-psychotic medications. The decision was taken to exclude participants with diagnoses of other psychotic disorders, such as bipolar disorder, in order to enhance the clarity of any findings as there may be significant variation across diagnostic groups in terms of emotional functioning and the regulation of emotions.

Potential participants were identified through three routes:

1. Principally from the caseload of the Community Outreach Team (COT) - An assertive outreach team for individuals who have severe and enduring mental health problems with complex long-term support needs.
2. The Continuing Care Clinic - Out-patient psychiatry clinic for those with severe and enduring mental health problems.
3. The local psychiatric out-patient day hospital.
Table 2: Inclusion and exclusion criteria.

<table>
<thead>
<tr>
<th>Inclusion Criteria</th>
<th>Exclusion Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnosis of schizophrenia or related disorder (schizophreniform, schizoaffective etc) based on ICD-10 criteria.</td>
<td>Diagnosis of other psychotic disorder – i.e. Bipolar Disorder.</td>
</tr>
<tr>
<td>Currently in contact and being monitored by NHS psychiatric services as an outpatient.</td>
<td>Currently experiencing an acute psychotic episode/ inpatient admission.</td>
</tr>
<tr>
<td>Care manager/ psychiatrist/ senior nurse considers it appropriate to approach individual for participation in the study.</td>
<td>Care manager/ psychiatrist/ senior nurse considers it inappropriate to approach individual for participation in the study – for example in the case of recent bereavement or current serious physical ill health.</td>
</tr>
<tr>
<td>The individual is considered by staff to have a sufficient level of cognitive functioning to complete the questionnaires, either independently or with assistance from the investigator as a structured interview.</td>
<td>The individual is considered to experience a degree of cognitive impairment that would compromise their ability to respond reliably to the measures being used.</td>
</tr>
</tbody>
</table>

In total 129 suitable individuals were identified and invited to participate in the study. 40 of these individuals completed the study giving an overall participation rate of 31.01 per cent (although this varied considerably according to the service through which potential participants were identified – see Table 3). As a substantial proportion of the participants were recruited from the COT caseload, the sample is weighted towards those with more complex care needs. Sampling was not deliberate in terms of current substance-use status.
Table 3: Breakdown of identified individuals and participation rates by service.

<table>
<thead>
<tr>
<th></th>
<th>Community Outreach Team</th>
<th>Continuing Care Clinic</th>
<th>Day Hospital</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of appropriate individuals identified</td>
<td>54</td>
<td>49</td>
<td>26</td>
<td>129</td>
</tr>
<tr>
<td>N participated</td>
<td>25 (46.30%)</td>
<td>10 (20.41%)</td>
<td>5 (19.23%)</td>
<td>40 (31.01%)</td>
</tr>
<tr>
<td>N opted-out</td>
<td>7</td>
<td>8</td>
<td></td>
<td>21</td>
</tr>
<tr>
<td>N declined when contacted</td>
<td>9</td>
<td>12</td>
<td></td>
<td>21</td>
</tr>
<tr>
<td>N no longer appropriate (e.g. change in circumstances)</td>
<td>4</td>
<td>0</td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>N willing but not possible to accommodate</td>
<td>0</td>
<td>4</td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>N not contactable</td>
<td>8</td>
<td>15</td>
<td></td>
<td>23</td>
</tr>
</tbody>
</table>

Reasons for non-participation were not recorded by Day Hospital Staff.

The final sample of 40 participants was comprised of 9 females (22.5%) and 31 males (77.5%). The overall mean age of the participants was 40.85 years (SD=12.595). In terms of age and gender distribution the sample is very similar to that recruited by Blanchard et al. (1999). The sample is likely to be biased towards those who are relatively well engaged with services and active clinic attendees and individuals who are willing to reflect on and discuss their emotional experiences. Therefore the sample may represent a particular subgroup within the broader diagnostic group.
2.4 Measures and rationale for their selection.

In order to address the broad research question and specific hypotheses it was necessary to employ a screening measure of substance use which would allow individuals to be allocated to groups on the basis of whether or not they currently misuse substances. The research also calls for the application of at least one measure of emotion regulation and it was considered essential to measure levels of negative and positive emotional experience in order to establish whether these differed significantly between the groups, as this would have implications for how any findings in terms of emotion regulation might be interpreted and understanding what motivates specific efforts to regulate emotions.

2.4.1 Reliability of using self-report measures of emotional functioning with individuals diagnosed with schizophrenia.

Recent studies have shown that groups of psychiatric out-patients with diagnoses of schizophrenia are able to provide reliable and valid self-reports when using measures of anxiety and depression (Huppert et al., 2002). Additionally, research into emotional experience and expression in schizophrenia has made extensive use of the Positive and Negative Affects Scales (PANAS), and found participants diagnosed with schizophrenia and non-patient control participants to provide equally reliable responses (Kring & Neale, 1996). These findings indicate that individuals in these clinical groups are able to provide reliable reports regarding their subjective experiences of emotions using questionnaire measures. Recent research has also offered support for the reliability of self-reported substance use, when using a standardised measure, in this group, when
compared to clinician ratings or interviews (Kavanagh et al., 2002; Wolford et al., 1999). These findings support the adoption of a self-report methodology in the present study.

2.4.2 The DrugCheck – A brief screening instrument for substance abuse in psychosis (Kavanagh et al., 2000).

The DrugCheck is not yet generally available but was made available to the researcher for the purposes of the present research study by kind permission of Dr David Kavanagh of the University of Queensland, Australia.

The DrugCheck is a measure developed specifically to screen for current substance misuse in individuals with psychosis. It is a composite of established measures, the Severity of Dependence Scale (SDS) (Gossop et al., 1995) and the Alcohol Use Disorders Identification Test (AUDIT) (Saunders et al., 1993) both developed with the general population, and the addition of the Quantity and Frequency of Recent Substance Use (QF-RSU) assessment and the Problem List (PL).

The measure has been validated with samples of individuals with psychosis and cut-off scores established for identifying substance misuse using both the Problem List and AUDIT (Trembath, 2005) by validating the measure against diagnoses of misuse and dependence made using the ‘gold standard’ of the Composite International Diagnostic Interview (CIDI) (WHO, 1993). See Table 4 for a summary of the relevant statistics for the use of cut-off scores recommended by Trembath (2005). These established thresholds, scores on the PL and the AUDIT were used to classify participant’s current...
substance intake as misuse or not misuse. The unit content (or 'standard drink' on the DrugCheck) of alcoholic drinks was calculated using information from the Health Education Board for Scotland (HEBS, 1998).

Table 4: Summary of internal reliability, sensitivity and specificity data for the Problem List and AUDIT – SOS-I Validation Sample (N=50). Trembath (2005).

<table>
<thead>
<tr>
<th>Measure</th>
<th>Scale Reliability</th>
<th>Recommended Cut-Off (If &gt; indicates misuse)</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Correct Classification (Criterion of CIDI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Problem List</td>
<td>α=.89</td>
<td>1–any misuse</td>
<td>.94</td>
<td>.83</td>
<td>.90</td>
</tr>
<tr>
<td>AUDIT</td>
<td>α=.89</td>
<td>8-misuse/harmful</td>
<td>.94</td>
<td>.79</td>
<td>.84</td>
</tr>
<tr>
<td></td>
<td></td>
<td>12-dependence</td>
<td>.80</td>
<td>.80</td>
<td>.82</td>
</tr>
</tbody>
</table>

Prior to recent validation of the DrugCheck screening tool (Trembath, 2005), the use of the AUDIT with individuals with schizophrenia had already received some research support (Dawe et al., 2000). The measure was found to have good internal reliability (α=0.85), sensitivity (87%) and specificity (90%) of classification of alcohol misuse in this population. As yet there is no test-retest reliability data available for the Problem List nor was the researcher able to identify test-retest data for use of the AUDIT specifically with psychosis samples. However, there is good test-retest validity data for use of the AUDIT in general population samples (e.g.α=0.88 over a 6 week period, Trembath, 2005).

There is a need to use specifically developed and validated tools when screening for substance misuse amongst individuals who experience psychoses for several reasons. Firstly, as outlined in the introduction there is an excess vulnerability to substance misuse
in psychotic groups. Also, the misuse of substances by individuals with psychoses is associated with various negative outcomes both in terms of mental illness factors and psychosocial factors. Finally, and crucially, it has been demonstrated that individuals with psychoses experience significant adverse outcomes associated with the use of substances at much lower levels than would be the case in the general population and often at levels insufficient to result in physical dependency syndromes (Hussein Rassool, 2002). Therefore the indicators of misuse need to be set at a different level within this population.

Although the DrugCheck includes an assessment of both caffeine and nicotine intake, for the purposes of the present study these substances were not included in the assessment and classification of problematic use. If caffeine and nicotine had been included in the assessment of problematic substances it was considered that the screening tool would not adequately discriminate between individuals who clinically would be deemed to be demonstrating a problematic pattern of psychoactive substance use with implications for their mental health and psychosocial adjustment, and those who do not show such a pattern due to the high prevalence of nicotine and caffeine consumption in this population. Alcohol misuse was included in this assessment and indeed, the term substance misuse is used here to include alcohol misuse.

The authors recommend that when using the DrugCheck an appropriate alternative source to the participant, such as a friend, relative or a member of staff, also completes the Quantity and Frequency of Recent Substance Use (QF-RSU) assessment. However, in
the context of the present study this was not considered workable. Staff members who
identified potential participants were asked to indicate whether they were aware of any
substance misuse and asked to indicate the types, quantity and frequency involved, and
this was taken into consideration. Features of the study which facilitated accurate self-
report of substance intake include confidentiality (within professional guidelines) and a
non-punitive approach. Additionally, when an individual was asked about the quantity
and frequency that characterised their use, the researcher followed recommendations to
reduce the likelihood of minimizing by prompting with suggestions of high levels of
intake (Kavanagh et al., 2002).

Indeed, although it is generally recommended in the literature to corroborate the quantity
and frequency of self-reported use of substances with another source, this has been found
in some studies with this population to add little to the accuracy of self-report screening
(Kavanagh et al., 2002; Wolford et al., 1999) which has demonstrated the most accurate
assessment against criterion measures such as clinician ratings and structured diagnostic
interviews of all the alternative screening routes (Wolford et al., 1999). Wolford et al.
(1999) also found that it was difficult to identify and access appropriate collateral reports
when assessing psychotic groups. Urine screening, often considered a gold-standard in
substance-use screening, was found to be particularly prone to providing false-negative
results with this particular population (Wolford et al., 1999).

Only one other screening tool specifically designed to assess for the presence of
substance-use disorders in individuals with severe mental illness was identified in the
literature. The Dartmouth Assessment of Lifestyle Instrument (DALI) (Rosenberg et al., 1998) was developed using the most effective items from ten existing substance misuse screening instruments and has demonstrated high accuracy for classifying alcohol, cannabis and cocaine misuse. However, the DALI’s focus on the detection of alcohol, cannabis and cocaine misuse, as a result of its development in a US context where cocaine use is more prevalent, makes it perhaps less relevant to a UK context. The DrugCheck’s relevance and attention to a broader range of substances was considered a relative strength.

Although the DrugCheck does not include an assessment of past substance use, several supplementary questions were asked in the present study in order to gain some assessment of this. Participants were asked to describe their heaviest period of past consumption in terms of type, quantity and frequency of use. Although a full DrugCheck was not completed for past use, and so scores on the Problem List and AUDIT were not available for this assessment, a judgement was made by the researcher in terms of the pattern and quantity of consumption described, as to whether this was likely to represent misuse as operationally defined above. In general this judgement was not difficult as individuals were reporting patterns such as daily alcohol intake well in excess of the recommended level or, for example daily solvent inhalation. Staff reports of clinically noted periods of substance misuse were also taken into account when making these judgements. Indeed, most individuals who were later classified as meeting criteria for substance misuse at some point in their lifetime were identified from staff reports and clinical records as such and also reported themselves when asked about their previous
intake that their previous use had represented a problem for them. The categorisation of past use was relevant to a sub-sample of 8 individuals who did not meet criteria on the DrugCheck for current misuse. Where there was positive evidence of current misuse, an individual was automatically included in the lifetime misuse group in later analyses.

2.4.3 The Positive And Negative Affect Scales (PANAS): Watson, Clark & Tellegen (1988).

The PANAS is a brief self-report measure comprised of two 10-item scales measuring the dimensions of Positive Affect (PA) and Negative Affect (NA). The respondent uses a 5-point Likert scale to rate the degree to which they experience the specific states that comprise the scales. A total score is then derived for each scale (range 10-50). The PANAS can be used with several variations in terms of the time-period that respondents are asked to attend to in the instructions. For the present study the ‘how you feel in general’ instructions were adopted (See Appendix B). This was considered appropriate to gain a general measure of positive and negative emotions over a period of time likely to correspond best to the 3 month period over which substance use is assessed by the DrugCheck.

The PANAS has demonstrated good reliability and validity when used with non-clinical populations (Watson et al., 1988; Crawford & Henry, 2004), for whom comparison data is available. It has also been used widely in studies of emotional experience and expression in schizophrenia (e.g. Kring & Neale, 1996). Watson et al. (1988) also used the scale with a (N=61) sample of psychiatric inpatients, demonstrating good internal
reliability and providing some comparison data from a clinical population. See Table 5 for a summary of reliability coefficients for the two scales of the PANAS across various studies, some of which use different time period instructions, although Watson et al. (1988) found that the internal reliability of the scales seemed to be unaffected by the time period used. Test-retest reliability has also been demonstrated to be good in both undergraduate and psychiatric samples (Watson et al., 1988), as shown in Table 6 below.

Table 5: Summary of PANAS reliability coefficients.

<table>
<thead>
<tr>
<th>Study</th>
<th>Population Sampled</th>
<th>Instruction Used</th>
<th>N</th>
<th>Scale Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Watson et al. (1988)</td>
<td>Undergraduate university students (USA)</td>
<td>“in general”</td>
<td>663</td>
<td>Positive Affect</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>a=.88</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Negative Affect</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>a=.87</td>
</tr>
<tr>
<td>Watson et al. (1988)</td>
<td>Psychiatric inpatients (USA)</td>
<td>“in general”</td>
<td>61</td>
<td>Positive Affect</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>a=.85</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Negative Affect</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>a=.91</td>
</tr>
<tr>
<td>Jolly et al. (1994)</td>
<td>Psychiatric outpatients (USA) (excluding psychotic disorders)</td>
<td>“during the past week”</td>
<td>159</td>
<td>Positive Affect</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>a=.91</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Negative Affect</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>a=.89</td>
</tr>
<tr>
<td>Kring &amp; Neale (1996)</td>
<td>Individuals diagnosed with schizophrenia (USA) (DSM-IV)</td>
<td>“to what extent you feel this way right now” (In response to viewing mood-arousing film clips).</td>
<td>23</td>
<td>Positive Affect</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>a=.89</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Negative Affect</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>a=.91</td>
</tr>
<tr>
<td>Crawford &amp; Henry (2004)</td>
<td>General adult population (UK)</td>
<td>“during the past week”</td>
<td>1003</td>
<td>Positive Affect</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>a=.89</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Negative Affect</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>a=.85</td>
</tr>
</tbody>
</table>

Table 6: Test-retest reliability of the PANAS.

<table>
<thead>
<tr>
<th>Study</th>
<th>N</th>
<th>Population Sampled</th>
<th>Instructions Used</th>
<th>Interval</th>
<th>Positive Affect</th>
<th>Negative Affect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Watson et al. (1988)</td>
<td>101</td>
<td>Undergraduates (USA)</td>
<td>“in general”</td>
<td>8 weeks</td>
<td>.68</td>
<td>.71</td>
</tr>
<tr>
<td>Watson et al. (1988)</td>
<td>57</td>
<td>Psychiatric inpatients (USA)</td>
<td>“in general”</td>
<td>1 week</td>
<td>.79</td>
<td>.81</td>
</tr>
</tbody>
</table>
On the basis of its brevity, accessibility and research providing comparison and reliability data, the PANAS was selected as an appropriate measure of the experience of positive and negative emotions for the purposes of this study.
2.4.4 Measures of Emotion Regulation.

2.4.4.1 The Emotion Regulation Questionnaire (ERQ); Gross & John (2003).

The Emotion Regulation Questionnaire (ERQ) is a 10 item self-report measure of the use of two particular emotion regulation strategies; Cognitive Reappraisal (6 items) and Expressive Suppression (4 items) (See Appendix C). Cognitive Reappraisal is defined as 'construing a potentially emotion-eliciting situation in a way that changes its emotional impact' (p.349) and has been found to have an effect on both emotional experience and expression (Gross & John, 2003). Expressive Suppression is defined as 'a form of response modulation that involves inhibiting ongoing emotion-expressive behaviour' (p.349) and occurs only once the emotion is already active and has been found to impact mainly on emotional expression and not to be very effective in altering emotional experience (Gross & John, 2003). These two strategies were selected by the authors of the ERQ to represent both an antecedent and response-focused emotion regulation strategy which are both relatively well-defined and can be manipulated in research. Research has demonstrated the two dimensions to be largely independent from one another (Gross & John, 2003).

The respondent rates their agreement with each item using a 7-point Likert Scale (scored 1-7) in terms of how they respond to their emotions. Total scores for each dimension are then calculated; Cognitive Reappraisal (6-42) and Expressive Suppression (4-28). Research using the scales with non-clinical samples has demonstrated good internal
reliability (Reappraisal \(\alpha=.79\); Suppression \(\alpha=.73\)) and test-retest reliability (\(\alpha=.69\) over 3 months for both scales) (Gross & John, 2003). No research that has used this measure with clinical groups was identified and therefore the only comparison data available is in terms of scores for non-clinical groups.

Standardised self-report measures of emotion regulation or affect regulation have only begun to emerge in recent years and are currently limited in number. Available comparison data is restricted to that for non-clinical groups. Also, some of the available measures, such as the Trait Meta-Mood Scale (TMMS) (Salovey et al., 1995) are somewhat complex and abstract. After piloting both the TMMS and ERQ with a small number of respondents the ERQ was selected as it seemed to be more accessible for participants and less burdensome to complete due to its relative brevity. It also has the advantage of the strategies measured being derived from a relatively well developed process model of emotion regulation strategies which may facilitate interpretation of findings.

2.4.4.2 The Child and Adolescent Emotion Regulation Questionnaire (CA-ERQ) – Phillips (2003).

This measure was developed as a self-report measure of individual differences in emotion regulation for children and adolescents, motivated by the lack of existing measures. It is based on a conceptual model of emotion regulation strategies, derived from the literature, which classifies strategies in terms of a two by two structural model where the two dimensions represent the strategy’s functionality and whether the resources it makes use
of are internal or external to the individual (Phillips, 2003). The functionality dimension is determined on the basis of generalised outcomes of the use of a strategy and whether it represents a basic rejection or acceptance of the informative value of the emotion (Phillips, 2003). In cases such as substance use or changing eating patterns, the resources involved are categorised as internal on the basis that, although the substance or foods are external to the individual, the strategy operates on the basis of changes to the body’s internal biochemistry. Respondents use a 5 point Likert Scale (scored 1-5) to indicate the frequency with which they make use of the strategies as a response to experiencing emotions in general.

Items were derived from the literature and classified in terms of the model based on the consensus of a panel of experts, leading to a 32-item pilot scale which was refined to a 19-item scale as the result of item analysis, MAP analysis and factor analysis of the scale structures following completion of the scale by a sample of 351 children and adolescents. Confirmatory factor analysis, using the same sample, led to the adoption of a model that allowed the two functional and the two dysfunctional scales to co-vary, which provided an excellent fit to the data.

Table 7: Numbers of items in the CA-ERQ Subscales.

<table>
<thead>
<tr>
<th></th>
<th>Internal</th>
<th>External</th>
<th>Total Items</th>
</tr>
</thead>
<tbody>
<tr>
<td>Functional</td>
<td>5</td>
<td>4</td>
<td>9</td>
</tr>
<tr>
<td>Dysfunctional</td>
<td>5</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Total Items</td>
<td>10</td>
<td>9</td>
<td>19</td>
</tr>
</tbody>
</table>
Internal reliability was good for the child and adolescent validation sample (See Table 8). At the time of writing there were no coefficients available for the test-retest reliability of the scales.

Table 8: Internal reliability of the CA-ERQ (19 item) subscales based on validation sample.

<table>
<thead>
<tr>
<th>Subscale</th>
<th>N</th>
<th>α</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internal-Functional</td>
<td>351</td>
<td>.758</td>
</tr>
<tr>
<td>Internal-Dysfunctional</td>
<td>351</td>
<td>.716</td>
</tr>
<tr>
<td>External-Functional</td>
<td>351</td>
<td>.659</td>
</tr>
<tr>
<td>External-Dysfunctional</td>
<td>351</td>
<td>.757</td>
</tr>
</tbody>
</table>

It is recognised that use of a measure that has not yet been validated with general adult or adult psychosis populations places limitations on the interpretation of any findings. Nonetheless, in the context of a paucity of appropriate measures in the area of emotion regulation research and specifically in terms of the exploratory stage of applying such measures with clinical groups, the CA-ERQ is considered to be a valuable edition to the research design, particularly due to its unique consideration of functionality and the accessibility of the language and concepts.

The construct validity of the CA-ERQ was examined by Phillips (2003) by comparing scores on this new measure with several existing measures relating to emotional and behavioural functioning in children and adolescents; the Strengths and Difficulties Questionnaire (SDQ-Parent version) (Goodman, 1997), the Psychosomatic Complaints Scale (Adolescent sub-sample only) (Currie et al., 2001) and the Kidscreen measure (Ravens-Sieberer et al., 2001). Comparisons were also made with the Negative Emotion Coupling Questionnaire (Adolescent sub-sample only), also in development by the same
research group as the CA-ERQ, which examines the frequency with which individuals experience five specific negative emotions as coupled to each other.

The overall outcome of these analyses of the construct validity of the CA-ERQ was very favourable. A strong relationship was demonstrated between the use of dysfunctional emotion regulation strategies and the coupling of negative emotions. Total difficulties scores on the SDQ were significantly positively correlated with dysfunctional, and negatively correlated with functional emotion regulation scores. Higher scores for psychosomatic complaints were specifically positively correlated with the two dysfunctional scales on the CA-ERQ and greater subjective ratings of Health Related Quality of Life (as measured by the Kidscreen) were significantly negatively correlated with the dysfunctional scales of the CA-ERQ whilst being significantly positively correlated with the functional scales. In summation the findings are consistently supportive of good construct validity for the CA-ERQ subscales, increasing the confidence with which this measure can be applied in the present study.
2.5 Procedure.

Potential participants were identified by the relevant case manager based on the inclusion and exclusion criteria. These individuals were all then issued with a letter of invitation and an information sheet about the study (See Appendix R). This was issued by post for individuals identified through the outreach team and the clinic, and by staff at the day hospital.

Potential participants were given the opportunity to opt-out of the study by contacting either the researcher or a member of staff at the relevant service within a week of receiving the information. If an individual did not opt-out, the researcher then contacted them by telephone where possible in the case of outreach team and clinic patients, or they were contacted by day hospital staff when they attended the day hospital. Where it was not possible to contact an individual by telephone, efforts were made to arrange for a member of staff to speak to the individual during a visit to ascertain their willingness to participate. However, in some cases this also proved to be difficult.

When an individual was contacted and agreed to participate in the study, a mutually convenient meeting time was arranged, either at a clinic or, where this was not possible and a home visit was considered appropriate this was arranged. The majority of participants were able to complete the measures within a single session lasting between 15 minutes and 45 minutes. However, in the case of two individuals, the measures had to
be completed over the course of 3 and 4 sessions of approximately 15 minutes duration, due to their difficulty coping with longer sessions.

A standard procedure was followed in the research sessions. The participant was first given the opportunity to reread the information sheet and ask any questions they had about the study. If they were then happy to continue the consent form was completed and the measures were then completed in a standard order (See Table 9). The literacy of participants was assessed only informally in terms of staff or individual report. When staff identified an individual as meeting the inclusion criteria for the study they were asked by the researcher what their impression of the individual’s level of literacy was. Participants where asked about their reading and writing abilities before completing the measures and encouraged to ask for help if they had any difficulty. The PANAS, ERQ and CA-ERQ were completed by participants where they were able and as structured interviews in five cases where individuals reported significant reading difficulties. The DrugCheck was completed as a structured interview in all cases.

Table 9: Order of the administration of measures.

<table>
<thead>
<tr>
<th>Order</th>
<th>Measure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>PANAS</td>
</tr>
<tr>
<td>2</td>
<td>ERQ</td>
</tr>
<tr>
<td>3</td>
<td>CA-ERQ</td>
</tr>
<tr>
<td>4</td>
<td>DrugCheck</td>
</tr>
</tbody>
</table>

Participants were given a photocopy of their consent form and a copy was also sent to the relevant treating team together with a letter informing the team that they had participated in the study. The individual’s GP was also informed by letter that their patient had taken
part in the study. This procedure and documentation used all conformed to COREC guidelines. Participants also had the researcher's contact details on the information sheet in order that they could re-contact the researcher with any further queries.

2.6 Ethical Approval.

The study proposal was reviewed by the relevant Local Research Ethics Committee (See Appendix Q), which approved the research being carried out in the Fife area.
3. RESULTS.

3.1 Assessment of Sample Characteristics.

3.1.1 Age distribution of sample.

Table 10: Descriptive statistics for age.

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean</th>
<th>Std Dev</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td>All participants (N=40)</td>
<td>40.85</td>
<td>12.595</td>
<td>38.50</td>
</tr>
<tr>
<td>Comparison Group (N=20)</td>
<td>45.20</td>
<td>13.923</td>
<td>49.00</td>
</tr>
<tr>
<td>Comorbid Group (N=20)</td>
<td>36.50</td>
<td>9.589</td>
<td>35.00</td>
</tr>
</tbody>
</table>

The overall mean age for the participants in the study was 40.85 years (SD=12.595). There is a difference in the mean age of the two groups, the comparison group being older on average than the comorbid group (Table 10), the median difference is slightly larger. A t test (2 tailed), the assumptions for which were met, confirmed the mean age difference between the groups to be significant (t(38)=2.607, p<.05).

A series of bivariate correlations was then carried out to assess whether age was significantly related to the variables being measured and would need to be controlled in further analyses (See Appendix E for a summary). The correlation between age and Negative Affect (NA) score on the PANAS indicated an association, whilst not quite reaching statistical significance, of sufficient strength (r=0.303), to treat age as a covariate in further analysis of this variable.
3.1.2 Gender distribution of the sample.

As is summarised in Table 11, the sample as a whole was predominantly male, particularly in the comorbid group. The association between gender and substance use status was examined using Fisher’s exact test \((d.f.=1)\), due to low expected values in two cells making Chi-Squared unsuitable in this case. The association was not significant \((p>.05)\) and therefore it was not considered necessary to control for gender in further analyses of between group differences.

### Table 11: Gender composition of groups.

<table>
<thead>
<tr>
<th></th>
<th>Comparison Group</th>
<th>Comorbid Group</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>14</td>
<td>17</td>
<td>31</td>
</tr>
<tr>
<td>Female</td>
<td>6</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>20</td>
<td>20</td>
<td>40</td>
</tr>
</tbody>
</table>

3.1.3 Types of substance misused in the comorbid group.

Of the 20 participants identified on the DrugCheck measure as reaching criteria for current misuse of psychoactive substances 16 reported misusing alcohol and 4 reported misuse of alcohol and cannabis. Although the range of substances is narrow, it is consistent with those found to be most frequently misused in other research into comorbidity with UK psychiatric outpatients (Weaver et al., 2003).
3.2 Between Group Comparisons Based On Current Substance Misuse.

It was planned to use two-tailed between subjects t-tests subject to parametric assumptions being met. To reduce repetition, unless stated otherwise, the parametric assumptions for conducting a t test have been assessed and satisfied in each case.

Whilst all measures being used in this study are based on Likert scales, which strictly produce ordinal level data as the intervals between scores may not be equal, it is conventional in psychological research to treat such data as interval if there is a sufficiently large range of possible scores for the dimensions to be analysed (Clark-Carter, 2004). The assumption is therefore made that the scales have approximately equal intervals and the researcher should make sufficient checks to ensure that this assumption is not significantly contravened, such as by examining the distribution of the data. Given that all of the measures to be used have undergone validation studies and some processes of standardisation, the assumption of approximation to interval level data is considered very reasonable.

When assessing homogeneity of variance, a rule of thumb of a difference no greater than a factor of four, when the groups are equal in size, and a factor of two when the groups differ in size, was adopted as recommended in Clark-Carter (2004). Levene’s test for heterogeneous variances was also routinely examined as part of the SPSS output. Where variances were found to be significantly heterogeneous following these assessments but no other assumptions were broken, it was considered appropriate to use the version of the t test (Welch’s t) where equal variances are not assumed (Clark-Carter, 2004).
The normality of distributions was examined with the aid of histograms (See Appendix F) and conversion of indexes of skewness and kurtosis to z scores using the formulae in Clark-Carter (2004) and the recommended critical value of +/- 2.58 for significant deviation from normality (α=0.01) (See Appendix G). The presence of outliers on boxplots was also taken into consideration when assessing the distribution.
Table 12: Summary of descriptive statistics, t tests and observed power for between group comparisons based on current substance use status.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Subscale/ Score</th>
<th>Group</th>
<th>Mean</th>
<th>Std Dev</th>
<th>Median</th>
<th>t</th>
<th>d.f.</th>
<th>p value (2-tailed)</th>
<th>Effect Size</th>
<th>Observed Power</th>
</tr>
</thead>
<tbody>
<tr>
<td>PANAS</td>
<td>Positive Affect</td>
<td>Comparison Group</td>
<td>26.60</td>
<td>6.369</td>
<td>26.50</td>
<td>0.567</td>
<td>38</td>
<td>&gt;.05</td>
<td>d=0.18</td>
<td>P=.086</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(N=20)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group</td>
<td>25.45</td>
<td>6.452</td>
<td>24.50</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Negative Affect</td>
<td>Comparison Group</td>
<td>23.00</td>
<td>11.012</td>
<td>21.00</td>
<td>0.125</td>
<td>29.093</td>
<td>&gt;.05</td>
<td>d=0.032</td>
<td>P=.052</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(N=20)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group</td>
<td>23.35</td>
<td>5.905</td>
<td>22.50</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ERQ</td>
<td>Expressive</td>
<td>Comparison Group</td>
<td>16.35</td>
<td>5.950</td>
<td>16.00</td>
<td>0.952</td>
<td>37</td>
<td>&gt;.05</td>
<td>d=0.28</td>
<td>P=.153</td>
</tr>
<tr>
<td></td>
<td>Suppression</td>
<td>(N=20)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group</td>
<td>18.00</td>
<td>4.773</td>
<td>19.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cognitive</td>
<td>Comparison Group</td>
<td>24.85</td>
<td>7.829</td>
<td>25.50</td>
<td>0.177</td>
<td>37</td>
<td>&gt;.05</td>
<td>d=0.06</td>
<td>P=.053</td>
</tr>
<tr>
<td></td>
<td>Reappraisal</td>
<td>(N=20)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group</td>
<td>25.32</td>
<td>8.564</td>
<td>25.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CA-ERQ</td>
<td>Internal-</td>
<td>Comparison Group</td>
<td>13.55</td>
<td>3.332</td>
<td>13.00</td>
<td>0.791</td>
<td>38</td>
<td>&gt;.05</td>
<td>d=0.21</td>
<td>P=.12</td>
</tr>
<tr>
<td></td>
<td>Functional</td>
<td>(N=20)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group</td>
<td>12.85</td>
<td>2.134</td>
<td>13.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Internal-</td>
<td>Comparison Group</td>
<td>12.55</td>
<td>4.513</td>
<td>13.00</td>
<td>0.077</td>
<td>38</td>
<td>&gt;.05</td>
<td>d=0.022</td>
<td>P=.051</td>
</tr>
<tr>
<td></td>
<td>Dysfunctional</td>
<td>(N=20)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group</td>
<td>12.45</td>
<td>3.620</td>
<td>12.50</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>External-</td>
<td>Comparison Group</td>
<td>10.00</td>
<td>2.471</td>
<td>10.00</td>
<td>1.013</td>
<td>38</td>
<td>&gt;.05</td>
<td>d=0.364</td>
<td>P=.167</td>
</tr>
<tr>
<td></td>
<td>Functional</td>
<td>(N=20)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group</td>
<td>9.10</td>
<td>3.110</td>
<td>9.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>External-</td>
<td>Comparison Group</td>
<td>6.85</td>
<td>1.954</td>
<td>6.00</td>
<td>0.167</td>
<td>38</td>
<td>&gt;.05</td>
<td>d=0.051</td>
<td>P=.053</td>
</tr>
<tr>
<td></td>
<td>Dysfunctional</td>
<td>(N=20)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group</td>
<td>6.75</td>
<td>1.832</td>
<td>6.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
3.2.1 Hypothesis 1: Individuals with comorbid substance misuse will score lower on a measure of positive emotions and higher on a measure of negative emotions than individuals who do not misuse substances.

Scores on the PANAS were used to assess Hypothesis 1. The groups were contrasted in terms of total scores on both the Positive and the Negative Affect scale (See Table 12).

3.2.1.1 Positive Affect Scale (PA).

There are small differences in both the mean and median scores in the predicted direction of lower PA scores in the comorbid group (See Table 12). Figure 1 shows that whilst the difference between the two median scores is small, the larger inter-quartile range of the comparison group indicates greater within-group variation.

Figure 1: Boxplot of Positive Affect scores by current substance use group.
A between-groups t test did not approach significance (t(38)=0.567, p>.05). The effect size of the difference was small and the observed power of the test was very low.

3.2.1.2 Negative Affect Scale (NA).

Inspection of the descriptive data shown in Table 12 indicates very little difference between the two groups in terms of measures of central tendency on the Negative Affect scale. Figure 2 illustrates that, although the median scores are similar, the comparison group scores are distributed over a wider range overall and the inter-quartile range is substantially larger than that of the comorbid group. Figure 2 identifies 3 outliers in the comorbid group who fall either side of the group's narrower range of scoring. The greater within-group variation of the comparison group is also manifested in noticeably larger variance. These features indicate that looking only at measures of central tendency may be misleading in terms of the apparent similarity of the two groups. Whilst the comorbid group seem to be a relatively similar and cohesive group on this measure, there is considerable variation within the comparison group.
Levene's test showed the variances to be significantly different ($F(38)=14.197, \ p<.001$). Given that the normality of the distribution was adequate, Welch's $t$ was used (Clark-Carter, 2004) which confirmed the between-groups mean difference not to be significant ($t(29.093)=0.125, \ p>.05$). The effect size of this contrast was very small and the observed power was correspondingly low (See Table 12).
3.2.1.2.1 Analysis of age as a covariate for between-group differences in Negative Affect (NA) scores.

As noted above, although the correlation between age and Negative Affect score did not reach statistical significance, it was sufficiently strong (r=.303) to treat age as a covariate in analysis of this variable. An ANCOVA was conducted to provide an estimate of group mean scores for Negative Affect that could be expected if both groups were matched for the age of participants. Levene’s test for homogeneity of variance was significant (F(1,38)=7.320, p<.05) but given the relative robustness of ANOVAs under conditions where one of the relevant parametric assumptions is not fulfilled (Clark-Carter, 2004), this option was pursued. The result did not provide support for the assertion that, had the two groups been matched for age, there would have been a significant between-group difference detectable in terms of Negative Affect (F(1, 37)=.345, p>.05). However, the power of these analyses was also low and therefore the possibility of a Type II error is correspondingly high. Future research would benefit by taking account of these issues and matching groups for age.

3.2.1.3 Implications of analyses for Hypothesis 1.

There were no significant differences between the mean scores of the comparison and comorbid groups on either the Positive or Negative Affect Scale. As such the results do not support Hypothesis 1, which predicts that the comorbid group will show lower positive emotion and higher negative emotion. For both subscales there were small differences in the predicted directions but these did not approach significance.
Prospective power analyses had established that on the assumption of an effect size of 0.6 or greater, the desired number of participants was 45 in each group. This target was not met and given the small effect sizes the study has insufficient statistical power to be able to reliably reject hypothesis 1, confirmed by observed power analyses (see Table 12). It would be necessary to expand the research to include approximately 300 participants per group when examining differences in positive affect and in excess of 1000 participants per group when examining differences in negative affect to achieve power of 0.8.

One might question the clinical significance of group differences on these variables given the small effect sizes. However, the considerable variance within the comparison group may have been detrimental to the power of these analyses. This could indicate that the criteria for group allocation have resulted in individuals who differ in some important respect being included together in the no misuse group. The most obvious factor which may be relevant is past substance misuse in the absence of current substance misuse. Changes to the study design to take account of this may enhance the efficiency and power of the design.

Analysis of the association of substance use status and scoring for Negative Affect, treating age as a covariate, did not indicate that a genuine between-group difference had been obscured by difference in the age composition of the two groups. However, power was once again low, restricting reliable interpretation of this finding.
For a brief summary of comparisons of scoring on the PANAS in the present study to data from previous research see Appendix H.

3.2.2 Hypothesis 2: Individuals with comorbid substance misuse will score higher on a measure of the use of expressive suppression as an emotion regulation strategy than individuals who do not misuse substances.

Descriptive statistics for total scores on the Expressive Suppression subscale of the ERQ are presented in Table 12. The total scores for this subscale are based on summing the scores for 4 items and the possible range of scoring is 4-28. The number of participants in the comorbid group is 19 for this measure due to participant 24 declining to complete the measure.

The group mean total scores and median total scores show a difference in the predicted direction of higher scoring by the comorbid group, illustrated in Figure 3 together with the smaller overall and inter-quartile range of scores in the comorbid group, once again indicating greater variability within the comparison group.
A t test comparing group means did not approach significance ($t(37)=0.952$, $p>.05$). The effect size of the between-group difference, although in the predicted direction, was small ($d=0.28$) and observed power, was substantially below that desired ($P=0.153$). On the basis of the effect size found, approximately 300 individuals would be required in each group for sufficient statistical power (0.8) to be achieved.

3.2.2.1 Implications for Hypothesis 2.

In summary, there is insufficient support for Hypothesis 2; that individuals with current comorbid substance misuse will score higher than the comparison group for use of the emotion regulation strategy Expressive Suppression. However, due to the small effect...
size and insufficient statistical power observed, nor is not possible to reliably reject the research hypothesis in favour of the null hypothesis on the basis of these findings. A considerably larger study is necessary to achieve sufficient power and the small effect size may be prohibitive in terms of sustained interest in group differences on this variable. It is notable though, that as in the analysis of between-group differences on the PANAS above, the comparison group again shows greater within-group variation than the misuse group which may indicate that the design lacks efficiency in terms of grouping criteria. Further consideration needs to be given to how individuals are classified as these within-group differences may be obscuring relevant between-group differences.

For a summary of comparisons of scoring on the ERQ in the present study to data from previous research see Appendix I.

3.2.2.2 Analysis of Scoring on the Cognitive Reappraisal Subscale of the ERQ.

Although no specific hypotheses were formulated with regard to between-group differences in scoring on the Cognitive Reappraisal subscale of the Emotion Regulation Questionnaire, given the early stage of research in this area it was considered valuable to examine this subscale. Inspection of the descriptive statistics shown in Table 12 indicates very little difference between the two groups on this subscale in terms of measures of central tendency. Variances were also very similar, and a between-subjects t test (2 tailed) did not identify a significant difference between the group means, although once again the power of this comparison (P=.053) is significantly below optimal and therefore
the possibility of a real difference cannot be reliably dismissed. The similarity of the two groups is further illustrated in Figure 4 below.

Figure 4: Boxplot of scores on Cognitive Reappraisal Subscale of the ERQ.

![Boxplot of scores on Cognitive Reappraisal Subscale of the ERQ.](image)

The measures of central tendency indicate that both groups tended to score the Cognitive Reappraisal items neutrally on average. Once again, although it is not possible to reliably discount the presence of a significant difference due to the small effect size and lack of sufficient statistical power, the small effect size indicates that a between group difference on this measure is unlikely to be of clinical significance to dual diagnosis.
3.2.3 Hypotheses 3 & 4: Individuals with comorbid substance misuse will score higher for dysfunctional emotion regulation strategies and internal emotion regulation strategies than individuals who do not misuse substances.

3.2.3.1 Examination of the structure of the Child and Adolescent Emotion Regulation Questionnaire (CA-ERQ).

Although it is beyond the scope of this investigation to conduct a full validation of the CAERQ with this population, it is necessary to briefly assess whether the structure of the subscales has been adequately retained to allow further analyses to be confidently interpreted (See Appendix J for a full summary of these analyses). This rudimentary analysis using Pearson’s correlations allowed the researcher to be confident that the subscales had retained a good level of discriminant validity. The two functional (internal and external) subscales were significantly correlated, although the coefficient was relatively low (r=.362) and indeed, these two subscales were also found to correlate in the original development sample and the model of the four sub-types of emotion regulation strategy adopted following factor analysis allows for them to co-vary. Based on these brief analyses it was considered reasonable to proceed with subscale analyses.

3.2.3.2 Internal-Functional Scale.

Measures of central tendency for scoring on the Internal-Functional subscale are very similar for the two groups (See Table 12) and this is further illustrated by Figure 5 below. However the comparison group demonstrates somewhat greater variation in scoring, although Levene’s test for heterogeneous variances is not significant in this case. A between-groups t test (2 tailed) is consistent with this similarity of means and does not
identify a significant difference ($t(38)=.791, p>.05$), although the power is too low to reliably reject this possibility. With such a small effect size, the study would require to be expanded to include approximately 400 people in each group to provide sufficient statistical power.

Mean totals for both groups, when divided by the number of items in the scale suggest a typical response of ‘seldom’ to ‘often’ for the use of this group of emotion regulation strategies.

**Figure 5: Boxplot of total scores on the Internal-Functional scale of the CA-ERQ.**

![Boxplot of total scores on the Internal-Functional scale of the CA-ERQ.](image)

### 3.2.3.3 Internal-Dysfunctional Scale.

As with the previous scale, group mean total scores, and indeed median scores are also very similar for the internal-dysfunctional scale. However, Figure 6 below indicates a
somewhat greater weighting for the comparison group towards lower scoring on this particular scale. Once again a between-groups t test (2 tailed) does not identify a significant difference ($t(38)=.077, p>.05$) but the power of the analysis is insufficient to reliably reject the existence of a genuine difference. With the current effect size it would be necessary to have in excess of 1000 participants in each group to achieve optimum power, undermining the likely clinical significance of between group differences on this variable. Also similarly to the previous scale group mean total scores indicate that, for both groups, respondents are likely to be making typical item responses of 'seldom' to 'often' for use of this group of strategies.

Figure 6: Boxplot of total scores on the Internal-Dysfunctional scale of the CA-ERQ.
3.2.3.4 External-Functional Scale.

On this particular scale there is a small difference between group means in keeping with the hypothesised differences; the comparison group mean being slightly higher on a functional scale. This is illustrated by Figure 7 below. Nonetheless, although the effect size is somewhat greater than for the other CA-ERQ subscales considered so far, it remains small and the between-groups t test did not produce a significant result ($t(38)=1.013$, $p>.05$). The observed power is insufficient to permit reliable rejection of a genuine group difference on this variable and expansion of the study to 180 participants per group would be required to reliably avoid a Type II error.

Mean total scores on this subscale are slightly lower due to there being one fewer item. However, as before they still indicate an average item response, for both groups, of between ‘seldom’ and ‘often’.

Figure 7: Boxplot of total scores on the External-Dysfunctional scale of the CA-ERQ.
3.2.3.5 External-Dysfunctional Scale.

The group mean total scores for this final subscale of the CA-ERQ are noticeably lower than those for previous subscales, including the shorter 4 item External-Functional subscale. Measures of central tendency are also very similar for both groups, further illustrated in Figure 8 below. As expected therefore, a between-groups t test does not identify a significant difference between group mean totals ($t(38)=.167$, $p>.05$), although owing to a very small effect size and low observed power (See Table 12), once again it is not possible to reliably reject the presence of a genuine difference between the two groups. Indeed, in excess of 1000 participants would be needed in each group in order to attain sufficient statistical power should the design remain unchanged.

Figure 8: Boxplot of total scores on the External-Dysfunctional subscale of the CA-ERQ.
It is interesting to observe that the group mean total scores equate to an average item response of ‘never’ to ‘seldom’ for items on this scale for both groups, whilst on the other three subscales this has equated to scoring between ‘seldom’ and ‘often’ for each item. This difference is further illustrated by Figure 9 below. It is notable that the same pattern of lower scoring on the External-Dysfunctional scale is demonstrated by both groups.

Owing to the lack of comparison data from other adult samples it is not clear whether this profile is particularly characteristic of individuals with schizophrenia or perhaps represents a normative profile. Indeed, relatively low scoring on a dysfunctional subscale could be interpreted as an encouraging finding. The fact that lower scoring is specific to the External-Dysfunctional scale and is not also found on the Internal-Dysfunctional scale is of particular interest.

It seems clear from Figure 9 in particular that the hypothesised relatively higher scoring for internal and dysfunctional emotion regulation strategies amongst individuals with comorbid substance misuse has not been borne out in this study. When the four subscales are compared (after dividing group mean totals by the number of items in order to make all subscales comparable), there is relative consistency between groups and across three subscales with a clear dip in scoring for both groups on the external-dysfunctional scale. Also, the two groups seem to be particularly closely matched on both the ‘dysfunctional’ scales whilst the comparison group show slightly higher scoring (although this did not attain significance here) on both the ‘functional’ subscales. It is particularly interesting
that, as the groups are currently composed, this scoring profile is very similar for both groups.

**Figure 9: Group mean total scores for the four CA-ERQ subscales divided by the number of items in the scale.**

3.2.3.6 Implications Of Analyses For Hypotheses 3 and 4.

3.2.3.6.1 Hypothesis 3: Dysfunctional Scales.
For both subscales measuring the use of dysfunctional emotion regulation strategies (Internal-Dysfunctional and External-Dysfunctional) there was no significant difference identified between the mean total scores for the two groups. Indeed, although the inadequate statistical power means that reliable rejection of this possibility is not possible on this basis, the group means were highly similar and the effect sizes were correspondingly very low. Therefore, on the basis of these analyses there is insufficient support for the retention of Hypothesis 3, that the substance misuse group will score more
highly for dysfunctional emotion regulation strategies, although reliable interpretation of these findings is limited by the low statistical power involved. For both subscales, on the basis of the effect sizes found, the study would need to be expanded to include in excess of 1000 participants in each group to allow for reliable rejection of the hypothesis. A re-evaluation of the study design to increase power would seem a more efficient approach.

3.2.3.6.2 Hypothesis 4: Internal Scales.

Similarly, no significant differences were identified between the two groups for either of the scales measuring the use of internal emotion regulation strategies (Internal-Functional and Internal-Dysfunctional). Once again, inadequate statistical power means that on the basis of these findings it is not possible to reliably dismiss the hypothesis that individuals with comorbid substance misuse make greater use of internal emotion regulation strategies. The study would need to be expanded to a size that is unachievable as a single-site study and the appropriate way forward to improving the power of the study seems to be to re-evaluate the existing design.
3.2.4 Summary of findings for planned between-group comparisons.

The planned analyses carried out have not provided sufficient support for any of the hypothesised between-group differences. However, all the analyses have also had insufficient statistical power to be able to reliably accept the relevant null hypothesis which restricts the conclusions that can be drawn. In some cases there were small mean differences in the predicted direction, two of which showed small effect sizes which may be particularly worth examining further in a larger or more efficient research project these being differences on the Positive Affect scale of the PANAS, the Expressive Suppression scale of the ERQ and the Internal and External-Functional scales of the CA-ERQ.

Across the planned analyses conducted, the Comparison group have consistently demonstrated greater within-group variation in scoring on the different measures than the Comorbid group, sometimes significantly so. This may indicate that genuine differences between the two groups are being obscured by this level of within-group difference. It may be that a re-examination of the grouping criteria could enhance the power of the study design.
3.3 Exploratory Analyses.

3.3.1 Re-examination of grouping criteria.

The preceding analyses, based on group comparisons in terms of current substance misuse status, have not identified any significant between group differences other than in variance and although the observed power is low in all cases, the effect sizes involved are not encouraging. The issue of whether any group differences may have been obscured and the power of the design reduced by the grouping criteria was considered to merit further consideration.

The comparison group included several individuals who had identified themselves and been identified by staff as having misused substances in the past, but currently were either abstinent or using substances in a non-problematic way as measured by the DrugCheck. The comparison group have consistently shown greater within-group variation in scoring than the current comorbid group. There may be an underlying bi-modal distribution within this group due to the presence of two distinct groups; those who have never misused substances and those that have misused substances in the past. If there are genuine group differences in emotion regulation in relation to substance misuse, these may persist beyond the cessation of misuse and represent enduring individual differences.

To evaluate the validity of reclassifying the groups in terms of lifetime substance misuse (i.e. never misused vs. ever misused), the participants were recoded into ‘never’ (N=12),
‘past’ (N=8) and ‘current’ (N=20) substance misuse groups on the basis of self and staff-reports of past use. Descriptive statistics, boxplots and histograms were employed to examine the distributions and assess whether the past misusers bore greater similarity to the current misusers than to those who had never misused substances (See Appendices K, L & M for a summary).

On the majority of the variables measured the ‘past’ and ‘current’ groups were more similar in terms of measures of central tendency. Indeed, on both of the PANAS subscales the ‘past’ misuse group mean scores were the lowest overall, with the ‘current’ group scores being intermediate between the ‘past’ and ‘never’ groups. This supports the view that it was inappropriate to classify the ‘past’ and ‘never’ groups together. Examination of scoring on the Expressive Suppression subscale of the ERQ indicated greater similarity in the distribution of scores between the ‘past’ and ‘current’ groups whilst for the Cognitive Reappraisal subscale the picture was less clear.

For the subscale scores on the CA-ERQ, in terms of measures of central tendency, for three of the four subscales the ‘past’ and ‘never’ groups are the most dissimilar. Inspection of the boxplots for the three groups provides a more mixed picture (See Appendix L. For two subscales (Internal-Functional and External-Dysfunctional) the distributions for the ‘past’ and ‘current’ groups do indeed appear to be more similar than the ‘past’ and ‘never’ groups. However, for the External-Functional and Internal-Dysfunctional subscales it is less clear whether this is the case.
A series of t tests were then used to assess whether there were any significant differences between the 'current' and 'past' misuse groups which would be contraindicative to treating them as one group in further analyses (See Appendix N). The assumption of homogeneity of variances was not met in one case and Welch's t was used to take account of this. None of the contrasts were significant (two-tailed). However, all comparisons also showed very low observed power, partially due to the differences in the group sizes, and therefore the possibility of the group means being different cannot be reliably rejected. Nonetheless, for the current exploratory purposes, it was considered reasonable to proceed with comparisons between two groups based on lifetime criteria for substance misuse; those for whom there was no indication of current or past substance misuse, hereafter referred to as comparison group B (N=12) and those for whom there was indication of either current or past substance misuse, hereafter referred to as comorbid group B (N=28).

Individuals were still generally classified into these groups on the basis of self-report. When completing the DrugCheck the researcher routinely asked for a brief summary of past substance use, however, in one case (id 8) where staff had reported past diagnosis of abuse but the participant had not provided reports consistent with this on the DrugCheck, the individual was classified on the basis of staff-report. It is recognised that the basis for the re-grouping is less stringent than that for the original classifications, but given the exploratory nature of these post-hoc analyses, this is considered reasonable.
3.3.2 Assessment of group characteristics.

3.3.2.1 Age and gender.
On the basis of the new ‘lifetime’ grouping criteria the group numbers are now uneven. The association between gender and substance use group, female participants being proportionally fewer in the substance misuse group, was not statistically significant as assessed by Fisher’s exact test (p>.05), although this was only the case by a narrow margin. Therefore it was considered prudent to examine gender as a covariate in later analyses. Also, there remained a significant difference between the group mean ages (t(38)=2.607, p<.01), the mean age of the comparison group B being 48.25 (SD=11.717) and that of the comorbid group B being 37.68 (SD=11.766). However, as it had been established earlier (See Appendix E) that age was not significantly related to any of the other measured variables and the relationship between age and Negative Affect, which was of sufficient strength to warrant further attention although not achieving statistical significance, has already been examined, it was not considered necessary to control for between-group age differences in further analyses.

Table 13: Gender distribution of sample – Lifetime criteria.

<table>
<thead>
<tr>
<th></th>
<th>Comparison Group B</th>
<th>Comorbid Group B</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>7</td>
<td>24</td>
<td>31</td>
</tr>
<tr>
<td>Female</td>
<td>5</td>
<td>4</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>12</td>
<td>28</td>
<td>40</td>
</tr>
</tbody>
</table>
3.3.2.2 Types of substances used.

Table 14: Types of substance reported to be currently misused or misused in the past.

<table>
<thead>
<tr>
<th>Type of Substance</th>
<th>Alcohol</th>
<th>Cannabis</th>
<th>Solvents</th>
<th>LSD Type</th>
<th>Stimulants and Amphetamines</th>
<th>Opiates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>26</td>
<td>13</td>
<td>1</td>
<td>3</td>
<td>9</td>
<td>2</td>
</tr>
</tbody>
</table>

Total frequencies in Table 14 exceed 28 as several individuals reported current or past poly-substance use. With the exception of 2 individuals, alcohol misuse was ubiquitous and in 14 of 28 cases it was the only misused substance identified. Figure 10 further illustrates the frequency of misuse of the different groups of substances. These findings are in keeping with two consistent findings regarding substance use in schizophrenia, that alcohol and cannabis are the most commonly misused substances (Weaver et al., 2003) and that poly-substance use is also common (Blanchard et al., 2000). It is also clear that a broader range of substances were misused in the past than are currently (alcohol and cannabis only).

Figure 10: Bar graph showing frequencies of types of substance used.
Table 15: Summary of between-group comparisons using t tests – Groups based on lifetime criteria.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Subscale/Score</th>
<th>Group</th>
<th>Mean</th>
<th>Std Dev</th>
<th>Median</th>
<th>t</th>
<th>d.f.</th>
<th>p value (2 tailed)</th>
<th>Effect Size</th>
<th>Observed Power</th>
</tr>
</thead>
<tbody>
<tr>
<td>PANAS</td>
<td>Positive Affect</td>
<td>Comparison Group B (N=12)</td>
<td>28.58</td>
<td>6.33</td>
<td>29.00</td>
<td>1.707</td>
<td>38</td>
<td>&lt;05</td>
<td>d=0.58</td>
<td>P=0.384</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group B (N=28)</td>
<td>24.93</td>
<td>6.15</td>
<td>23.50</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Negative Affect</td>
<td>Comparison Group B (N=12)</td>
<td>24.50</td>
<td>11.23</td>
<td>25.5</td>
<td>0.534</td>
<td>15.474</td>
<td>&gt;05</td>
<td>d=0.17</td>
<td>P=0.093</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group B (N=28)</td>
<td>22.61</td>
<td>7.58</td>
<td>22.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ERQ</td>
<td>Expressive Suppression</td>
<td>Comparison Group B (N=12)</td>
<td>14.50</td>
<td>5.870</td>
<td>13.50</td>
<td>2.139</td>
<td>37</td>
<td>&lt;05</td>
<td>d=0.65</td>
<td>P=0.549</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group B (N=27)</td>
<td>18.33</td>
<td>4.836</td>
<td>19.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cognitive Reappraisal</td>
<td>Comparison Group B (N=12)</td>
<td>25.00</td>
<td>8.914</td>
<td>26.50</td>
<td>0.039</td>
<td>37</td>
<td>&gt;05</td>
<td>d=0.01</td>
<td>P=0.05</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group B (N=27)</td>
<td>25.11</td>
<td>7.876</td>
<td>25.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CA-ERQ</td>
<td>Internal-Functional</td>
<td>Comparison Group B (N=12)</td>
<td>14.83</td>
<td>3.380</td>
<td>14.00</td>
<td>2.200</td>
<td>15.164</td>
<td>&lt;05</td>
<td>d=0.689</td>
<td>P=0.718</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group B (N=28)</td>
<td>12.50</td>
<td>2.203</td>
<td>13.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Internal-Dysfunctional</td>
<td>Comparison Group B (N=12)</td>
<td>13.83</td>
<td>4.260</td>
<td>14.50</td>
<td>1.383</td>
<td>38</td>
<td>&gt;05</td>
<td>d=0.446</td>
<td>P=0.271</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group B (N=28)</td>
<td>11.93</td>
<td>3.877</td>
<td>12.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>External-Functional</td>
<td>Comparison Group B (N=12)</td>
<td>10.25</td>
<td>2.417</td>
<td>11.00</td>
<td>1.032</td>
<td>38</td>
<td>&gt;05</td>
<td>d=0.414</td>
<td>P=0.172</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group B (N=28)</td>
<td>9.25</td>
<td>2.952</td>
<td>9.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>External-Dysfunctional</td>
<td>Comparison Group B (N=12)</td>
<td>7.08</td>
<td>1.832</td>
<td>6.50</td>
<td>0.632</td>
<td>38</td>
<td>&gt;05</td>
<td>d=0.218</td>
<td>P=0.093</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid Group B (N=28)</td>
<td>6.68</td>
<td>1.906</td>
<td>6.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
3.3.3 Re-examination of PANAS Scores.

Following group reconfiguration there was a clear difference between the group mean scores for Positive Affect in the direction of lower scores in the comorbid group B (See Table 15). A t test confirmed this difference between group means to be statistically significant \((t(38)=1.707, \, p<.05)\) with a medium effect size \((d=0.58)\).

Turning to the Negative Affect scale, there was a small difference between the group mean scores in the opposite direction to that originally predicted, that the comorbid group would score more highly than the comparison group. Due to the group variances differing by slightly more than the recommended factor of two (Clark-Carter, 2004), Welch’s t was used and did not demonstrate a significant difference \((t(15.474)=0.534, \, p>.05)\). However, although slightly improved relative to initial analyses, the analysis had insufficient statistical power \((P=.093)\), due to the small effect size \((d=0.17)\) and uneven groups involved and therefore it is not possible to reliably reject the possibility that there may be an underlying difference between these groups in terms of negative affect based solely on these analyses. A sufficiently powerful study would require approximately 400 participants in each group to be able to reliably make this discrimination.
3.3.4 Re-examination of ERQ Scores.

Inspection of the new group mean scores for the Expressive Suppression scale shows there to be a clear difference in the originally predicted direction of higher scores in the comorbid group B (See Table 15). A t test confirmed this difference to be statistically significant ($t(37)=2.139, p<.05$), with medium effect size of ($d=0.65)$.

Inspection of the group mean scores for the Cognitive Reappraisal subscale shows them to be very similar (See Table 15) and this similarity is confirmed by the non-significant result of a two-tailed t test ($t(37)=0.039, p>.05$). The effect size in this case was exceedingly small ($d=0.01$) and, although the inadequate statistical power ($P=.05$) does not allow a reliable assertion that there is no between-groups difference on this variable, the effect size indicates that further research is unlikely to reveal a substantial and clinically significant difference.
Figures 11 & 12: Boxplots for total scores on PANAS subscales – Lifetime groups.

Figures 13 & 14: Boxplots for total scores on ERQ subscales – Lifetime groups.
3.3.4.1 Analysis of the difference between the two subscale means on the ERQ.

To assess whether participants tended to score more highly on the Cognitive Reappraisal or Expressive Suppression scales, the individual’s mean score for the latter scale was subtracted from that for the former. The mean score was used because the participant’s total scores for each scale are not directly comparable as one is based on the total of 4 items and the other on the total of 6 items. Thus a negative score indicates higher scoring on Expressive Suppression whilst a positive score indicates higher scoring for Cognitive Reappraisal. It was predicted that the comorbid group B would show higher relative scoring for Expressive Suppression indicated by larger negative scores.

Previous research using factor analysis (Gross & John, 2003) has found that these two scales are independent, high scoring on one being no more or less likely to be accompanied by high scoring on the other. Indeed the correlation between scoring on the two scales was found to be exceptionally low ($r = -0.01$) in the undergraduate samples used for validation. As factor analysis of the measure’s structure is beyond the scope of this study and what the present data can justify, it is unclear whether an independence model of the two scales continues to be appropriate in this sample. Indeed whilst overall sample mean scores on the two scales were not significantly correlated ($r(39) = 0.309$, $p > 0.05$), the association was very close to significance. If the scales have retained relative independence (although this does not look likely) this may negate the value of attempting to make generalisable observations about relative scoring on the two scales. However, in the context of this exploratory analysis examination of relative scoring on the two subscales could provide useful information and therefore was pursued.
In previous research, group mean scores for Cognitive Reappraisal were larger than those for Expressive Suppression (by a margin of +0.96 for men and +1.25 for women) in a sample of undergraduates (Gross & John, 2003). However, in the current sample, the converse was true in 20 of 39 cases. Although only group means are available for the undergraduate sample, from which the level of individual variation is unclear, this may represent an important difference between a non-clinical sample and a sample of individuals with schizophrenia.

The ‘difference’ scores (Cognitive Reappraisal mean – Expressive Suppression mean) were compared for the lifetime criteria groups. The group mean ‘difference’ score was positive for the Comparison group B, indicating higher mean scoring on the Reappraisal scale, and negative for the Comorbid group B, indicating higher mean scoring on the Suppression scale (See Table 16). This difference between the groups is further illustrated in Figure 15 which shows a greater proportion of the Comorbid group scoring in the negative range.

Table 16: Descriptive statistics for ERQ mean score differences.

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Mean</th>
<th>SD</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comparison Group B</td>
<td>12</td>
<td>0.54</td>
<td>1.72</td>
<td>0.04</td>
</tr>
<tr>
<td>Comorbid Group B</td>
<td>27</td>
<td>-0.40</td>
<td>1.46</td>
<td>-0.50</td>
</tr>
</tbody>
</table>
Figure 15: Boxplot of ERQ subscale difference scores by lifetime substance misuse group.

A t test (2 tailed) demonstrated the difference between the groups mean 'difference' scores to be significant in the predicted direction, that the comorbid group B would show relatively higher scoring for Expressive Suppression, \(t(37)=1.753, p<.05\), with a medium effect size \((d=0.55)\). These analyses support the view that individuals who have a history of substance misuse make greater relative use of Expressive Suppression than Cognitive Reappraisal and may indicate a greater reliance on response-focused emotion regulation strategies of which expressive suppression is an exemplar.

However, it would be prudent to conduct further research into the factor structure of this measure in clinical populations as part of future research in this area. This would allow
more reliable conclusions to be drawn as to whether an independence model of the two scales continues to be appropriate in clinical groups.

3.3.5 Re-examination of CA-ERQ scores.

3.3.5.1 Internal-Functional scale.

Inspection of the descriptive statistics for the two groups on the Internal-Functional scale of the CA-ERQ reveals a clear difference in mean scores with the Comparison group B scoring more highly. Levene’s test ($F(38)=4.866, p<.05$) indicated significantly heterogeneous variances but all other conditions for the use of a t test were satisfied and therefore Welch’s t was used (2 tailed) and confirmed a the difference between the two group means on this scale to be statistically significant ($t(15.164)=2.200, p<.05$).

3.3.5.2 Internal-Dysfunctional scale.

There is a difference of approximately two points between both the group means and medians on this particular subscale in the direction of higher scoring by the Comparison group B (See Table 15), further illustrated by Figure 16 below. However, this difference did not attain statistical significance following analysis using a two-tailed t test ($t(38)=1.383, p>.05$). The statistical power of the analysis is insufficient to allow the reliable conclusion that there is not a genuine difference between the two groups and the effect size of the difference found is not inconsiderable at $d=0.446$. Expansion of the study to an achievable size of 60-70 participants per group would afford sufficient statistical power for a reliable conclusion to be drawn. Although not significant this
difference is of particular interest as it was originally hypothesised, on the basis of substance misuse being designated an internal-dysfunctional strategy, that the misuse group would score generally higher for internal and dysfunctional strategies. This has not been borne out by the data, the comparison group B’s mean score being the higher of the two.

3.3.5.3 External-Functional scale.

There was a small difference between the two group means, and a slightly larger median difference, with the comparison group B scoring more highly on this subscale. This difference, though, was not statistically significant (t(38)=1.032, p>.05). There was a small to medium effect size but the observed power was insufficient to allow a reliable rejection of the existence of a genuine difference on this variable. The study would require to be expanded to include approximately 100 individuals in each group to result in sufficient statistical power given this effect size.

3.3.5.4 External-Dysfunctional scale.

There were only slight differences in terms of measures of central tendency on this final subscale of the CA-ERQ in favour of slightly higher scoring by comparison group B. However, predictably this did not translate into a statistically significant difference between group means (t(38)=.632, p>.05). The effect size of the difference was small and the observed power very low indeed, meaning that the existence of such a difference again cannot be reliably dismissed on the basis of these analyses. The study, as currently
designed would have to include a somewhat prohibitive 400 participants in each group to afford sufficient statistical power.

As was the case for previous analyses based on 'current' substance misuse groups, the group means and medians are noticeably lower on this subscale than is the case for the other 5 item subscales (Internal-Functional and Internal-Dysfunctional) and even than the previous 4 item subscale, the External-Functional scale. Figure 16 illustrates that the two groups show a similar overall profile. Unfortunately, due to the lack of non-clinical adult comparison data for this measure it is not possible to assess whether a lower score for External-Dysfunctional strategies is also found in non-clinical groups. This seems not unlikely as the group of external-dysfunctional emotion regulation strategies are relatively high 'cost' in terms of the likely consequences and thus perhaps are used at a much lower frequency than other groups of emotion regulation strategies.

Figure 16: Line graph of group scale means divided by the number of scale items.
3.3.5.5 Implications of reanalyses for originally hypothesised differences in emotion regulation 'style' (Hypotheses 3 & 4).

Due to lower than anticipated effect sizes in several cases, there are restrictions on what can be reliably interpreted from these findings due to inadequate observed power. The comparison group B scored higher than the comorbid group B for all four subscales but the margin between the two groups on the Internal-Functional scale was the only one which demonstrated the anticipated medium effect size and reached statistical significance. For the other three subscales the presence of a genuine difference between the groups cannot be reliably rejected due to insufficient statistical power. It was somewhat surprising that the comparison group B also scored higher (although not significantly so) on the Internal-Dysfunctional scale, which would suggest that substance use is not necessarily accompanied by a generally greater relative use of this group of strategies as a means to regulate emotions.

It is particularly interesting that the two groups demonstrate remarkably similar ‘profiles’ across the subscales when group mean total scores are divided by the number of items contributing to the subscale. This indicates that, whilst there may be some differences in the degree to which particular groups of strategies are made use of, there is considerable degree to which the structure of emotion regulation is shared between these two groups. Indeed, the main feature of this profile for both groups is consistency across three of the four subscales accompanied by a dip in scoring on the External-Dysfunctional subscale.
On the basis of these analyses, there is no support for the contention that the comorbid group will be typified by higher scoring for either internal or dysfunctional strategies when compared to the comparison group. Nor is there any indication of increased scoring in these areas relative to other domains amongst this group.

3.3.6 Analyses of gender as a covariate in 'lifetime' substance-use groups.

Given that the difference in the gender composition of the two groups following changes in the grouping criteria, whilst not reaching was approaching statistical significance, it was considered prudent to conduct some brief analyses treating gender as a covariate. ANCOVAs were conducted with gender entered as a covariate in order to gain an estimate of 'lifetime' group means on the emotion and emotion regulation variables had both groups been equal in terms of gender composition. The aim of these analyses was to provide an indication of whether the significant differences identified above were genuine and not just an artefact of gender differences and also, whether any other genuine differences were likely to be being obscured by the differences in gender composition of the two groups. The outcomes of these analyses are summarised in Appendix P and the most relevant findings shall be briefly outlined here. The conclusions that can be reliably drawn on the basis of this additional analysis are, unfortunately, somewhat limited due to inadequate statistical power in the majority of cases.
Differences between group mean scores on the Positive Affect scale of the PANAS, which had been found to differ significantly in previous analyses, as estimated when gender is treated as a covariate were very close to significance \((p=.07)\) with a medium effect size when statistical power was approximately half that which is optimal \((P=.442)\). This is a fairly strong indicator that, given sufficient power, this effect would be demonstrated to be genuine and not an artefact of gender differences between the groups, although further research where groups were matched for gender and of sufficient size to provide adequate power would be needed to assess this more reliably.

The estimated group means for the Expressive Suppression scale of the ERQ when gender is treated as a covariate, which had also been found to differ significantly in the previous analyses based on ‘lifetime’ groups, were also very close to being significantly different \((p=.089)\), whilst the power of the analyses was rather low \((P=.415)\). This provides a similar indication of a likely genuine group difference which requires to be reassessed in larger samples matched for gender composition of the substance misuse and comparison groups.

Additionally, the estimated group means for scoring on the Internal-Functional scale of the CA-ERQ were found to remain significantly different, as had been the case in the earlier analyses, when gender was treated as a covariate \((F(1,37=4.133, p<.05)\). This finding in particular confirms a difference between the two groups in terms of the use of emotion regulation strategies, as measured by the CA-ERQ, which is not an artefact of gender differences in the composition of the two groups.
These analyses did not provide any indications of further significant between-group differences when age was treated as a covariate, although due to the generally low observed power resulting from smaller than expected effect sizes and uneven groups, it is not possible to reliably reject the possibility of further significant differences. The indications are that all three between group differences found to be significant when the influence of gender was not accounted for, are likely to be genuine differences and not artefacts of the gender composition of the groups. However, with respect to the Positive Affect and Expressive Suppression scores this requires to be re-examined under conditions which afford greater statistical power.
3.3.7 Summary of findings from re-analyses using lifetime grouping criteria.

The rationale for changing the grouping criteria was that the original criteria may have reduced the efficiency of the research design by grouping people who differed in important ways relevant to the research hypotheses together within the initial comparison group. Changing the criteria was an attempt to examine whether this within-group variation had obscured between group differences. When the grouping criteria were changed to lifetime criteria for misuse, based on self and staff reports, despite the detrimental effect of uneven groups on statistical power, significant between-group differences were found for scoring on measures of Positive Affect, Expressive Suppression and the use of Internal-Functional emotion regulation strategies. The comorbid ‘lifetime’ group scored significantly lower for Positive Affect, higher for the use of Expressive Suppression and lower for the group of emotion regulation strategies classified as Internal-Functional.

The reclassified ‘lifetime’ groups were close to being significantly different in terms of gender composition and therefore it was important to briefly examine gender as a covariate to gain an estimate of group mean scores on the various measures had both groups been equal for gender composition. This helps to address queries as to whether any apparent group differences, which the research sought to identify in terms of differences in substance misuse, were more likely to be the outcome of group differences in gender composition. The low statistical power of these analyses is somewhat of a barrier to reliable interpretation of the results. However, with respect to the three significant group differences identified using t tests, one remained significant (Internal-
Functional scores) and two were very close to statistical significance (Positive Affect and Expressive Suppression) where the observed power was low, providing a reasonable indication that given sufficient power, the difference would attain significance even with the influence of gender statistically extracted. Nonetheless, replication of these findings under more statistically rigorous conditions and with gender composition matched comparison groups is advisable.
4. DISCUSSION.

4.1 Summary of findings.

The following discussion focuses on the results of the reanalysis, where individuals were reallocated to two groups on the basis of lifetime substance misuse status; those where there was no indication of substance misuse at any time in their life (comparison group), and those for whom there was evidence of either current or past misuse (comorbid group) (Results section p.106-126). Unless specified otherwise, results discussed shall are for these analyses.

4.1.1. Lower positive affect scores in the comorbid group.

Between-groups analyses identified significantly lower scoring by the comorbid group for positive affect on the PANAS. This finding has relevance to hypothesis 1, which predicted that individuals in the comorbid group would score lower on a measure of positive emotion and higher on a measure of negative emotion than the comparison group. It would be prudent to replicate this finding in another sample, with groups matched for gender and age composition.

The between-group difference in positive affect was very close to significance when the effect of gender was statistically partialled out using analysis of covariance. Given that the power of this analysis was relatively low, it is likely, given sufficient statistical power, the between-group difference on this variable would be confirmed to be genuine
and not just an artefact of group differences in gender composition. However, further research is essential to confirm this.

Lower positive affect in the comorbidity group is in keeping with research literature, which has consistently found that one of the key self-reported reasons for using psychoactive substances in comorbid groups is to enhance positive affect (Spencer et al., 2002; Dixon et al., 1991). This ‘enhancement’ rationale may indicate that comorbid groups experience a deficit in positive affect, relative to patients without comorbid substance misuse, and the findings of this study are consistent with such an interpretation. A possible alternative interpretation is, that the comorbid group could have a higher subjective threshold for optimal levels of positive affect. These possibilities would be both interesting and challenging to disentangle in future studies.

With respect to negative affect these analyses are not sufficiently statistically powerful to draw reliable conclusions as to the presence, or absence, of a genuine difference between the groups. The small effect size requires substantially more participants or a significantly more powerful design for reliable conclusions to be made. The failure to confirm a significant difference between the groups in terms of negative affect is inconsistent with earlier findings by Blanchard et al. (1999), although this may be due to differences in the two research designs. Blanchard et al. (1999) found a significant association between trait negative affectivity, associated with a greater propensity to experience negative emotion, and the severity of substance misuse within a comorbid
group. This was based on a within group analysis, rather than a between-groups design as was used in this study.

The predicted between-group differences, with respect to positive and negative affect, were mainly based on the key propositions of Blanchard et al.'s (2000) proposed integrative model of substance misuse in schizophrenia, which suggests that a more severe level of aversive emotional experience ought to characterise this group. The findings of the present study provide partial support for this contention, in terms of lower levels of positive emotions experienced, although this requires future confirmation. Conclusions as to whether there is a genuine difference between comorbid and non-comorbid groups in negative affect cannot be reliably made on the basis of this data.

Current levels of positive and negative emotion in the comorbid group may have been influenced by the effects and consequences of substance misuse and may, therefore, not be particularly informative about initial motivations for use. The small, non-significant group difference in current negative affect measured by the PANAS may have been reduced as a result of substance misuse alleviating some degree of dysphoria in the comorbid group. Equally, it is possible that substance misuse has over time led to a reduction of positive affect in the comorbid group. A lower level of positive affect, in a group where recourse is being made to a function of substance misuse being to enhance positive emotion, may indicate that substance use is not proving effective in meeting this hypothesised need, undermining the relevance of emotion regulation to this behaviour. However, the impact of substance use on emotion may be relatively discreet and short-
term, whilst respondents were asked to indicate their general level of emotion on the PANAS. A mismatch in the relevant time frames could have reduced the clarity of the relationships between measured emotion-related variables and the use of substances.

These observations draw attention to a general limitation of this study design, the cross-sectional nature of the study restricts interpretation of the direction of relationships between variables. It is not possible, within this approach, to establish whether the two groups differed in terms of positive or negative affect prior to the commencement of substance misuse nor to assess how these variables may have changed over time. Longitudinal research using prospective designs will be essential to elucidate such factors.
4.1.2. Higher scoring on a measure of the emotion regulation strategy expressive suppression (ERQ) in the comorbid group.

The comorbid group scored significantly higher than the comparison group on a measure of the emotion regulation strategy expressive suppression, although again replication is advised to ensure the validity of this finding. This is especially important given that when the influence of group gender differences was adjusted for, the difference was narrowly non-significant, although statistical power was well below that desired. Given the sub-optimal power, it remains likely that there is a genuine between-group difference on this particular variable which is independent of the influence of gender, but this requires verification. Hypothesis 2, which predicted such a difference in the use of expressive suppression, was developed on the basis of Gross’ model of emotion regulation strategies which distinguishes antecedent and response-focused strategies (Gross, 1998). Substance use is classified within this model together with expressive suppression as response-focused, being an emotion regulation strategy used to modify physiological, cognitive or emotional aspects of the individual’s experience of an emotion once generated. The issue considered by this study was whether substance misuse constitutes one manifestation of an emotion regulation profile dominated by response-focused strategies. Several theorists have proposed that individuals develop a stable style of emotion regulation (e.g. Thompson, 1994) and it may be possible to characterise such a style in terms of the model of emotion regulation, such as that developed by Gross (1998).
The finding of a between-group difference on a measure of expressive suppression supports one of the key propositions of this study and indeed of Blanchard et al.'s (2000) model; that the two groups differ significantly in terms of their use of emotion regulation processes. It is also consistent with the proposition that the comorbid group may rely to a greater extent on the generally less effective group of response-focused strategies (Gross & Levenson, 1997). This is further supported by the finding that when mean-difference scores are calculated for the two types of strategy measured by the ERQ, there is a significant difference between the two groups consistent with the comorbid group having a higher mean score for the response-focused strategy and the no misuse group having a higher mean score for the antecedent-focused strategy.

Response-focused strategies are generally less effective at achieving meaningful change in an individual's subjective experience of an emotion, because the emotion in question has already been generated (Gross & Levenson, 1997). A caveat to this is that expressive suppression has been associated with reduced subjective experience of positive emotions (Gross & Levenson, 1997). Based on this, it is a reasonable assumption that individuals who rely to a greater degree on expressive suppression and other response-focused strategies, such as substance use (following Gross' 1998 model) will be less effective at regulating their emotion experience. The finding of the present study—that a group who by definition make greater use of one strategy classified as response-focused (substance use) also score more highly on another (expressive suppression)—is consistent with the view that individuals develop a relatively stable 'style' of emotion regulation (Thompson,
and that in the case of substance misusers, this style may be more heavily weighted towards response-focused strategies.

However, the evolution of emotion regulation models, such as that developed by Gross (1998) and used as a framework in this study, is at a relatively early stage and the available models are perhaps somewhat simplistic relative to the complexity of the phenomena they seek to explain. Specifically, it is unclear whether it is appropriate to consider substance use/misuse as a consistently response-focused emotion regulation strategy. The reality seems likely to be considerably more complex. In certain scenarios, substance use may be more in keeping with an antecedent-focused approach to emotion regulation, particularly if it is being used to elicit positive emotional experiences. A combination of antecedent and response-focused functions may well be relevant to specific instances of substance use. Further investigations and theoretical development are required, to develop understanding of how to characterise substance use within models of emotion regulation.

The two main findings discussed thus far, of lower positive affect and higher expressive suppression in the comorbid substance misuse group, may be inter-related. Research has found that suppressing expression of positive emotions tends to reduce subjective experience of these emotions, which has not been found to be generally the case with negative emotions (Gross & Levenson, 1997). Item 4 of the ERQ specifically enquires about suppressing expression of positive emotions and, indeed, the comorbid group mean for this item was significantly higher than the comparison group mean (see Appendix O),
indicating a tendency which could contribute to significantly lower experience of positive emotions. If lowered positive emotion then motivates substance misuse as a means to enhance these experiences, this may be one route via which a particular pattern of emotion regulation could influence substance misuse.
4.1.3. Lower scores for the use of Internal-Functional emotion regulation strategies by the comorbid group on the CA-ERQ.

This study identified a significant between-group difference for scoring on the Internal-Functional scale of the CA-ERQ, with the comorbid group scoring lower on average. The group of emotion regulation strategies designated as Internal-Functional are, as the name would suggest, generally advantageous strategies that draw on resources internal to the individual, which are, therefore, consistently available for the regulation of emotions. The comorbid group are, therefore, likely to be at a relative disadvantage to achieve effective emotion regulation as a result of this difference. Indeed, of the three significant between-group differences identified in this study, this particular difference is the only one to remain robustly significant, even when the effects of group differences in gender composition are adjusted for, which reinforces the validity of this finding.

Whilst this study did not identify any significant differences between the two 'lifetime' criteria groups on the other three subscales of the CA-ERQ, there were also interesting features of these results. On the Internal-Dysfunctional scale, which pertains most directly to the differences predicted between the groups in hypotheses 3 and 4, the effect size was small-medium, but in the opposite direction to predictions made at the outset, the mean score for the comparison group being higher on this scale. The statistical power being low, it is not possible to reach a reliable conclusion on this between-group difference. The results were similar for the External-Functional scale which, although not reaching significance, is more in keeping with the originally hypothesised differences. However, on the External-Dysfunctional scale, the comparison group mean
score was once again higher—although the effect size of this last difference was very small. In summary, across all four subscales the comparison group scored higher, on average, than the comorbid group, the difference only reaching a reliable level of significance in the case of the Internal-Functional scale. However, it is interesting nonetheless, that the comparison group seem to make generally greater use of all emotion regulation strategies measured relative to the comorbid group, regardless of the nature of resources used and the functionality of these strategies as defined by the CA-ERQ. Further evaluation of group differences on this measure, in a larger sample with even-sized groups, should allow sufficient statistical power to establish reliably whether the comorbid group manifest a generally lower level of strategy use.

With respect to the predictions made by Hypotheses 3 and 4—that the comorbid group would score relatively higher on scales that had an Internal and a Dysfunctional component—this has not been borne out by the data gathered in this study. Indeed, although not significantly so in three scales, the comparison group scored higher on all four scales. Nor was there an indication, within the profile of the comorbid group across the four scales, that within this group scoring was relatively higher on scales with an Internal or Dysfunctional component. Indeed, it was particularly interesting to chart the ‘profiles’ for both groups across the four subscales and note the high level of similarity between the two, with relative consistency of scoring across three scales and a clear dip for both groups in scoring on the External-Dysfunctional scale. It would be particularly valuable, through further research and validation of this measure with both clinical and
non-clinical adult groups, to establish whether this profile is normative or specifically characteristic of groups with schizophrenia.
4.2. Implications of findings for an emotion regulation model of comorbidity.

The aim of this study was to begin to examine and compare emotion experience and regulation in a group of individuals with schizophrenia and comorbid substance misuse with a comparison group of individuals with schizophrenia and no comorbid substance misuse, in order to assess whether differences in these variables could account for a degree of the excess vulnerability to substance misuse associated with schizophrenia. Essentially, Blanchard et al.’s (2000) model involves three key propositions that the present study sought to examine:

1. Individuals with comorbid schizophrenia and substance misuse experience higher negative affect and lower positive affect than those with schizophrenia only.
2. Individuals with comorbid schizophrenia and substance misuse are less effective at regulating their emotions than individuals with schizophrenia only.
3. These features confer a significant degree of vulnerability to substance misuse.

Overall, this study provides some qualified support for the model. It has identified significant differences between groups of individuals with schizophrenia who have never misused substances (comparison group) and those who currently do so, or have done so in the past, (comorbid group) in terms of both the nature of their emotional experience and their use of specific emotion regulation strategies. These findings are consistent with two of the central propositions of Blanchard et al.’s (2000) model. The comorbid group’s mean scores for emotion experience indicated a less desirable ratio of positive and negative emotions—positive being significantly lower. Their mean scores for emotion regulation measures indicated both a greater use of a strategy that has been found to be
less effective for modifying emotion experience, expressive suppression, and lower scores for the use of the ‘desirable’ Internal-Functional group of emotion regulation strategies.

In terms of the third proposition, that differences in emotion and its regulation constitute a significant vulnerability to substance misuse, there is some indirect support for this from the finding that these features differentiate those with current or past comorbid substance misuse, from those with no history of comorbid substance misuse. Blanchard et al.’s (2000) previous research had not made such comparisons, having examined within-group associations between traits related to emotional experience and the severity of substance misuse. However, there are restrictions on what can be concluded, in terms of whether these differences represent preceding vulnerabilities to substance misuse due to the cross-sectional nature of this study.

Although the main findings of this study are consistent with the key propositions of Blanchard et al.’s (2000) model, they are not exclusively so and alternative interpretations are possible. There is a need to replicate these between-group differences in a large study, where groups are matched for both age and gender, in order to ensure their validity. Therefore, a cautious approach is necessary when considering the implications of these findings.
4.3 Further observations.

The findings of this study suggest that, on some emotion and emotion regulation variables, individuals with histories of comorbid substance misuse, in the absence of current misuse, are more similar to current misusers than to individuals with no such history. This is consistent with enduring individual differences in emotion regulation style that are stable over time (Thompson, 1994). Another implication of the fact that past misusers seemed to obscure between group differences when originally classified in the comparison group, is that it is less likely that the group differences identified are direct effects of misuse, although it remains possible that these differences may be associated with long-term consequences of substance misuse. They may instead represent enduring vulnerabilities which contribute to the development and maintenance of substance misuse, and perhaps also to the development of psychosis, in keeping with an emotion regulation approach to understanding comorbidity, although this remains to be reliably established.

The results of the present study indicate that changes in substance misuse status are not necessarily accompanied by changes in an individual’s emotion regulation style. When current and past misusers were compared directly, during an evaluation of the grouping criteria, they did not differ significantly on any of the measured variables and when grouped together they differed significantly from the comparison group on three variables. The fact that the past group still differed from the non-users on these variables, and yet were not currently misusing, does not negate an emotion regulatory function for
substance use. Indeed, although substance use change may be accompanied by changes in emotion regulation that have not been tapped by the present research, it seems likely that other factors contribute to the process of changing patterns of substance use.

Use of psychoactive substances and substance selection by individuals with schizophrenia, for emotion regulation or indeed other purposes, is influenced by beliefs and expectancies regarding likely outcomes (Mueser et al., 1995), which form part of a decisional-balance with respect to the perceived costs and benefits of substance use. Experiences of substance misuse and its impact, may lead to changes in expectancies and, over time, there may be changes in the relative importance of different substance-related outcomes and awareness of substance-related costs. For example, an individual who previously valued the short-term effects of a substance on their emotional experience may gradually link adverse effects on various areas of functioning—such as occupational functioning, or the level of psychotic symptoms experienced—to patterns of substance use. The relative priority attributed to these outcomes will influence the individual’s ongoing behaviour. Prohibitive costs may come to be associated with substance misuse, as the result of negative and possibly traumatic experiences, which may then contribute to behaviour change. These routes to change do not require that an individual’s emotion regulation skill or style has changed—just that there is a change in the relative balance of costs and benefits relating to use. However, this does not negate the possibility that substance use may have served an emotion regulation function for the individual.
Previous research findings offer some support for this understanding of how change in substance misuse may come about in a dual diagnosis sample. Blume and Schmaling (1997) found that scores on a measure of readiness to change, in a sample of dually diagnosed individuals (psychiatric inpatients with Axis I disorders and comorbid substance misuse), were associated with reports of increased negative substance-related sequelae, particularly physical sequelae. The same researchers also identified a significant association between substance-related fears for the future and changes in substance use (Blume & Schmaling, 1998). Additionally, Blume and Marlatt (2000) reported an association between important substance-related losses and readiness to change for substance use in this group. Whether the individual attributed losses to substance use, was of greater importance than either the importance, or frequency, of the relevant losses, indicating that changes to substance-specific expectancies were most relevant to intentions to change. These analyses place changing substance use in dual diagnosis samples in the context of Prochaska and DiClemente’s (1982) transtheoretical stages of change model, which essentially proposes that the disadvantages of a behaviour must be perceived to exceed its benefits before change can be sustained. Blume and Marlatt’s (2000) findings particularly emphasise that the individual needs to link adverse consequences experienced to substance misuse, for changes in that specific behaviour to become likely.

In addition to an individual’s beliefs and expectations regarding the effects of psychoactive substances, the beliefs an individual holds about emotions, and their ability to influence and modulate their emotional experiences, are of great relevance to
understanding substance use as a means to modulate emotions. Beliefs about the meaning of emotions, their amenability to influence and the individual’s, and significant other’s, abilities to modulate them, are shaped by the developmental processes which mould emotion regulation systems (Calkins, 1994). These beliefs make an important ongoing contribution to the emotion regulation system.

Several participants in this study made spontaneous comments whilst completing the measures, which are very illuminating in terms of their emotion-regulation beliefs. One individual commented that he considered no emotions to be positive in nature when completing the ERQ, which asks respondents to specifically consider their responses to negative and positive emotions on different items (participant id 33). This view indicates that participant 33 experiences all emotions, of whatever valence, as undesirable in some way. This individual and another participant (id 24) both expressed the view, when attempting to complete the ERQ, that it was not possible to influence emotional experiences in any way. Participant 24 found the possibilities suggested by the items in the ERQ to be totally novel and confusing, to the extent that he declined to complete the measure. Other participants made comments to the effect that they were unfamiliar with the strategies in the cognitive reappraisal items of the ERQ and questioned whether it is possible to alter emotional experiences in these ways. Although anecdotal, in summary, several participants indicated that the concept of modulating and influencing emotional experiences was unfamiliar to them. The implication of this is that such individuals do not experience themselves as effective in modulating their own emotional experiences
and are likely to be less able to do so in ways which minimise adverse effects on their functioning.
### 4.4 Critique of the study design

One of the key limitations of this study arises from the nature of the cross-sectional design, as previously indicated, which means it is not possible to reliably establish the direction of any causal relationships between variables. For example, it is not possible to establish whether between-group differences in emotion regulation represent a vulnerability to the development of substance misuse in the comorbid group, or whether these differences are due to the effects of substance use.

Within the scope of the present research project, a longitudinal design was not achievable. However, given the early stage of research in this area, the use of cross-sectional designs to begin to identify group differences is nonetheless valuable. Moreover, in the longer term, it would be beneficial for this research program to develop to include prospective research, following a developmental psychopathology model wherein vulnerability factors, such as emotion regulation strategies, can be assessed and their association with the development of later difficulties followed-up over time. This would allow consideration of whether emotional dysfunction and dysregulation confer vulnerability to the development of schizophrenia and substance misuse, and the pathways involved.

The lifetime prevalence rate for substance misuse in this sample—70 per cent—was somewhat higher than is generally reported in the literature—usually reported rates are between 40 and 60 per cent (Kavanagh et al., 2004). One possible explanation for this
difference is that rates are often found to be elevated in high intensity services (Kavanagh et al., 2004) and a substantial proportion of participants in this study were clients of an assertive outreach team for people with complex needs, which could be defined as a high intensity service.

Other potential sources of bias may have contributed to a higher lifetime prevalence rate in this particular sample. There may have been an element of researcher bias in the recruitment process. The clinical staff, who identified potential participants, reported to the researcher any clinical indications or knowledge of substance misuse in each case. It is possible that the researcher was more persistent in her efforts to contact individuals with known substance misuse histories, due to the particular relevance of such individuals to the study. Similarly, clinical staff may have demonstrated a degree of bias in their selection of potential participants, believing certain individuals to be more relevant to the study. Finally, there may have been a bias in the responses of participants to the invitation to take part in the study. Individuals with a current, or past, substance use disorder, may have perceived the study to be more relevant to them and therefore may have been more likely to agree to participate.

Whilst it is possible that this research study sampled a population with a particularly high rate of comorbidity, this is unlikely, given the breadth of prevalence studies that have been conducted, although the current study did make use of a measure designed to be a more sensitive screening tool for substance misuse in psychosis, which research has found can often be under-recognised (Trembath, 2005). Alternatively, it may be that the
thresholds for misuse on the DrugCheck measure were too sensitive and may have led to some false positives in this sample. The DrugCheck was the best screening option available and has been specifically tailored to detect substance misuse in psychosis, with good validation data available for three Australian clinical samples (Trembath, 2005). However, it would be preferable to validate the scale with a UK sample and adjust the cut-off scores used accordingly, as there may be important differences from the Australian samples. One positive indication of the high level of comorbidity detected using the DrugCheck, is that this is not consistent with the view that participants would fail to disclose problematic patterns of substance use on a self-report measure, supporting the validity of this methodology.

The ‘current’ comorbid group were classified on the basis of scoring above the established threshold on either the Problem List measure or the AUDIT, both of which are part of the DrugCheck. There were five cases where the individual did not score above the threshold on both measures. In all cases the individuals scored above the AUDIT threshold for alcohol misuse, but below the Problem List threshold. On the basis that the AUDIT score was based on a more objective measure of level and pattern of alcohol intake and the experience of common alcohol-related problems, and depends less on an individual’s judgement of whether any problems experienced are associated with substances than the Problem List, it was decided to classify these individuals on the basis of their AUDIT score alone. Also, there is a broader range of empirical research supporting the use of the AUDIT in these groups (Dawe et al., 2000). It would have been
preferable to have established clearer criteria for dealing with such inconsistencies in advance.

After conducting analyses based on between-group comparisons for current substance use status, the decision was taken that it would be valuable to reclassify individuals in terms of lifetime substance misuse status. It was acknowledged that the basis on which individuals were reclassified was less stringent than the assessment of current substance use. Participants did not complete the DrugCheck in full for past use, but were asked to describe their heaviest period of substance use in the past in terms of type, quantity and frequency. In most cases the quantities and frequencies involved for the eight individuals who were reclassified, for example daily inhalation of solvents or daily consumption of half a bottle of whisky, were clearly indicative of misuse, given the thresholds set for current use on the DrugCheck. Staff reports were also used to guide these judgements, and in one case (id 8), where there was past diagnosis of substance abuse which was not disclosed by the participant, the individual was allocated to the lifetime misuse group on the basis of staff report. These methods of classification may not be optimal, but it was considered sufficiently worthwhile to proceed with the post-hoc analyses. In future, a more rigorous assessment of past misuse would be preferable.

An opportunity sample was used in this study, whereas deliberate sampling would be preferable, incorporating the DrugCheck as a screening measure into the sampling process. It was fortunate that the original ‘current’ groups were even in size, although once the groups were reorganised in terms of ‘lifetime’ substance use status, this was no
longer the case. This reorganisation adversely impacted on statistical power. Nonetheless, this study did represent exhaustive sampling of individuals in three local services which cover the majority of individuals in the local area with diagnoses of schizophrenia and are involved with secondary NHS services, with the caveat of clinical staff deeming it appropriate to invite the individuals to participate and individuals being contactable. The participation rate was fairly typical at 31.01% and although it was not possible to recruit the number of participants desired, based on prospective power analyses, to do so would have required considerably greater resources than were available, in terms of accessing participants beyond the local area.

In light of the significant group differences in age and near significant differences in gender composition, which have made interpretation of some of the findings in this study more difficult, future research would benefit by matching groups for composition on these variables. It would also be desirable to examine these variables in other comparison or control groups—which was beyond the scope of the present study. A primary substance misuse group where there is no comorbid SEMI would be particularly useful, although it is likely to prove difficult to recruit such a group which does not also have significant psychiatric comorbidity. Involvement of a non-clinical adult comparison group would further help to partial out the differences on these emotion and emotion regulation variables that relate to psychosis, to substance misuse and specifically to comorbidity of the two. For example, inclusion of these groups would help to establish whether the profile shown by both groups in this study, across the four CA-ERQ scales, is unique to these groups or normative.
The need to have clinical staff select appropriate individuals to participate in the study from their caseloads leads to both benefits and the potential for bias. This was necessary due to the management of case information in the services involved. The benefits include that individuals are not unnecessarily burdened where there are good contra-indications to inviting them to participate in the study. However, clinical staff may tend to select individuals on the basis of who they predict will take part and to be conservative in some cases and so the sample may be shaped more by staff views on appropriate participants, than the explicit inclusion and exclusion criteria. This can be ameliorated by providing clear guidance regarding inclusion and exclusion criteria, which was done in this case.

It is worth considering the relative merits of the method by which emotion experience and emotion regulation have been assessed in this study. The use of self-report measures relies greatly on the awareness and reliability of the individual concerned. However, many emotion regulation processes are likely to be relatively non-conscious, or at least not executed with full awareness of the emotion regulation implications. Therefore, to rely on self-report could reduce the accuracy of assessment—a concern which applies to any sample and not specifically to individuals diagnosed with schizophrenia. However, although there may be difficulties inherent in using the few self-report measures available, there is currently a lack of alternative methods.

Research into the development of emotion regulation in young children has tended to use behavioural observation of individuals in emotion-eliciting situations as a methodology,
borrowing heavily from the paradigms developed in attachment research (e.g. Thompson, 1994). However, this relies greatly on examining the behavioural correlates of emotion and would require considerable revisions to the situations used to make it developmentally appropriate for adults. Also, an extraordinary level of knowledge of typical behaviour, when emotions are unregulated or regulated in different ways, is required to make reliable inferences about emotion regulation on this basis (Gross, 1999).

Self-report measures of emotion and emotion regulation do seem to offer one of the most appropriate and realistic methodologies for exploring individual and group differences in these constructs with adults. However, what measures of emotion regulation are available have only very recently been developed and no research was identified which reports on or has validated their use with groups of individuals with psychiatric diagnoses or with individuals with substance misuse, which has restricted their reliability and interpretation in this study. Nonetheless, the measures (ERQ and CA-ERQ) were made use of in the context of their being the best and most rigorous options available. It will be important for further research into emotion regulation that measures such as the second part of the CA-ERQ, which was not used in this study in order to avoid the burden on respondents becoming excessive, which assesses the strategies used to regulate specific emotions, are developed further and that relevant emotion regulation measures are validated for use with a wider range of populations. Perhaps another useful way to maximise the quality of information being gathered could be to use measures of emotion regulation within a time or experience sampling methodology which ought to enhance the ecological validity of measures.
In this study five participants completed the measures as structured interviews with the assistance of the researcher, as this was their preference. The possibility for participants responses to be influenced by pressures for socially desirable responding are greater in this format, than when an individual completes a questionnaire measure independently. This may have reduced the validity of this subset of the data. Although it is preferable to use a consistent response format across all participants, it is also important to be flexible, particularly with this population, and make the research materials as accessible to participants as possible. This was the approach taken in the present study.

As outlined previously, Gross (1999) raises a key difficulty in the field of emotion regulation theory and research; that in order to understand how emotions are regulated, what the likely outcomes are, when this will occur and how it can be recognised, it is necessary to have a ‘formidable’ level of understanding of emotion in it’s unregulated form, which in many cases is not available. Gross (1999) also raises the question of whether it is possible to understand these as separable processes, the generation and regulation of emotion being so interwoven that it is unlikely to be possible to separately establish a full understanding of unregulated emotion to inform understanding of emotion regulation. It seems that it does not make sense to separate the two processes, although as a theoretical heuristic it may be helpful, but that understanding of both aspects must develop in an integrated way.
4.5 Areas for further research.

Initially the key findings from this research project require replication, particularly the differences in Positive Affect (PANAS) and Expressive Suppression (ERQ) in order to ensure their independence from gender effects. Beyond this, there remains a great deal about emotion regulation in schizophrenia and comorbid groups that is unknown. The pace of investigations will be tempered by the need to develop and validate measures of emotion regulation with these groups and, indeed, to develop alternative paradigms. Much also remains to be investigated in terms of emotion regulation in the general population. This would provide a broader context for findings in relation to schizophrenia and comorbid substance misuse and, moreover examination of these variables in primary substance misuse groups, as outlined in the previous section, is also necessary.

If it is more reliably established in the future, that particular emotion regulation difficulties characterise comorbid groups, either in terms of severity or type, a key question that then needs to be addressed is whether emotion regulation difficulties precede the onset of substance misuse, schizophrenia or both, and can be understood as conferring significant vulnerability to one or both conditions. Research using a prospective model will be needed to examine these questions. Longitudinal research will also facilitate teasing apart the influence of emotion regulation on substance use and of substance use on emotions and their regulation.
Researchers may also begin to address the relationships between emotion regulation variables, substance misuse and schizophrenia by looking in a more sophisticated way at differences between individuals with schizophrenia who have never misused substances, those who have done so in the past and those who are currently doing so to try and elucidate the crucial vulnerabilities and processes of change involved. The present study, although interpretations are limited as a result of sample sizes, provided some indications that there may be important differences, at least in terms of emotion experience, between individuals who have misused substances in the past and those who currently do so. Future research which is specifically designed to compare these three groups, current, past and never, would allow for more powerful comparisons. Additionally, and perhaps more powerfully, the relationships of schizophrenia, substance misuse and comorbidity factors to emotion regulation would be further elucidated by studying both non-clinical adult groups and primary substance misuse groups, as identified in the previous section.

It is unclear whether it is appropriate to continue to treat individuals with diagnoses of schizophrenia and comorbid substance misuse as a cohesive group. Doing so could obscure important differences in the variables of interest. Dual diagnosis groups may subsume distinct subgroups characterised by different pathways and vulnerabilities. For particular subgroups the aetiology of comorbidity may be more consistent with secondary substance misuse, such as in the supersensitivity model, whilst for others a common underlying vulnerability to the two conditions may be a more accurate characterisation. Blanchard et al.’s (2000) proposed model goes some way to bridging the gap between secondary and common-factor models, incorporating elements of both. Refining research
designs to take account of the potential for different subgroup aetiologies, within dual diagnosis groups, may reveal that models have varied degrees of relevance to specific subgroups and emotion regulation approaches may be particularly relevant for a certain subgroup of individuals.

Pathways to emotional dysfunction in this group may also differ in character from those associated with the development of psychosis without comorbid substance misuse (Birchwood, 2003), which merits future investigation. It will be particularly interesting to consider the outcomes of any such research in relation to previously published findings that indicate that, in relation to social functioning, dually diagnosed individuals tend to have demonstrated better premorbid adjustment than individuals who develop schizophrenia in the absence of substance misuse (Graham et al., 2004). Given the important role that emotional functioning and emotion regulation are considered to have in social functioning, it will be important to consider how these findings fit in with knowledge of emotional functioning in comorbidity as this develops.

In terms of specifically developing the design of the present research, it will be valuable to attempt to replicate these findings with sufficiently large and evenly sized groups, matched for age and gender, that will afford sufficient statistical power to validate the key findings of the present study and reach firm conclusions on other group differences—in particular in terms of negative affect. With respect to the CA-ERQ, it will also be important to validate this measure for use with these populations. Similarly for the ERQ and the DrugCheck with appropriate United Kingdom samples in order to strengthen the
validity of these measures, and the DrugCheck’s use as a means to classify individuals as meeting criteria for comorbid substance misuse in future research.

Of particular interest, following this investigation and the comments made by some participants, is the development of research which would take a side-step from the focus of this study and look at the beliefs of individuals with schizophrenia and specifically those with comorbid substance misuse regarding emotions and emotion regulation. Such beliefs will have an important role in shaping attempts to regulate emotions and indeed offer a route to improving our understanding of internal models of emotion and emotion regulation in these groups. A qualitative study, perhaps using grounded theory methodology, could provide valuable insights to contribute to the development of models of emotion regulation in these groups at this early stage in the research program.
4.6 Clinical implications of findings.

Although it is difficult to reliably extract broader clinical implications on the basis of this study alone, as the research program develops it is anticipated that the findings will have significant implications for clinical work with individuals with comorbid SEMI and substance misuse. Whilst it is certainly true that enhancing emotion regulation skills is embedded in many therapeutic approaches to psychological problems, this is generally fairly implicit and is perhaps given less emphasis in psychological work with psychosis than is the case with other clinical groups. Although, the implications of the present research for clinical work remain to be clarified through further validation, they include the importance of being mindful of, and seeking to identify, any possible emotion regulatory functions of substance misuse when working with a client with comorbid difficulties. Identifying beliefs about emotions and their regulation, working to enhance self-regulation skills and looking for links with the role of emotional functioning in the course of their psychosis, will also be important. If difficulties in emotion regulation do come to be understood as a common factor which confers vulnerability to developing both schizophrenia and substance misuse in some individuals, a therapeutic approach which places emotional functioning and the enhancement of self-regulation centrally should have a positive impact on both aspects of dual diagnoses.

The findings of this study indicate that a particular area of importance, for therapeutic work, may be a relative deficit in Positive Affect and also a tendency to suppress the expression of positive emotions. Based on this, it would be particularly important to
work clinically with individuals to broaden and strengthen their repertoire of skills for the regulation and appropriate expression of positive emotions. It is perhaps the case that the tendency is to focus clinically on the regulation of, and coping with, negative emotions to the detriment of potentially very valuable work addressing deficits in positive emotions. Whilst the two are no doubt inter-related in complex ways, it may become increasingly apparent, as this research program develops, that specific positive affect work is extremely important in this particular group. Challenging unhelpful beliefs that people may hold about emotions generally, such as that they are ‘uncontrollable’, and working with individuals to develop an inquisitive attitude towards their emotional responses, as meaningful and useful signals, would be part of such work. Another clinical implication from this study would be the importance of working with comorbid groups in particular to strengthen their repertoire of Internal-Functional emotion regulation strategies, which have been identified here as a relative weakness.

Given that many authors propose that individuals develop a stable style of emotion regulation which is then internalised and less amenable to change (e.g. Thompson, 1994), it is likely that therapeutic work with the aim of enhancing emotion regulation skills and working towards change in a dysfunctional style of emotion regulation will need to be undertaken over a reasonably long-term, in order to achieve significant progress.
4.7 Conclusions.

This study has provided some cautious support for the central propositions of an emotion regulation model of comorbid substance misuse and schizophrenia, such as Blanchard et al.'s (2000). The propositions of particular relevance are, briefly, that differences in emotion experience and the way individuals regulate their emotions confer a significant degree of excess vulnerability to substance misuse in this group and differentiate comorbid groups from individuals with schizophrenia who do not misuse substances. This study has provided some qualified support for these core propositions, although due to limitations in the design of the study, these findings must be interpreted with caution. The results of this study are by no means exclusively consistent with an emotion regulation model of comorbidity and require development in future research studies to clarify the status of an emotion regulation model. Also, due to the exploratory nature of much of the analyses reported, these findings have not arisen in a strict hypothesis testing framework and ought to be replicated under such conditions. Particularly imperative is validation of the group differences identified in larger samples, with groups matched for age and gender composition, in order to properly evaluate the influence of these demographic variables on the emotion and emotion regulation variables of interest.

A great deal of exploration is still required in the area of emotion regulation and comorbidity and potential avenues have been identified for future development—some of which would address shortcomings identified in the design of the present study. Research into emotion regulation in schizophrenia and comorbid substance misuse is at a
very early stage and, although this initial study, together with previous research has supported the relevance of these factors to substance misuse in the context of psychosis and treating these co-occurring conditions, these findings need to be built on in order that the relevance of an emotion regulation model can be firmly established. The challenges involved in developing research in this area arise in part from the complexity and accessibility of the constructs in question and the relatively early stage of emotion regulation theory in general. Nonetheless, indications are that attending to emotion experience and regulation may provide valuable insights into substance misuse in the context of schizophrenia, which could contribute to further development of effective treatment approaches.
5. REFERENCES.


Flack & J. D. Laird (Eds.), *Emotions in Psychopathology: Theory and Research* (pp.315-322). Oxford: Oxford University Press.


6. APPENDICES.

Appendix A  DrugCheck measure.
Appendix B  Positive and Negative Affect Scales.
Appendix C  Emotion Regulation Questionnaire.
Appendix D  Child and Adolescent Emotion Regulation Questionnaire.
Appendix E  Results of bivariate correlations between age and measured variables.
Appendix F  Histograms based on current substance misuse status groups.
Appendix G  Indexes of skew and kurtosis and conversion to z scores – current and lifetimes groups.
Appendix H  Comparison of scoring on the PANAS in this study (current misuse status) to previous research findings.
Appendix I  Comparison of scoring on the ERQ in this study (current misuse status) to previous research findings.
Appendix J  Summary of subscale analyses for the CA-ERQ.
Appendix K  Descriptive statistics for comparison of current, past and no misuse groups.
Appendix L  Boxplots for comparison of current, past and no misuse groups.
Appendix M  Histograms for comparison of current, past and no misuse groups.
Appendix N  Results of t tests comparing scoring on measures for current and past misuse groups.
Appendix O  Analysis of ERQ item 4.
Appendix P  Summary of results of analyses of gender as a covariate – lifetime groups.
Appendix Q  Confirmation of approval from NHS Local Research Ethics Committee.
Appendix R  Study information sheet – Community Outreach Team version.
**DrugCheck**

**Patient's Name:** ___________________________  **DOB:** ___________________________

**Service:** ___________________________  **UR:** ___________________________

**RSH Screen Completed by:** ___________________________  **Diagnosis:** ___________________________

---

**INSTRUCTIONS FOR CLINICIANS:**

*By using different markers on the form, indicate at least 2 sources of the information used to complete this assessment. For each source, use a different marker and indicate this below (i.e., red and blue pens etc)*

Patient: ____________  File/Treating Team: ____________  Relative: ____________  Other: ____________

---

**Q. “During the last 3 months have you had any….”**

<table>
<thead>
<tr>
<th><strong>FREQUENCY</strong></th>
<th><strong>QUANTITY</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Q. “How often have you had…”</strong></td>
<td><strong>Q. “How much do you usually have…”</strong></td>
</tr>
<tr>
<td><strong>Tea, coffee or cola drinks?</strong></td>
<td><strong>Tea/coffee:</strong> cups per day</td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td><strong>Cola drinks – coke, pepsi:</strong> cans per day</td>
</tr>
<tr>
<td><strong>Alcoholic drinks?</strong></td>
<td><strong>Brand:</strong> ………………………… <strong>Type:</strong> …………………………</td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td><strong>Cola drinks – coke, pepsi:</strong> cans per day</td>
</tr>
<tr>
<td>If yes, complete the AUDIT (over page)</td>
<td><strong>Sleeping tablets or sedatives?</strong></td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td>(like valium or normison)</td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td><strong>Brand:</strong> ………………………… <strong>Type:</strong> …………………………</td>
</tr>
<tr>
<td><strong>Cigarettes?</strong></td>
<td><strong>Sleeping tablets or sedatives?</strong></td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td>(like valium or normison)</td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td><strong>Brand:</strong> ………………………… <strong>Type:</strong> …………………………</td>
</tr>
<tr>
<td><strong>Other painkillers?</strong></td>
<td><strong>Other painkillers?</strong></td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td>(like valium or normison)</td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td><strong>Brand:</strong> ………………………… <strong>Type:</strong> …………………………</td>
</tr>
<tr>
<td><strong>Marijuana, cannabis, or hash?</strong></td>
<td><strong>Other painkillers?</strong></td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td>(like valium or normison)</td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td><strong>Brand:</strong> ………………………… <strong>Type:</strong> …………………………</td>
</tr>
<tr>
<td><strong>Drugs you sniff, like petrol/glue?</strong></td>
<td><strong>Marijuana, cannabis, or hash?</strong></td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td>(like valium or normison)</td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td><strong>Brand:</strong> ………………………… <strong>Type:</strong> …………………………</td>
</tr>
<tr>
<td><strong>Drugs like LSD?</strong></td>
<td><strong>Drugs you sniff, like petrol/glue?</strong></td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td>(like valium or normison)</td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td><strong>Brand:</strong> ………………………… <strong>Type:</strong> …………………………</td>
</tr>
<tr>
<td><strong>Speed, ecstasy, crack or cocaine?</strong></td>
<td><strong>Drugs like LSD?</strong></td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td>(like valium or normison)</td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td><strong>Brand:</strong> ………………………… <strong>Type:</strong> …………………………</td>
</tr>
<tr>
<td><strong>Heroin, morphine or methadone?</strong></td>
<td><strong>Speed, ecstasy, crack or cocaine?</strong></td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td>(like valium or normison)</td>
</tr>
<tr>
<td>□ No □ Yes</td>
<td><strong>Brand:</strong> ………………………… <strong>Type:</strong> …………………………</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td><strong>Heroin, morphine or methadone?</strong></td>
</tr>
<tr>
<td>---------------</td>
<td>-----------------</td>
</tr>
<tr>
<td>---------------</td>
<td>-----------------</td>
</tr>
</tbody>
</table>

**Q. “You said that you have been using….(summarize the drugs that were identified from the list above), which of these drugs have caused you the most problems or hassles in the last 3 months?**

Take into consideration the various risk factors associated with the substances the patient is presently using & circle the most problematic drugs based on ALL available information.
**PROBLEM LIST**

Q. "In the last 3 months...... (use the most problematic drug in this section)"

1. Did (substance) cause any money problems for you?  
   0 No  1 A bit  2 A lot
2. Did (substance) make you have problems at work, or at school (Tafe/University/ training courses)?  
   0 No  1 A bit  2 A lot
3. Did you have housing problems because of (substance)?  
   0 No  1 A bit  2 A lot
4. Were there problems at home or with your family because of (substance)?  
   0 No  1 A bit  2 A lot
5. Has (substance) caused any trouble with the law, or the police?  
   0 No  1 A bit  2 A lot
6. Has (substance) caused any health problems or injuries?  
   0 No  1 A bit  2 A lot
7. Have you done anything ‘risky’ or ‘outrageous’ after using (substance)?  
   eg. driving under the influence; unprotected sex; sharing needles;  
   other..................................................(circle risky behaviours)

Q. "Did your use of (substance) in the last 3 months result in you......”

9. Being uninterested in your usual activities?  
   0 No  1 A bit  2 A lot
10. Feeling depressed?  
    0 No  1 A bit  2 A lot
11. Being suspicious or distrustful of others?  
    0 No  1 A bit  2 A lot
12. Having strange thoughts?  
    0 No  1 A bit  2 A lot
13. Missing doses of medication?  
    0 No  1 A bit  2 A lot

**THE SEVERITY OF DEPENDENCE SCALE (SDS)**

Q. "During the past 3 months......”

1. Did you ever think your use of (substance) was out of control?  
   0 □ never/almost never  1 □ sometimes  2 □ often  3 □ always/nearly always
2. Did the prospect of missing a fix (or dose) or not chasing, make you anxious or worried?  
   0 □ never/almost never  1 □ sometimes  2 □ often  3 □ always/nearly always
3. Did you worry about your use of (substance)?  
   0 □ never/almost never  1 □ sometimes  2 □ often  3 □ always/nearly always
4. Did you wish you could stop?  
   0 □ not difficult  1 □ quite difficult  2 □ very difficult  3 □ impossible
5. How difficult did you find it to stop, or go without (substance)?  
   0 □ not difficult  1 □ quite difficult  2 □ very difficult  3 □ impossible

**READINESS TO CHANGE:**

Do you want to change your use of (substance) right now?  
0 □ no  1 □ probably not  2 □ unsure  3 □ possibly  4 □ definitely

**CONFIDENCE TO CHANGE:**

Do you think you could change your use of (substance) now if you wanted to?  
0 □ definitely could not  1 □ probably could not  2 □ unsure  3 □ probably could  4 □ definitely could
Please place a mark in the box next to your answer

1. How often do you have a drink containing alcohol?
   □ never □ monthly or less □ once a week □ 2 to 4 times a week □ 5 or more times a week

2. How many 'standard drinks' (see below) do you have on a typical day when you are drinking?
   □ 1 □ 2 □ 3 or 4 □ 5 or 6 □ 7 or more

3. How often do you have six or more drinks on one occasion?
   □ never □ less than monthly □ monthly □ weekly □ daily or almost daily

4. How often during the last 3 months have you found that you were not able to stop drinking once you had started?
   □ never □ less than monthly □ monthly □ weekly □ daily or almost daily

5. How often during the last 3 months have you failed to do what was normally expected from you because of your drinking?
   □ never □ less than monthly □ monthly □ weekly □ daily or almost daily

6. How often during the last 3 months have you needed an alcoholic drink in the morning to get yourself going after a heavy drinking session?
   □ never □ less than monthly □ monthly □ weekly □ daily or almost daily

7. How often during the last 3 months have you had a feeling of guilt or remorse after drinking?
   □ never □ less than monthly □ monthly □ weekly □ daily or almost daily

8. How often during the last 3 months have you been unable to remember what happened the night before because you had been drinking?
   □ never □ less than monthly □ monthly □ weekly □ daily or almost daily

9. Have you or someone else been injured as a result of your drinking?
   □ no □ yes, but not in the last 3 months □ yes, during the last 3 months

10. Has a relative or friend, or a doctor or other health worker been concerned about your drinking or suggested you cut down?
    □ no □ yes, but not in the last 3 months □ yes, during the last 3 months

TOTAL AUDIT SCORE = □

HAZARDOUS/HARMFUL LEVELS YES/NO

Key to AUDIT scoring:
For questions 1-8 responses are scored 0, 1, 2, 3, 4
For questions 9-10 responses are scored 0, 2; 4
- Males: a score of ≥ 7 suggests a pattern of hazardous or harmful drinking.
- Females: a score of ≥ 6 suggests a pattern of hazardous or harmful drinking.
- A score of > 13 for both sexes indicates that the person is likely to be alcohol dependent.

NOTE: A schooner of normal strength beer contains about 2 standard drinks; a bottle about 3. The average light beer is about half the strength of normal beer.
Appendix B: Positive and Negative Affect Schedule (Watson et al., 1988).

The Positive and Negative Affect Schedule

This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you generally feel this way, that is, how you feel on the average. Use the following scale to record your answers.

<table>
<thead>
<tr>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very slightly or not at all</td>
<td>A little</td>
<td>Moderately</td>
<td>Quite a bit</td>
<td>Extremely</td>
</tr>
</tbody>
</table>

| Interested | Irritable |
| Distressed | Alert |
| Excited | Ashamed |
| Upset | Inspired |
| Strong | Nervous |
| Guilty | Determined |
| Scared | Attentive |
| Hostile | Jittery |
| Enthusiastic | Active |
| Proud | Afraid |

Thank you.
Appendix C: The Emotion Regulation Questionnaire (Gross & John, 2003).

**EMOTION REGULATION QUESTIONNAIRE**

We would like to ask you some questions about your emotional life, in particular, how you control (that is, regulate and manage) your emotions.

The questions below involve two distinct aspects of your emotional life. One is your emotional experience, or what you feel like inside. The other is your emotional expression, or how you show your emotions in the way you talk, gesture, or behave.

Although some of the following questions may seem similar to one another, they differ in important ways.

Please choose a number from the following scale and place it in the box next to each statement:

```
1------------2-------------3--------------4---------------5---------------6---------------7
Strongly disagree neutral Strongly agree
```

1. When I want to feel more positive emotion (such as joy or amusement), I __________ change what I’m thinking about.

2. I keep my emotions to myself.

3. When I want to feel less negative emotion (such as sadness or anger), I __________ change what I’m thinking about.

4. When I am feeling positive emotions, I am careful not to express them.

5. When I’m faced with a stressful situation, I make myself think about it in a way that helps me stay calm.

6. I control my emotions by not expressing them.

7. When I want to feel more positive emotion, I __________ change the way I’m thinking about the situation.

8. I control my emotions by changing the way I think about the situation I’m in.

9. When I am feeling negative emotions, I make sure not to express them.

10. When I want to feel less negative emotion, I __________ change the way I’m thinking about the situation.

THE EMOTION REGULATION QUESTIONNAIRE

We all experience lots of different feelings or emotions. For example, different things in our lives make us feel happy, sad, angry and so on...

The following questions ask you to think about how often you do certain things in response to your emotions. You do not have to think about specific emotions but just how often you generally do the things listed below.

Please tick the box that best reflects how often you do the thing described.

We all respond to our emotions in different ways so there are no right or wrong answers.

<table>
<thead>
<tr>
<th>In GENERAL how do you respond to your emotions?</th>
<th>Never</th>
<th>Seldom</th>
<th>Often</th>
<th>Very Often</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I try to see a positive side</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>2. I talk to someone about how I feel</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>3. I pretend the situation doesn't exist</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>4. I take my feelings out on others verbally (e.g. shouting, arguing)</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>5. I eat too much</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>6. I seek physical contact from friends or family (e.g. a hug, hold hands)</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
</tbody>
</table>
**In GENERAL how do you respond to your emotions?**

<table>
<thead>
<tr>
<th></th>
<th>Never</th>
<th>Seldom</th>
<th>Often</th>
<th>Very Often</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>7. I review (rethink) my thoughts or beliefs</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>8. I harm or punish myself in some way</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>9. I do something energetic (e.g. play sport, go for a walk)</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>10. I avoid everything that makes me feel bad</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>11. I dwell on my thoughts and feelings (e.g. It goes round and round in my head and I can't stop it)</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>12. I change my environment (e.g. go outside, leave the room)</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
</tbody>
</table>
In GENERAL how do you respond to your emotions?

<table>
<thead>
<tr>
<th></th>
<th>Never</th>
<th>Seldom</th>
<th>Often</th>
<th>Very Often</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>13. I drink or take drugs alone</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>14. I ask others for advice</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>15. I review (rethink) my goals or plans</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>16. I take my feelings out on others physically (e.g. fighting, lashing out)</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>17. I stop myself from eating</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>18. I put the situation into perspective</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>19. I concentrate on a pleasant activity</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
</tbody>
</table>

In GENERAL how do you respond to your emotions?

<table>
<thead>
<tr>
<th></th>
<th>Never</th>
<th>Seldom</th>
<th>Often</th>
<th>Very Often</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>20. I help others in order to make myself feel better</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>21. I force myself to feel something else</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>22. I do something peaceful and relaxing</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>23. I try to make others feel bad (e.g. being rude, ignoring them)</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>24. I think about people better off and make myself feel worse</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
</tbody>
</table>
In GENERAL how do you respond to your emotions?

<table>
<thead>
<tr>
<th></th>
<th>Never</th>
<th>Seldom</th>
<th>Often</th>
<th>Very Often</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>25. I plan what I could do better next time</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>26. I keep this feeling to myself until it's appropriate for me to express it</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>27. I resign myself to my fate</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>28. I keep the feeling locked up inside</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>29. I think about people worse off than myself</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>30. I bully other people (e.g. saying nasty things to them, hitting them)</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>31. I take my feelings out on objects around me (e.g. deliberately causing damage to my house, school or outdoor things)</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>32. Things feel unreal (e.g. I feel strange, things around me feel strange, I daydream)</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
</tbody>
</table>

THANK YOU FOR COMPLETING THIS QUESTIONNAIRE

Although the original pilot version of the CA-ERQ was used in data collection in this study (32 items), the final validated 19 item version was used for the purposes of analysis. The relevant items were extracted and totalled using SPSS to provide total scores for each subscale. The relevant items are detailed below in Table 17.

Table 17: Subscale structure of the final (19 item) version of the CAERQ.

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Internal-Functional</th>
<th>Internal-Dysfunctional</th>
<th>External-Functional</th>
<th>External-Dysfunctional</th>
</tr>
</thead>
<tbody>
<tr>
<td>Items</td>
<td>7, 15, 25, 18, 19.</td>
<td>8, 11, 24, 28, 32.</td>
<td>2, 6, 9, 14.</td>
<td>4, 16, 23, 30, 31.</td>
</tr>
<tr>
<td>Scale alpha</td>
<td>0.758</td>
<td>0.716</td>
<td>0.659</td>
<td>0.757</td>
</tr>
<tr>
<td>Range of scoring</td>
<td>5-25</td>
<td>5-25</td>
<td>4-20</td>
<td>5-25</td>
</tr>
</tbody>
</table>
Appendix E: Table 18 - Results of bivariate correlations between age and measured variables.

<table>
<thead>
<tr>
<th>Variable</th>
<th>$r$</th>
<th>$p$</th>
<th>$N$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive Affect Total (PANAS)</td>
<td>.135</td>
<td>&gt;.05</td>
<td>40</td>
</tr>
<tr>
<td>Negative Affect Total (PANAS)</td>
<td>-.303</td>
<td>&gt;.05</td>
<td>40</td>
</tr>
<tr>
<td>expressively suppressive Total (ERQ)</td>
<td>-.047</td>
<td>&gt;.05</td>
<td>39</td>
</tr>
<tr>
<td>Cognitive Reappraisal Total (ERQ)</td>
<td>-.026</td>
<td>&gt;.05</td>
<td>39</td>
</tr>
<tr>
<td>CA-ERQ subscale totals</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internal-Functional</td>
<td>.074</td>
<td>&gt;.05</td>
<td>40</td>
</tr>
<tr>
<td>Internal-Dysfunctional</td>
<td>-.175</td>
<td>&gt;.05</td>
<td>40</td>
</tr>
<tr>
<td>External-Functional</td>
<td>-.028</td>
<td>&gt;.05</td>
<td>40</td>
</tr>
<tr>
<td>External-Dysfunctional</td>
<td>.163</td>
<td>&gt;.05</td>
<td>40</td>
</tr>
</tbody>
</table>
Appendix F: Histograms For Current Substance Misuse Groups.

Figures 17 & 18: Histograms of PANAS total for the Positive Affect Scale

Figures 19 & 20: Histogram of total scores for PANAS Negative Affect Scale
Figures 21 & 22: Histograms of scores on the Expressive Suppression subscale of the ERQ

Figures 23 & 24: Histograms of scores on the Cognitive Reappraisal subscale of the ERQ
Figures 25 & 26: Histograms of scores on the Internal Functional subscale of the CA-ERQ.

Figures 27 & 28: Histograms of scores on the Internal Dysfunctional subscale of the CA-ERQ.
Figures 29 & 30: Histograms of scores on the External Functional subscale of the CA-ERQ.

Figures 31 & 32: Histograms of scores on the External Dysfunctional subscale of the CA-ERQ.
Appendix G: Summary data for skewness and kurtosis.

Table 19: Indexes of Skewness and z scores with critical value for significant departure from normality – Current substance use groups.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Scale/ Variable</th>
<th>Group</th>
<th>Skewness Index</th>
<th>Z score Converted following formula in Clark-Carter (2004)</th>
<th>Critical Value (+/-) which preserves α at 0.01</th>
<th>Significant Departure From Normality?</th>
</tr>
</thead>
<tbody>
<tr>
<td>PANAS</td>
<td>Positive Total</td>
<td>Comparison</td>
<td>0.288</td>
<td>0.526</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid</td>
<td>0.352</td>
<td>0.643</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Negative Total</td>
<td>Comparison</td>
<td>0.602</td>
<td>1.099</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid</td>
<td>0.530</td>
<td>0.968</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td>ERQ</td>
<td>Expressive Suppression</td>
<td>Comparison</td>
<td>-0.126</td>
<td>-0.230</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>Comorbid</td>
<td>-0.459</td>
<td>-0.817</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Cognitive Reappraisal</td>
<td>Comparison</td>
<td>-0.124</td>
<td>-0.226</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>Comorbid</td>
<td>0.446</td>
<td>0.794</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td>CA-ERQ</td>
<td>Internal- Functional</td>
<td>Comparison</td>
<td>0.391</td>
<td>0.714</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid</td>
<td>0.361</td>
<td>0.659</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Internal- Dysfunctional</td>
<td>Comparison</td>
<td>0.076</td>
<td>0.139</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid</td>
<td>-0.280</td>
<td>-0.511</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>External- Functional</td>
<td>Comparison</td>
<td>-1.140</td>
<td>-2.56</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid</td>
<td>0.795</td>
<td>1.451</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>External- Dysfunctional</td>
<td>Comparison</td>
<td>0.891</td>
<td>1.627</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid</td>
<td>0.753</td>
<td>1.375</td>
<td>2.58</td>
<td>x</td>
</tr>
</tbody>
</table>

Table 20: Indexes of Kurtosis and z scores with critical value for significant departure from normality – Current substance use groups.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Scale/ Variable</th>
<th>Group</th>
<th>Kurtosis Index</th>
<th>Z score Converted following formula in Clark-Carter (2004)</th>
<th>Critical Value (+/-) which preserves α at 0.01</th>
<th>Significant Departure From Normality?</th>
</tr>
</thead>
<tbody>
<tr>
<td>PANAS</td>
<td>Positive Total</td>
<td>Comparison</td>
<td>-0.769</td>
<td>-0.702</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid</td>
<td>0.533</td>
<td>0.487</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Negative Total</td>
<td>Comparison</td>
<td>-0.259</td>
<td>-0.478</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid</td>
<td>-1.726</td>
<td>-1.576</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td>ERQ</td>
<td>Expressive Suppression</td>
<td>Comparison</td>
<td>-0.543</td>
<td>-0.496</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>Comorbid</td>
<td>-0.022</td>
<td>-0.020</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Cognitive Reappraisal</td>
<td>Comparison</td>
<td>-0.702</td>
<td>-0.641</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>Comorbid</td>
<td>0.586</td>
<td>0.521</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td>CA-ERQ</td>
<td>Internal- Functional</td>
<td>Comparison</td>
<td>.235</td>
<td>.215</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid</td>
<td>-.144</td>
<td>.131</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Internal- Dysfunctional</td>
<td>Comparison</td>
<td>-.799</td>
<td>-.729</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid</td>
<td>-.273</td>
<td>.249</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>External- Functional</td>
<td>Comparison</td>
<td>-1.407</td>
<td>-1.284</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid</td>
<td>.831</td>
<td>.759</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>External- Dysfunctional</td>
<td>Comparison</td>
<td>-.510</td>
<td>-.466</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid</td>
<td>-.766</td>
<td>-.699</td>
<td>2.58</td>
<td>x</td>
</tr>
</tbody>
</table>
Table 21: Indexes of Skewness and z scores with critical value for significant departure from normality – Lifetime substance use groups.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Scale/ Variable</th>
<th>Group</th>
<th>Skewness Index</th>
<th>Converted following formula in Clark-Carter (2004)</th>
<th>Critical Value (+/-) which preserves α at 0.01</th>
<th>Significant Departure From Normality?</th>
</tr>
</thead>
<tbody>
<tr>
<td>PANAS</td>
<td>Positive Total</td>
<td>Comparison B</td>
<td>0.052</td>
<td>0.074</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid B</td>
<td>0.436</td>
<td>0.942</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Negative Total</td>
<td>Comparison B</td>
<td>0.700</td>
<td>0.990</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid B</td>
<td>0.255</td>
<td>0.551</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td>ERQ</td>
<td>Expressive</td>
<td>Comparison B</td>
<td>-0.117</td>
<td>-0.165</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Suppression</td>
<td>Comorbid B</td>
<td>-0.212</td>
<td>-0.450</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>Comparison B</td>
<td>-0.256</td>
<td>0.362</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid B</td>
<td>0.455</td>
<td>0.965</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td>CA-ERQ</td>
<td>Internal-</td>
<td>Comparison B</td>
<td>-0.112</td>
<td>-2.42</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Functional</td>
<td>Comorbid B</td>
<td>-0.014</td>
<td>-0.20</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid B</td>
<td>-0.139</td>
<td>-0.300</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Expressive</td>
<td>Comparison B</td>
<td>-0.443</td>
<td>-0.626</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Suppression</td>
<td>Comorbid B</td>
<td>-0.908</td>
<td>-0.642</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>Comparison B</td>
<td>-0.369</td>
<td>-0.391</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid B</td>
<td>-0.092</td>
<td>-0.701</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>-0.388</td>
<td>-0.412</td>
<td>2.58</td>
<td>x</td>
</tr>
</tbody>
</table>

Table 22: Indexes of Kurtosis and z scores with critical value for significant departure from normality – Lifetime substance use groups.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Scale/ Variable</th>
<th>Group</th>
<th>Kurtosis Index</th>
<th>Converted following formula in Clark-Carter (2004)</th>
<th>Critical Value (+/-) which preserves α at 0.01</th>
<th>Significant Departure From Normality?</th>
</tr>
</thead>
<tbody>
<tr>
<td>PANAS</td>
<td>Positive Total</td>
<td>Comparison B</td>
<td>-0.601</td>
<td>0.425</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid B</td>
<td>0.279</td>
<td>0.301</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Negative Total</td>
<td>Comparison B</td>
<td>0.469</td>
<td>0.332</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid B</td>
<td>-0.358</td>
<td>-0.387</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td>ERQ</td>
<td>Expressive</td>
<td>Comparison B</td>
<td>-0.908</td>
<td>-0.642</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Suppression</td>
<td>Comorbid B</td>
<td>-0.369</td>
<td>-0.391</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>Comparison B</td>
<td>-0.992</td>
<td>-0.701</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comorbid B</td>
<td>-0.388</td>
<td>-0.412</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td>CA-ERQ</td>
<td>Internal-</td>
<td>Comparison B</td>
<td>-.796</td>
<td>-.563</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Functional</td>
<td>Comorbid B</td>
<td>.739</td>
<td>.798</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comparison B</td>
<td>.061</td>
<td>.043</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Dysfunctional</td>
<td>Comorbid B</td>
<td>-.981</td>
<td>-1.060</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comparison B</td>
<td>-1.030</td>
<td>-.728</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Functional</td>
<td>Comorbid B</td>
<td>.300</td>
<td>.324</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Comparison B</td>
<td>.251</td>
<td>.177</td>
<td>2.58</td>
<td>x</td>
</tr>
<tr>
<td></td>
<td>Dysfunctional</td>
<td>Comorbid B</td>
<td>-.798</td>
<td>-.862</td>
<td>2.58</td>
<td>x</td>
</tr>
</tbody>
</table>
Appendix H: Comparison of scoring on the PANAS in this sample to previous research data.

Table 23: Summary of descriptive data for scoring on the PANAS in the present study and previous relevant studies.

<table>
<thead>
<tr>
<th>GROUP</th>
<th>POSITIVE AFFECT SCALE</th>
<th>NEGATIVE AFFECT SCALE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Std Dev</td>
</tr>
<tr>
<td>Results from this study</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No misuse</td>
<td>26.60</td>
<td>6.37</td>
</tr>
<tr>
<td>Current misuse</td>
<td>25.45</td>
<td>6.45</td>
</tr>
<tr>
<td>Normative Data</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Undergraduates-USA (Watson et al, 1988)</td>
<td>35.00</td>
<td>6.40</td>
</tr>
<tr>
<td>Inpatients-USA (Watson et al, 1988)</td>
<td>32.50</td>
<td>7.50</td>
</tr>
<tr>
<td>Outpatients-USA excluding psychoses (Jolly et al, 1994)</td>
<td>23.90</td>
<td>9.40</td>
</tr>
</tbody>
</table>

For both groups in this study mean scores for positive and negative affect are more similar than is the case in the previous research groups. Participants in this study don’t show the pattern of the other psychiatric outpatient sample of mean negative affect exceeding mean positive affect. Jolly et al’s (1994) sample excluded individuals with psychoses and may have been characterised by depression and anxiety diagnoses where such a pattern may be more typical.

Z scores were used to assess whether the mean scores for either group in this study were significantly different from those identified in a non-clinical comparison group using data from Watson et al’s (1988) undergraduate sample (See Table 23). Both groups were more than one standard deviation lower on the positive affect scale and over three quarters of a standard deviation higher for the negative affect scale but the differences were statistically significant.

Table 24: Z Scores For Comparisons Of PANAS Group Mean Scores To Mean Scores From An Undergraduate Sample.

<table>
<thead>
<tr>
<th>Group</th>
<th>PANAS Subscale</th>
<th>Z score</th>
<th>Significance of difference (1 tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No misuse (N=20)</td>
<td>Positive Affect</td>
<td>-1.31</td>
<td>p&gt;0.05</td>
</tr>
<tr>
<td>Misuse (N=20)</td>
<td>Scale</td>
<td>-1.49</td>
<td>p&gt;0.05</td>
</tr>
<tr>
<td>No misuse (N=20)</td>
<td>Negative Affect</td>
<td>0.831</td>
<td>p&gt;0.05</td>
</tr>
<tr>
<td>Misuse (N=20)</td>
<td>Scale</td>
<td>0.890</td>
<td>p&gt;0.05</td>
</tr>
</tbody>
</table>

In summary, relative to the profiles of previous research groups on the PANAS, the present groups are not significantly different from a non-clinical comparison group. However, the current profile is dissimilar from that identified with another psychiatric outpatient group (who differed in terms of diagnostic composition) in that scoring did not indicate greater negative than positive affect.
Appendix I: Comparison of scoring on the ERQ in the present study with previous research.

The available data for comparison (Gross & John, 2003) of scores on the ERQ are in the form of mean scores rather than total scores and are disaggregated by gender (See Table 25). For the purposes of these brief comparisons the male means will be used as the majority of the present sample is male.

Examination of the mean scores indicated that all participants in the current study tended to respond slightly more neutrally than the comparison group whose mean scores indicate a degree more disagreement (See Table 25). However, these differences were small and were not significant when z scores which treated the Gross and John (2003) data as a control. This suggests that use of expressive suppression, as measured by the ERQ, does not differ significantly between this non-clinical group and the outpatients who participated in this study when comparing overall group means. However, more detailed analysis comparing individual scores may produce different results.

Table 25: Comparisons Of Current Data Set To Normative Data For The Expressive Suppression Subscale.

<table>
<thead>
<tr>
<th>Study</th>
<th>Group</th>
<th>Mean</th>
<th>SD</th>
<th>Z score</th>
<th>Significance of difference (1 tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gross &amp; John (2003)</td>
<td>Male</td>
<td>3.64</td>
<td>1.11</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Undergraduate Sample (N=39)</td>
<td>Female</td>
<td>3.4</td>
<td>1.18</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Present study</td>
<td>No current misuse (N=20)</td>
<td>4.09</td>
<td>1.49</td>
<td>0.41</td>
<td>p&gt;0.05</td>
</tr>
<tr>
<td></td>
<td>Current misuse (N=19)</td>
<td>4.5</td>
<td>1.19</td>
<td>0.77</td>
<td>p&gt;0.05</td>
</tr>
</tbody>
</table>
Appendix J: Examination of the structure of the Child and Adolescent Emotion Regulation Questionnaire (CA-ERQ).

This CA-ERQ is based on a theoretical model of emotion regulation strategies where the two dimensions are the nature of the resources used in the strategy (internal or external to the individual) and the functionality of the strategy (functional versus dysfunctional), based on typical outcomes. Although it is beyond the scope of this investigation to conduct a full validation of the CA-ERQ with this population, it is necessary to consider whether the structure of the subscales has been adequately retained to allow further analyses.

Table 26: Model of emotion regulation strategies embodied in CA-ERQ.

<table>
<thead>
<tr>
<th>Dimension</th>
<th>Functional</th>
<th>Dysfunctional</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internal</td>
<td>Internal-Functional</td>
<td>Internal-Dysfunctional</td>
</tr>
<tr>
<td>External</td>
<td>External-Functional</td>
<td>External-Dysfunctional</td>
</tr>
</tbody>
</table>

To investigate whether the subscales and dimensions had transferred meaningfully to this population, an exhaustive series of bivariate correlations were conducted between subscales.

Table 27: Results of Correlations Between Subscales of the CA-ERQ (Pearson’s Product Moment Correlation Coefficient).

<table>
<thead>
<tr>
<th>Dimension 1</th>
<th>Dimension 2</th>
<th>N</th>
<th>r</th>
<th>2 tailed Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internal-Functional</td>
<td>Internal-Functional</td>
<td>40</td>
<td>.285</td>
<td>p&gt;.05</td>
</tr>
<tr>
<td>Internal-Functional</td>
<td>Internal-Dysfunctional</td>
<td>40</td>
<td>.362</td>
<td>p&lt;.05</td>
</tr>
<tr>
<td>Internal-Functional</td>
<td>External-Functional</td>
<td>40</td>
<td>-.002</td>
<td>p&gt;.05</td>
</tr>
<tr>
<td>Internal-Functional</td>
<td>External-Dysfunctional</td>
<td>40</td>
<td>.056</td>
<td>p&gt;.05</td>
</tr>
<tr>
<td>Internal-Dysfunctional</td>
<td>External-Functional</td>
<td>40</td>
<td>.112</td>
<td>p&gt;.05</td>
</tr>
<tr>
<td>Internal-Dysfunctional</td>
<td>External-Dysfunctional</td>
<td>40</td>
<td>-.130</td>
<td>p&gt;.05</td>
</tr>
</tbody>
</table>

Although the correlations between the internal and external-functional scales is statistically significant, the strength of this association as demonstrated by the r value is acceptably low. Indeed, in the original studies undertaken for the development of this measure the two functional and the two dysfunctional scales were found to correlate significantly and the model developed following factor analysis allowed the two pairs of scales to co-vary. All other r values are very low and indicate that the structure of this measure has indeed been maintained when applied to this new population. Therefore it is considered appropriate to proceed with further analysis using the individual subscale scores from the CA-ERQ.
Appendix K: Descriptive statistics for comparison of three substance misuse status groups – Current misuse, past misuse and no history of misuse.

Table 28: Summary of descriptive statistics.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Group</th>
<th>Mean</th>
<th>SD</th>
<th>Variance</th>
<th>Median</th>
<th>Skewness Index</th>
<th>Kurtosis Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>PANAS Positive Affect</td>
<td>No misuse (N=12)</td>
<td>28.58</td>
<td>6.331</td>
<td>40.083</td>
<td>29.00</td>
<td>.502</td>
<td>-.601</td>
</tr>
<tr>
<td></td>
<td>Past misuse (N=8)</td>
<td>23.63</td>
<td>5.502</td>
<td>30.268</td>
<td>22.00</td>
<td>.613</td>
<td>-.760</td>
</tr>
<tr>
<td></td>
<td>Current misuse (N=20)</td>
<td>25.45</td>
<td>6.452</td>
<td>41.269</td>
<td>24.50</td>
<td>.352</td>
<td>.533</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>No misuse (N=12)</td>
<td>24.50</td>
<td>11.229</td>
<td>126.091</td>
<td>25.50</td>
<td>.700</td>
<td>.469</td>
</tr>
<tr>
<td></td>
<td>Past misuse (N=8)</td>
<td>20.75</td>
<td>11.016</td>
<td>121.357</td>
<td>15.50</td>
<td>.600</td>
<td>-.837</td>
</tr>
<tr>
<td></td>
<td>Current misuse (N=20)</td>
<td>23.85</td>
<td>5.905</td>
<td>34.871</td>
<td>22.50</td>
<td>.530</td>
<td>1.726</td>
</tr>
<tr>
<td>ERQ Expression Suppression</td>
<td>No misuse (N=12)</td>
<td>14.50</td>
<td>5.870</td>
<td>34.455</td>
<td>13.50</td>
<td>-.117</td>
<td>-.908</td>
</tr>
<tr>
<td></td>
<td>Past misuse (N=8)</td>
<td>19.13</td>
<td>5.222</td>
<td>27.268</td>
<td>18.00</td>
<td>.243</td>
<td>1.398</td>
</tr>
<tr>
<td></td>
<td>Current misuse (N=19)</td>
<td>18.00</td>
<td>4.733</td>
<td>22.778</td>
<td>19.00</td>
<td>-.459</td>
<td>.022</td>
</tr>
<tr>
<td>Cognitive Reappraisal</td>
<td>No misuse (N=12)</td>
<td>25.00</td>
<td>8.914</td>
<td>79.455</td>
<td>26.50</td>
<td>-.256</td>
<td>-.992</td>
</tr>
<tr>
<td></td>
<td>Past misuse (N=8)</td>
<td>24.63</td>
<td>6.435</td>
<td>41.411</td>
<td>25.50</td>
<td>.317</td>
<td>.473</td>
</tr>
<tr>
<td></td>
<td>Current misuse (N=19)</td>
<td>25.32</td>
<td>8.564</td>
<td>73.339</td>
<td>25.00</td>
<td>.446</td>
<td>-.586</td>
</tr>
<tr>
<td>CA-ERQ Internal-Functional</td>
<td>No misuse (N=12)</td>
<td>14.83</td>
<td>3.380</td>
<td>11.414</td>
<td>14.00</td>
<td>.240</td>
<td>-.796</td>
</tr>
<tr>
<td></td>
<td>Past misuse (N=8)</td>
<td>11.63</td>
<td>2.264</td>
<td>5.125</td>
<td>12.50</td>
<td>-1.350</td>
<td>1.721</td>
</tr>
<tr>
<td></td>
<td>Current misuse (N=20)</td>
<td>12.85</td>
<td>2.134</td>
<td>4.555</td>
<td>13.00</td>
<td>.361</td>
<td>-.144</td>
</tr>
<tr>
<td>Internal-Dysfunctional</td>
<td>No misuse (N=12)</td>
<td>13.83</td>
<td>4.260</td>
<td>18.152</td>
<td>14.50</td>
<td>-.014</td>
<td>.061</td>
</tr>
<tr>
<td></td>
<td>Current misuse (N=20)</td>
<td>12.45</td>
<td>3.620</td>
<td>13.103</td>
<td>12.50</td>
<td>-.280</td>
<td>-.273</td>
</tr>
<tr>
<td>External-Functional</td>
<td>No misuse (N=12)</td>
<td>10.25</td>
<td>2.417</td>
<td>5.841</td>
<td>11.00</td>
<td>-.443</td>
<td>-1.030</td>
</tr>
<tr>
<td></td>
<td>Past misuse (N=8)</td>
<td>9.63</td>
<td>2.669</td>
<td>7.125</td>
<td>8.50</td>
<td>.296</td>
<td>-1.624</td>
</tr>
<tr>
<td></td>
<td>Current misuse (N=20)</td>
<td>9.10</td>
<td>3.110</td>
<td>9.674</td>
<td>9.00</td>
<td>.795</td>
<td>.831</td>
</tr>
<tr>
<td>External-Dysfunctional</td>
<td>No misuse (N=12)</td>
<td>7.08</td>
<td>1.832</td>
<td>3.356</td>
<td>6.50</td>
<td>.918</td>
<td>.251</td>
</tr>
<tr>
<td></td>
<td>Past misuse (N=8)</td>
<td>6.50</td>
<td>2.204</td>
<td>4.857</td>
<td>5.50</td>
<td>1.281</td>
<td>-.242</td>
</tr>
<tr>
<td></td>
<td>Current misuse (N=20)</td>
<td>6.75</td>
<td>1.832</td>
<td>3.355</td>
<td>6.00</td>
<td>.753</td>
<td>-.766</td>
</tr>
</tbody>
</table>
Appendix L: Boxplots for comparisons of substance misuse groups – Current misuse, past misuse and no history of misuse.

Figure 33: Positive Affect Scale - PANAS

Figure 34: Negative Affect Scale - PANAS
Figure 35: Expressive Suppression Subscale – ERQ

Figure 36: Cognitive Reappraisal Subscale – ERQ.
Figure 37: Internal-Functional Scale – CA-ERQ.

Figure 38: Internal-Dysfunctional Scale – CA-ERQ.
Figure 39: External-Functional Scale – CA-ERQ.

Figure 40: External-Dysfunctional Scale – CA-ERQ.
Appendix M: Histograms for comparing the distributions of scores for three groups of substance misuse status – Current, past and no history of misuse.

All the histograms shown below use percentages on the y axis in order to facilitate comparisons between groups of different sizes.

Figures 41-43: Histograms For Scores On The Positive Affect Scale (PANAS).
Figures 44-46: Histograms for scores on the Negative Affect Scale (PANAS).

Figures 50-52: Histograms for scores on the Cognitive Reappraisal scale (ERQ).

Figures 56-58: Histograms for scores on the Internal-Dysfunctional Scale (CA-ERQ).

Figures 62-64: Histograms for scores on the External-Dysfunctional Scale (CA-ERO).
Appendix N: Results of t tests to compare current and past misusers.

Table 29: Summary of t test results.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Variable</th>
<th>d.f.</th>
<th>t</th>
<th>p (2 tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PANAS</td>
<td>Positive Affect Total</td>
<td>26</td>
<td>0.702</td>
<td>&gt;.05</td>
</tr>
<tr>
<td></td>
<td>Negative Affect Total</td>
<td>8.659</td>
<td>0.632</td>
<td>&gt;.05</td>
</tr>
<tr>
<td></td>
<td>(Welch’s t reported as variances differed significantly F=11.005, p&lt;.01)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ERQ</td>
<td>Expressive Suppression</td>
<td>25</td>
<td>0.544</td>
<td>&gt;.05</td>
</tr>
<tr>
<td></td>
<td>Cognitive Reappraisal</td>
<td>25</td>
<td>0.204</td>
<td>&gt;.05</td>
</tr>
<tr>
<td>CA-ERQ</td>
<td>Internal-Functional</td>
<td>26</td>
<td>1.349</td>
<td>&gt;.05</td>
</tr>
<tr>
<td></td>
<td>Internal-Dysfunctional</td>
<td>26</td>
<td>1.131</td>
<td>&gt;.05</td>
</tr>
<tr>
<td></td>
<td>External-Functional</td>
<td>26</td>
<td>0.419</td>
<td>&gt;.05</td>
</tr>
<tr>
<td></td>
<td>External-Dysfunctional</td>
<td>26</td>
<td>0.308</td>
<td>&gt;.05</td>
</tr>
</tbody>
</table>
Appendix O: Analysis of ERQ Item 4 – Expressive suppression of positive emotions.

Given that the between group differences identified are of lower levels of positive emotions and higher use of expressive suppression amongst individuals with current or past substance misuse, it seems particularly relevant to specifically examine item 4 on the ERQ which asks participants to rate their expressive suppression of positive emotions; 'When I am feeling positive emotions I am careful not to express them.' Research has indeed demonstrated that expressive suppression of positive emotions is associated with reduced experience of these emotions.

The full sample of 40 is available for analysis of this item as participant 24 answered item 4 before declining to complete the remainder of the ERQ. Table 30 shows a small difference between the mean scores in the predicted direction of greater suppression in the misuse group, however the higher group mean is close to the neutral rating rather than indicating agreement with the item.

Table 30: Descriptive Statistics For ERQ Item 4

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean</th>
<th>Std Dev</th>
<th>Variance</th>
<th>Median</th>
<th>Skewness Index</th>
<th>Kurtosis Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>No misuse (n=12)</td>
<td>2.33</td>
<td>1.303</td>
<td>1.697</td>
<td>2</td>
<td>0.735</td>
<td>-0.118</td>
</tr>
<tr>
<td>Current or Past Misuse (n=28)</td>
<td>3.61</td>
<td>1.988</td>
<td>3.951</td>
<td>4</td>
<td>0.165</td>
<td>-1.045</td>
</tr>
</tbody>
</table>

Figure 65 below illustrates the between-group differences in the distribution of scores for this particular item. There are no outliers and the no misuse group are concentrated at the lower end of the scale, indicating disagreement, whilst the misuse group are more towards neutral responses, with their range extending to include some individuals who agree with this item.

Although the variances differ marginally more than the recommended factor of two where groups are of uneven size (Clark-Carter, 2004), Levene’s test did not detect a significant difference in variance (F(38)=3.416, p>0.05) and all other conditions for the use of a t test were met. The result confirmed a significant difference between groups on this particular item (t(38)=-2.033, p<0.05), with a large effect size (d=0.9). This supports the possibility that one factor which may contribute to reduced experience of positive affect in the substance misuse group is greater expressive suppression of the experience of positive emotions. However, there was no significant overall correlation between scores on the Expressive Suppression Subscale and the Positive Affect Scale of the PANAS. Further research will be necessary to explore this issue more rigorously.
Figure 65: Boxplot for ERQ Item 4
Appendix P: Summary of results of analyses of gender as a covariate – lifetime groups.

One-way ANCOVAs were used with the subscale scores on the three measures entered as the Dependent Variable, lifetime substance use group entered as a fixed factor and gender entered as a covariate in order to further assess the influence of gender differences between the two groups on the measured emotion and emotion regulation variables. The aim of this analysis being to gain an estimate of whether any significant differences identified between the groups would still have occurred if both groups were equal in the distribution of gender, and also of whether any differences not identified in previous analyses may have come to light had the two groups been matched for gender of the participants.

Table 31: Summary of findings from ANCOVAs treating gender as a covariate.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Scale</th>
<th>Homog. Of Var?</th>
<th>df</th>
<th>F</th>
<th>p</th>
<th>Partial Eta²</th>
<th>Observed Power</th>
</tr>
</thead>
<tbody>
<tr>
<td>PANAS</td>
<td>PA</td>
<td>✓</td>
<td>1, 37</td>
<td>3.469</td>
<td>&gt;.05 (p=.07)</td>
<td>.086 Medium</td>
<td>.442</td>
</tr>
<tr>
<td></td>
<td>NA</td>
<td>✓</td>
<td>1, 37</td>
<td>.053</td>
<td>&gt;.05</td>
<td>.001 Small</td>
<td>.056</td>
</tr>
<tr>
<td>ERQ</td>
<td>ES</td>
<td>✓</td>
<td>1, 36</td>
<td>3.210</td>
<td>&gt;.05 (p=.082)</td>
<td>.082 Medium</td>
<td>.415</td>
</tr>
<tr>
<td></td>
<td>CR</td>
<td>✓</td>
<td>1, 36</td>
<td>.351</td>
<td>&gt;.05</td>
<td>.01 Small-Med</td>
<td>.089</td>
</tr>
<tr>
<td>CA-ERQ</td>
<td>IF</td>
<td>×</td>
<td>1, 37</td>
<td>4.133</td>
<td>&lt;.05</td>
<td>.1 Medium</td>
<td>.508</td>
</tr>
<tr>
<td></td>
<td>ID</td>
<td>✓</td>
<td>1, 37</td>
<td>.827</td>
<td>&gt;.05</td>
<td>.022 Medium</td>
<td>.144</td>
</tr>
<tr>
<td></td>
<td>EF</td>
<td>✓</td>
<td>1, 37</td>
<td>.085</td>
<td>&gt;.05</td>
<td>.002 V small</td>
<td>.059</td>
</tr>
<tr>
<td></td>
<td>ED</td>
<td>✓</td>
<td>1, 37</td>
<td>.847</td>
<td>&gt;.05</td>
<td>.022 Medium</td>
<td>.146</td>
</tr>
</tbody>
</table>
Appendix Q: Confirmation of approval from NHS Local Research Ethics Committee

SL.14 Favourable opinion following consideration of further information
Version 2, October 2004

Fife and Forth Valley Local Research Ethics Committee
Room 507
Hayfield House
Hayfield Road
KIRKCALDY
Fife
KY2 5AH

Telephone: 01592 643355 ext 8976
Facsimile: 01592 648142
L1162 S0501-75
Email: Alison.smit@nhs.net

15 December 2004

Miss Amy Hodgson
Trainee Clinical Psychologist
Fife NHS Department of Clinical Psychology
Stratheden Hospital
Cupar
Fife
KY15 5RR

Dear Miss Hodgson

Full title of study: A study of emotion regulation strategies amongst individuals with Schizophrenia and co-morbid substance use.

REC reference number: 04/S0501/75
Protocol number:

Thank you for your letter of 9 December 2004, responding to the Committee's request for further information on the above research and submitting revised documentation which was considered by the Chairman on 15 December 2004.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised.

The favourable opinion applies to the research site listed on the attached form.

Conditions of approval

The favourable opinion is given provided that you comply with the conditions set out in the attached document. You are advised to study the conditions carefully.

cont'd/2
Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

<table>
<thead>
<tr>
<th>Document Type</th>
<th>Version</th>
<th>Dated:</th>
<th>Date Received:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protocol</td>
<td>1.0</td>
<td>01/10/2004</td>
<td>24/11/2004</td>
</tr>
<tr>
<td>Covering Letter</td>
<td></td>
<td>09/12/2004</td>
<td>14/12/2004</td>
</tr>
<tr>
<td>Copy of Questionnaire</td>
<td></td>
<td></td>
<td>24/11/2004</td>
</tr>
<tr>
<td>Letters of Invitation to Participants</td>
<td>2.0</td>
<td>09/12/2004</td>
<td>14/12/2004</td>
</tr>
<tr>
<td>Letters of Invitation to Participants</td>
<td>1.0</td>
<td>04/11/2004</td>
<td>24/11/2004</td>
</tr>
<tr>
<td>Participant Information Sheet</td>
<td></td>
<td></td>
<td>24/11/2004</td>
</tr>
<tr>
<td>Participant Consent Form</td>
<td>1.0</td>
<td>04/11/2004</td>
<td>24/11/2004</td>
</tr>
<tr>
<td>Supervisor CV</td>
<td></td>
<td></td>
<td>24/11/2004</td>
</tr>
<tr>
<td>Non-validated questionnaire</td>
<td>1.0</td>
<td>17/11/2004</td>
<td>24/11/2004</td>
</tr>
<tr>
<td>Demographic Information Sheet</td>
<td>1.0</td>
<td>14/11/2004</td>
<td>24/11/2004</td>
</tr>
</tbody>
</table>

Management approval

The study should not commence at any NHS site until the local Principal Investigator has obtained final management approval from the R&D Department for the relevant NHS care organisation.

Notification of other bodies

The Committee Administrator will notify the research sponsor that the study has a favourable ethical opinion.
SL14 Favourable opinion following consideration of further information
Version 2, October 2004

Miss Amy Hodgson :3: L1162 S0501-75

Statement of compliance
The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

04/S0501/75 Please quote this number on all correspondence

With the Committee's best wishes for the success of this project,

Yours sincerely,

Chair
Fife and Forth Valley Local Research Ethics Committee

Enclosures
Standard approval conditions
Site approval form (SF1)

cc:
Mick Power, University of Edinburgh, Kennedy Tower (Psychiatry Dept) Royal Edinburgh Hospital EH10 5HF
Single Site application

NHS Fife - Primary Care Trust.

Department of Clinical Psychology and the Community Outreach Team based at Haldane House, Dunfermline.

Fife Local Research Ethics Committee

15/12/2004

Approved by the Chair on behalf of the REC

<table>
<thead>
<tr>
<th>Name</th>
<th>(Name)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Date of Awardable Investigator</td>
<td>Post</td>
</tr>
<tr>
<td>Date of Awardable Investigator</td>
<td>Post</td>
</tr>
<tr>
<td>Site Assessor</td>
<td>Site Assessor</td>
</tr>
<tr>
<td>Research Site</td>
<td>Research Site</td>
</tr>
<tr>
<td>Notes</td>
<td>Notes</td>
</tr>
</tbody>
</table>

The notes column may be used by the main REC to record the early closure of withdrawal of a site (where notified by the Chief Investigator or Sponsor). The submission of the submission of the Principal Investigator for an individual site or any other relevant development. The data should be recorded.

The Principal Investigator of the site will determine the beneficial opinion for this site and inform the REC of the closure or withdrawal of the site. The data should be recorded.
For all studies requiring site-specific assessment, this form is issued by the main REC to the Chief Investigator and sponsor with the favourable opinion and following subsequent notifications from the assessors. For issue 2 onwards, all sites with a favourable opinion are listed in the relevant REC decision letter and following subsequent notifications from the assessors. For all sites requiring site-specific assessment, the form is issued by the main REC to the Chief Investigator and sponsor with the favourable opinion and following subsequent notifications from the assessors. For all sites requiring site-specific assessment, the form is issued by the main REC to the Chief Investigator and sponsor with the favourable opinion and following subsequent notifications from the assessors.

<table>
<thead>
<tr>
<th>Full title of study:</th>
<th>A study of emotion regulation strategies amongst individuals with schizophrenia and comorbid substance use.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chief Investigator:</td>
<td>Miss Amy Hodgson</td>
</tr>
<tr>
<td>REC Reference number:</td>
<td>04/S050175</td>
</tr>
<tr>
<td>Date of issue:</td>
<td>15 December 2004</td>
</tr>
<tr>
<td>Issue number:</td>
<td>1</td>
</tr>
</tbody>
</table>

For all studies requiring site-specific assessment, this form is issued by the main REC to the Chief Investigator and sponsor with the favourable opinion and following subsequent notifications from the assessors. For issue 2 onwards, all sites with a favourable opinion are listed in the relevant REC decision letter and following subsequent notifications from the assessors. For all sites requiring site-specific assessment, the form is issued by the main REC to the Chief Investigator and sponsor with the favourable opinion and following subsequent notifications from the assessors. For all sites requiring site-specific assessment, the form is issued by the main REC to the Chief Investigator and sponsor with the favourable opinion and following subsequent notifications from the assessors.
Appendix R: Study information sheet – Community Outreach Team version.

A study of how people who experience mental health difficulties manage their emotions.

PARTICIPANT INFORMATION SHEET – QUESTIONNAIRE STUDY

I would like to invite you to take part in a research study. Before you decide whether you want to take part it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. If you have any questions or anything is not clear then contact me and we can talk more about the study. Take time to decide whether or not you wish to take part. I will be telephoning you in about one week to find out if you are willing to take part and you can ask me any questions you have about the study then. If you don’t want me to contact you then please let your Care Manager/Support Worker know in the next 3 days.

What is the purpose of the study?

I am carrying out some research to look at how individuals with mental health difficulties experience and cope with different emotions and feelings. I am particularly interested in whether people use alcohol or drugs as a way of coping with difficult feelings. It is important for me to talk to both people who do use alcohol and drugs and those who don’t to see what the important differences are in their experiences. I plan to look at these issues by asking people to fill in some questionnaires at a meeting with me. I may also ask you to meet me a second time for an interview about your experiences.

Why have I been asked to take part?

I will be asking a large number of people who are clients of the Community Outreach Team based at Haldane House in Dunfermline to take part in the study. The fact that I am asking you to take part does not mean that I am making any assumptions about whether or not you drink alcohol or use drugs.

Do I have to take part?

It is up to you to decide whether or not to take part. If you do decide to take part you will be asked to sign a consent form and given a copy of both this information sheet and the consent form to keep. If you decide to take part you are still free to leave the study at any time and you do not have to give a reason for leaving. A decision to leave the study at any time, or not to take part, will not affect the standard of care you receive now or in the future.

What will happen to me if I take part?

I will contact you by telephone about a week after you receive this information to ask you if you are willing to take part. If you agree to take part I will arrange an appointment in the next few weeks either at a clinic in Dunfermline or at the day hospital, whichever is most convenient for you. If it is very difficult for you to travel to one of these places then I will try to arrange somewhere more convenient to meet you or I may come and see you at home with a member of the Community Outreach Team who you already know.

At the appointment I will ask you to fill in two questionnaires about how you deal with different emotions. I will also ask you to fill in two short questionnaires about your mood and to answer some questions about whether and how often you drink alcohol or use any drugs.

I expect that completing these questionnaires will take about 45 minutes to 1 hour. You will be able to take a break at any time if you need to. If there is not time to complete the questionnaires at one appointment I will ask you to come back for a second meeting but this is unlikely and again will be your choice. I will be asking a small number of people to come back for an interview about their experiences. If I ask you to do this I will give you detailed information about what is involved.

What are the possible disadvantages and risks of taking part?

Some of the issues covered by the questionnaires might be difficult for you to discuss. You will be able to ask to stop the appointment or leave the study at any time. If you feel that you need additional support to cope with any issues that arise then, with your permission I will discuss this with the Community Outreach Team.
If it costs you money to travel to the appointment with me then we can offer you a contribution to your travel expenses of up to £2 if you keep your tickets or receipt.

**What are the possible benefits of taking part?**

Although taking part will not change the treatment that you receive we hope that our interest in your experiences and views will be valuable to you. The aim of the study is to increase understanding of how people with mental health difficulties manage their feelings and this may help us to improve treatment and support of individuals like you in the future.

**Will my taking part in this study be kept confidential?**

All information which is collected about you during the course of the research will be kept strictly confidential. Any information about you which leaves the hospital or clinic will have your name and address removed so that you cannot be recognised from it and will be stored in a secure location.

We are obliged to inform both your GP and the Community Outreach Team of your participation in the research as they are involved with your care. However, the information that you give me as part of the study will not be shared with your GP or the Community Outreach Team unless you agree to this.

We take your confidentiality very seriously but I must make you aware that if you give me any information during the study that causes me concern that you or others may be at risk of harm then I am obliged to pass that information on to the relevant professionals.

**What will happen to the results of the research study?**

The results of this research will be written up and submitted to the University of Edinburgh as part of the requirements of my training. I also hope to publish the findings in a journal article in order that the findings can be shared with other professionals. You will not be identifiable in any publication from the study and your name will not be used.

**Who is organising and funding the research?**

This research is being carried out as part of the requirements of a Doctorate in Clinical Psychology at the University of Edinburgh. The University of Edinburgh has approved the research and is acting as the sponsor. It will therefore provide indemnity and/or compensation should you incur any suffering (negligent or non-negligent) as a consequence of taking part in this research.

**Who has reviewed the study?**

The study has been reviewed and approved by the NHS Fife Research Ethics Committee.

If you require any further information then please contact:
Amy Hodgson (Trainee Clinical Psychologist) or Gillian Wilkie (Team Administrator)
Community Outreach Team
Haldane House
Priory Lane
Dunfermline KY12 7DT
Tel: 01383 432725

Thank you for taking the time to read this. I will be telephoning you in about a week to see if you are willing to take part.

This information sheet was issued on Fife Primary Care headed paper in 10 pt type.