What basic emotions are experienced in bipolar disorder and how are they regulated

Louise Carolan

Submitted in part fulfilment of the requirements for the degree of

Doctorate in Clinical Psychology at the University of Edinburgh

October 2009

Acknowledgements

I would like to thank all of the clients and staff at the local hospital, the lithium clinic and in Community Mental Health Teams 3 and 4 for their co-operation, time and effort without their help it would have been impossible to carry out this research.

A special thanks to Mick Power and Elaine Whitefield for their wisdom, support and encouragement. I very much appreciate all that you have done.

Shell, you are such a great friend to me. You are absolutely amazing; thank you so much for everything – you're a star!!

To my family - mum, dad, Sarah and Andy – particularly dad for taking the time to proof read drafts (and not acting too bored!). The belief you lot have in me and the encouragement you have provided has been invaluable.

Last but certainly not least thanks to Stuart (my fiancé!!). I definitely could not have done this without you. You deserve the title just as much as I do! For taking over the house chores during the write up, forcing me to take breaks, putting up with my moans and groans, your computer expertise, proof reading drafts, packing envelopes, believing in me, making me laugh and supporting me in every single way possible – I love you so much.

Contents Page

List of Tables

List of Figures

Abstract

Introduction: There remains a lack of theoretical models which can adequately account for the key features of bipolar disorders (Power, 2005).

Objectives: Firstly, to test the predictions made by the SPAARS model that mania is predominantly characterised by the coupling of happiness with anger, while depression (unipolar and bipolar) primarily comprises of a coupling between sadness and disgust. Secondly, to investigate and compare the coping strategies employed to regulate positive and negative emotion between bipolar, unipolar and control groups.

Design: A cross sectional design was employed to examine the differences within and between the bipolar, unipolar and control groups in the emotions experienced and the strategies used to regulate emotion. Data were analysed using ANOVAs.

Method: Psychiatric diagnoses in the clinical groups were confirmed using the SCID. Current mood state was measured using the BDI-II, STAI and the MAS. The Basic Emotion Scale was used to explore the emotional profiles and the Regulation of Emotion Questionnaire was used to measure coping strategies.

Results: The results confirmed the predictions made by the SPAARS model about the emotions in mania and depression. Elevated levels of disgust were also found in the bipolar group generally. The clinical groups used internal dysfunctional strategies more often than the controls for negative emotion. The bipolar group used external dysfunctional strategies more frequently than the controls for positive emotion.

Conclusion: The results support the predictions made by the SPAARS model and suggest that disgust plays a key role in bipolar disorder. Strengths and limitations are discussed and suggestions for future research are explored.

Word Count: 29,025

CHAPTER ONE - INTRODUCTION

1.1 Brief Introduction

The following chapter presents the background research and rationale for the current study. A literature review was carried out to identify the key research in five areas; the prevalence, epidemiology, course and comorbidity of bipolar and major depressive disorder; the experience of emotion in mania and depression; the similarities and differences between bipolar and major depressive disorder; theories and models of bipolar and major depressive disorder; and coping strategies commonly used to regulate emotion in these disorders. Ovid, PsychInfo and Embase (electronic citation and journal databases that are updated weekly) were accessed to identify key research in these areas. A strict search criteria was used to exclude studies that focussed on the neurological aspects of bipolar disorder since it was beyond the scope of the current study to do this broad area of research justice.

Based on the literature reviewed, the first sections of the chapter provide an overview of the definitions of unipolar depression and bipolar disorder used in the current study and a summary of research on the classification, epidemiology, course and comorbidities of these disorders is presented. It is argued that unipolar depression (major depressive disorder) and bipolar disorder are highly prevalent mental illnesses, which are debilitating and recurrent in nature. Furthermore, the devastating impact that these illnesses can have on individuals and their families and/or carers is illustrated.

Despite the severity of these illnesses, bipolar disorders have been overlooked in psychological literature with research largely confined to biological models and psychopharmacological models. Interest in the last decade has intensified however and several biopsychosocial models have been developed to account for bipolar disorder. The next section of the chapter therefore, reviews four such models including the; Cognitive Therapy model (Beck, Rush, Shaw & Emery, 1979; Lam, Jones, Haywood & Bright, 1999), Behaviour Activation System model (BAS; Gray, 1976, 1982), Interpersonal Social Rhythm Therapy model (IPSRT; Frank, Schwartz & Kupfer, 2000) and the Interacting Cognitive Subsystems Model (ICS; Barnard, 1985; Barnard & Teasdale, 1991; Teasdale & Barnard, 1993). Although these models provide a good starting point, a recent review concluded that they were either too simplistic in their account of bipolar disorders or that they focussed on one particular aspect of the disorder at the expense of the others (Power, 2005). The Schematic, Propositional, Analogical and Associative Representation Systems (SPAARS; Power & Dalgleish, 1997) attempts to overcome these difficulties and represents a relatively new model which is heavily grounded in theory.

One of the key aims of the current study was to test the prediction made in the SPAARS model that five basic emotions (happiness, sadness, fear, anger and disgust) shape all emotional experience (normal and disordered) and that the coupling of these emotions provide the basis for emotional disorder. Therefore, the following section of the chapter explored the literature previously carried out on the emotions experienced in bipolar disorder.

Finally, research has emphasised the important role that adaptive coping strategies play in the severity and duration of psychopathology (Nolen-Hoeksema, 1991). Dysfunctional coping strategies are implicated in the DSM-IV criteria for almost all of the psychiatric disorders, therefore the second aim of the current study was to investigate and compare the strategies commonly employed by individuals with bipolar disorder compared to unipolar depression and a control group. Subsequently, chapter goes on to review the literature previously conducted in this area. Overall, the aim of this chapter is to highlight the gaps in the literature and illustrate how the current study contributes to this literature.

1.2 What is unipolar depression?

Literature regarding the classification, epidemiology, course and comorbidity of unipolar depression will be examined in this section.

1.2.1 Classification

Two main classification systems are used for the diagnosis of psychiatric disorders; the *Diagnostic and Statistical Manual-Version IV* (DSM-IV; APA, 1994) and the *International Classification of Diseases-10* (ICD-10; WHO, 1992). There is much debate in the literature with regards to these systems. The large over lap and similarities that occur across the depressive disorders, has led many authors to question the separation of these into distinct categories, arguing instead that such disorders may be more accurately represented on a continuum (Akiskal, Bourgeois, Angst, Post, Moller, & Hirschfeld, 2000). Much of this debate centres around the criteria and thresholds for these disorders. On the whole there are many similarities between the ICD-10 and the DSM-IV, however the differences between them are important. For instance, ICD-10 describes a total of 22 mood disorders, while the DSM-IV describes 14. Mood disorders comprise of both depressive disorders and anxiety disorders. While the severe cases are likely to be classified similarly under both systems, the milder cases may meet criteria for classification under one system and not the other. Despite these difficulties, the ICD-10 and DSM-IV are generally conceived as representing the 'gold standard' for the diagnosis and classification of psychiatric disorders and as such they are widely used in clinical practice and research. The current study used the DSM-IV criteria for two reasons. Firstly, the DSM-IV criteria has been found to be more restrictive than ICD-10 for depressive

disorders (Bebbington, 2004). Secondly, the majority of the research reviewed used the DSM-IV criteria and therefore in order to draw consistent and accurate comparisons between the literature, it was considered that the DSM-IV criteria were the most appropriate.

'Unipolar depression' in the current study is defined in terms of the DSM-IV criteria for Major Depressive Disorder (MDD). Unipolar depression and bipolar disorder are both primarily characterised by mood disturbance and are therefore classified as affective or mood disorders. The term 'unipolar depression' is used in the literature to distinguish between depression that occurs in the absence of mania and bipolar disorder. Although many of the categories in the DSM-IV and ICD-10 are contentious, the distinction between unipolar depression and bipolar disorder is important given the different aetiologies and epidemiologies of these two groups[1]. The criteria for depressive episodes are similar in both the ICD-10 and DSM-IV[2]. The primary symptoms comprise of a period of either depressed mood or loss of interest/ pleasure that must be present nearly everyday, most of the day, over a two-week period. Three or four of the following symptoms must also be present in order to meet the criteria including; fatigue, weight change, sleep disturbance, impaired concentration, psychomotor disturbance, feelings of worthlessness/guilt and suicidal ideation or attempt In the DSM-IV, major depressive disorders are further broken down into four categories; major depressive disorder – single episode; major depressive disorder- recurrent; dysthymic disorder (comprising of depressed mood occurring more days than not over a two-year period); and depressive disorder not

[1] These are discussed in more detail in the following sections of this chapter.
[2] See Appendix 1 for DSM-IV criteria for a major depressive episode.

otherwise specified. Having outlined the definition of unipolar depression used in the current study, the following section goes on to explore the epidemiology of this disorder.

1.2.2 Epidemiology

There is much variance in the epidemiological research regarding depression and this is largely attributed to the classification system and assessment tools used. As such, the data can be difficult to interpret. Bebbington's (2004) review highlights this issue outlining that annual prevalence rates of depression around the world vary in the literature from 0.8% to 5.8%, while lifetime prevalence rates vary from 1.5% to 16.4%. Data from two large scale UK studies however are relatively consistent, reporting one-week prevalence rates of 2.3% and 2.6% respectively (Jenkins Bebbington, Brugha, Farrell, Gill, Lewis, *et al.,* 1997; Singleton Bumpstead, O'Brien, Lee & Meltzer, 2001). Based on the literature, Bebbington (2004) estimates a 5% annual prevalence rate of DSM-IV major depressive disorder. Wittchen, Muhlig and Pezawas (2003) present similar findings, reporting annual prevalence rates of 5–8% in the adult population for a depressive episode. Furthermore, the World Health Organisation (WHO; 2005) reports that of the 870 million people living in Europe at any one time, 100 million are suffering from anxiety and depression. This report also rates depression as the third leading cause of disability adjusted life years (DALYs; i.e. the sum of the potential years lost to ill health), accounting for 6.2% of the total DALYs.

The most consistent finding in the epidemiological literature is that females are more likely to suffer from depression than males. Research suggests that prevalence rates in females are double that for men (Weissman, Bland, Canino, Faravelli, Greenwald, Hwu, *et al.,* 1996). There appears to be a lack of clarity with regards to an explanation for these differences. Noel-Hoeksema (1987) for instance, reviewed five sets of hypotheses that sought to account for the gender differences. The first set are termed 'artifact hypotheses' (which emphasise the differences in income and help seeking between males and females); the second set are 'biological hypotheses' (highlighting the role of hormones and genetics); thirdly 'psychoanalytic/psychodynamic hypotheses' (which attribute increased rates of depression in females to personality structure); the fourth set are termed 'sex role hypotheses' (highlighting societal assumptions about gender roles) and finally 'learned helplessness hypotheses' (proposing that females may be more prone to depression due to expectations of inadequacy in comparison to males). In her review, Nolen-Hoeksema (1987) concludes that the evidence with regards to all of these hypotheses is inadequate and instead, argues that the gender differences may be better accounted for in terms of response styles to depressed mood. She argues that while men employ strategies aimed at distracting themselves from depressed thoughts or feelings, women tend to ruminate and talk about depressed symptomatology, subsequently amplifying depressed episodes.

Age also appears to affect the epidemiology of depression. Overall, the research indicates that prevalence rates decline with age (Bebbington, 2004). However some research has found that age has a different effect on depression in males and females.

Jenkins, Bebbington, Brugha, Farrell, Gill, Lewis, *et al.* (1997) for instance, report that while more females than males suffer from depression in participants aged between 16-54 years old (2.7% in females and 1.7% in males), the reverse is true for participants aged between 54 and 64 years old (2.0% in males compared to 1.1% in females). These figures suggest that while the female rates of depression decrease in later life, the rates for males increase in this age group. Jorm (1987) also reports this trend describing similar rates of depression in males and females during childhood, with female rates rising in adulthood and declining in elderly groups. Onset typically occurs in late adolescence however more recent evidence suggests that this is changing, with an increased prevalence in childhood and adolescent onset. Bebbington (2004) proposes that the relationship between age and depression maybe linked to those for gender emphasising biological and life transitions.

One of the most robust predictors of unipolar depression is negative life events (Brown & Harris, 1989). However, other sociodemographic factors affecting epidemiology include marital status, with some studies reporting an increased prevalence of depression in married than never married groups (Bebbington, Hurry, Tennant, Sturt & Wing, 1981). Lack of a support network (Targosz, Bebbington, Lewis, Brugha, Jenkins, Farrell, *et al.,* 2003), socio-economic status (Jahoda, 1982) and genetic vulnerability (McGuffin, Farmer, & Harvey, 1991) also appear to influence rates of depression. Furthermore there is evidence to suggest that depression is less prevalent in rural than urban populations (Ayuso-Mateos, Vazquez-Barquero, Dowrick, Lehtinen, Dalgard, Casey *et al.,* 2001). To summarise, the literature presented so far suggests that unipolar depression is highly prevalent,

particularly among females and it has indicated that this illness has a negative impact on social and occupational functioning. The following section examines the research on the course of this disorder.

1.2.3 Course

The National Institute for Clinical Excellence (NICE, 2004) guidelines describe depression as a time limited disorder, lasting up to six months after which point, individuals usually make a full recovery. However, research suggests that there is a high tendency for recurrence and relapse. Andlin-Sobocki and Wittchen (2005) propose that more than 70% of depressive disorders are likely to recur. Similarly, Kupfer (1991) reports that at least 50% of individuals will experience one more episode after the first. Risk of relapse appears to be particularly high in the first 5 years and where onset occurred before 20 years old (Simpson, Nee & Endicott, 1997). In line with the NICE (2004) guidelines, Akiskal (1986) proposes that treatment after the first episode is likely to be successful however, the outlook deteriorates as the number of episodes increases.

The average length of depressive episodes varies in the literature from 12 weeks (Andlin-Sobocki & Wittchen, 2005) to 16 weeks (Kessler, Berglund, Demler, Jin, Koretz, Merikangas, *et al.,* 2003). Hasin, Goodwin, Stinson and Grant (2005) however, report a median duration of 24 weeks for the longest or only episode. Research suggests that in addition to clear cut depressed episodes, patients also demonstrate subsyndromal symptoms. Kennedy, Abbott and Paykel (2004) for instance, propose that over time depressed patients are asymptomatic 52% of the

time, subsyndromal 20% of the time and in a major depressive episode 13% of the time. Hasin, Goodwin, Stinson and Grant's (2005) face-to-face survey of 43,000 participants reported a 3 yr lag between onset of the illness and 1st treatment. In this study, nearly half reported that they wanted die, one third reported suicidal ideation and 8% had attempted suicide. Furthermore, 9.6% required hospitalisation over the course of the illness. According to the NICE (2004) guidelines for depression, the impact on social and occupational functioning in depression is more common in this population than suicide and is the greatest cause of disability (Sartorious, 2001). Furthermore, the impact on physical health in depression is comparable to that of long term chronic conditions such as hypertension and diabetes (Cassano & Fava, 2002).

Some research proposes that age predicts the course of unipolar depression (MDD). Some studies suggest older age correlates with increased severity. For example, Mustafa, Rush, Sackeim, Wisniewski, McClintock, Craven, *et al.* (2005) conducted a large scale study of 1, 498 participants between the ages of 18-75yrs and found that patients aged between 51-65 yrs and 66-75 yrs old reported an increased number of major episodes, of a longer duration as well as an increased frequency of comorbid general medical conditions. However, similar findings have also been reported for childhood onset. A recent study by Korczak and Goldstein (2009) for example, compared onset in adulthood with onset in childhood and adolescence in a sample of 6778 participants with MDD. This study concluded that childhood onset results in more episodes of a longer duration, increased suicidality, increased need for hospitalisation and increased psychotic comorbidity.

Literature regarding the effect of gender on the course of MDD is more varied. Some research proposes that females are likely to suffer a more chronic course (Nolen-Hoeksema, 1987) and are more likely to experience recurring symptoms than males (Ernst & Anst, 1992). However, Simpson, Nee and Endicott's (1997) longitudinal study over the course of a 15-year period, and found no evidence to support this hypothesis. Similarly, Kessler, McGonagle, Swartz, Blazer and Nelson (1993) failed to find any significant differences in the course of depression between males and females. Psychotic symptoms also affect the course of depression, predicting increased severity and increased likelihood of relapse (Coryell, Leon, Winokur, Endicott, Keller, Akiskal, *et al.,* 1996).

In summary the research presented in this section, suggests unipolar depression tends to be a recurrent disorder. The impact of this condition in these cases is severe and is associated with physical ill health and impaired social and occupational functioning. The following section explores the psychiatric comorbidity associated with unipolar depression (MDD).

1.2.4 Co-morbidity

Major depressive disorder rarely occurs as the primary difficulty (Kessler, Bergund, Demler, Jin, Koretz, Merikangas, Rush, *et al.,* 2003; Brown, Campbell, Lehman, Grisham & Mancill, 2001). The high comorbidity of anxiety disorders with depression has been extensively documented. Kessler, Bergund, Demler, Jin, Koretz, Merikangas, Rush *et al.*'s (2003) large scale survey of 9090 participants, found that

over half of the participants with lifetime MDD (59.2%) and 12-month MDD (57.5%) had a comorbid anxiety disorder. Substance disorders were also common (24% and 8.5% respectively) in both groups. Particularly high associations have been reported for drug misuse and personality disorders (Hasin, Goodwin, Stinson & Grant, 2005). The highest associations were found for cluster C personality disorders (such as dependant and avoidant personality disorders) with the exception of obsessive compulsive personality disorders. This finding has also been replicated by Rossi, Daleluzzo, Arduini, Di Domenico, Pollice and Petruzzi (2001). Personality traits such as neuroticism have also been linked with depression (Fava & Kendler, 2000).

In summary so far, this chapter has examined the literature on the classification, epidemiology, course and comorbidity associated with unipolar depression. It has illustrated that unipolar depression is a common disorder which is frequently recurrent and which can have a detrimental impact on an individual's functioning and quality of life. The following section will examine this literature with respect to bipolar disorders.

1.3 What is bipolar disorder?

The classification, epidemiology, course and comorbidity of bipolar disorder is examined in this section.

1.3.1 Classification

Bipolar disorder is classified as an affective/mood disorder and is characterised by dramatic mood swings, where an individual frequently shifts between episodes of depressed and elated (manic) mood. Ultimately, the key distinguishing factor between unipolar depression and bipolar disorder is the presence of mania. The classification of bipolar disorders is complex. There are four different types of bipolar episodes; manic, hypomanic, mixed and depressed[3]. A manic episode primarily comprises of persistent expansive or irritated mood lasting at least one week. Three or more of the following symptoms must also be present in order to fulfil the criteria; inflated self esteem, decreased need for sleep, pressured speech, psychomotor disturbance, flight of ideas, increased goal-directed behaviour and/or excessive involvement in pleasurable activities. Furthermore, these symptoms must cause a marked disturbance in functioning, possibly requiring hospitalisation. Hypomanic episodes comprise of exactly the same symptoms as those required for a manic episode however, in a less severe form. Elated or irritated mood in hypomania must last at least four days rather than one week and these symptoms must not be so severe that they cause marked disturbance in functioning or require hospitalisation. Bipolar depressed episodes in DSM-IV use the same criteria as those for a major depressed episode[4] described previously in section 1.1.1. The DSM-IV criteria for a mixed episode requires the full criteria to be met for both mania and depression and in addition, those must be present nearly everyday for at least a one-week period.

[3] See Appendices 2-4 for the DSM-IV criteria for manic, hypomanic and mixed episodes.
[4] See Appendix 1 for the full DSM-IV criteria for major depressed episodes.

Bipolar disorders are further divided into four main categories in the DSM-IV. The first of those categories is bipolar I disorder (BDI) which primarily requires the presence of at least one manic or mixed episode and is further divided into six subtypes depending on current mood state. The second category is termed bipolar II disorder (BDII) which requires the presence of one or more depressed episodes with at least one hypomanic episode in the absence of mania or mixed episodes. Thirdly, cyclothymic disorder requires the presence of numerous periods with hypomanic and depressed symptoms (that fail to meet the full criteria for a major depressive episode) for at least two years. The final category is termed bipolar disorder not otherwise specified. Several specifiers can also be applied to these diagnoses for example, the rapid cycling specifier defining by the presence of at least four episodes (either (hypo)manic, depressed or mixed) over the previous 12 month-period.

The classification of bipolar disorders is an extremely contentious issue in the literature. Debates surrounding the nosology of the disorder date back to Kraeplin (1921). One of the main issues concerns the distinction between schizophrenia and bipolar disorder and whether these represent separate or overlapping illnesses. Kraeplin (1921) initially distinguished between dementia praecox (now known as schizophrenia) and manic depression, however later challenged his own view stating that it was impossible to distinguish between these illnesses. There is clearly overlap between these disorders. Research since the early 1930s has illustrated that many patients present with a mixture of affective and schizophrenic symptoms (Kasanin, 1933). The presence of psychotic symptoms in manic episodes for example, is well documented in the literature (Goodwin & Jamison, 1990; Mansell & Pedley, 2008).

Further evidence illustrating an overlap between these disorders comes from psychological literature (Bentall, Claridge & Slade, 1989) and research on genetics (Craddock & Owen, 2005). However the debate about the classification of these disorders and whether they should be represented as separate entities or on a continuum is ongoing in the literature.

Another source of debate surrounds the criteria for bipolar episodes and disorders. Much of this debate revolves firstly, around whether bipolar disorders should be represented along a continuum/spectrum rather than divided into separate categories and secondly, whether or not the criteria surrounding bipolar disorders are too narrow. Akiskal, Bourgeois, Angst, Post, Moller and Hirschfeld (2000) for instance, present evidence that the DSM-IV and ICD-10 criteria are too restrictive, neglecting many subtypes that lie in between unipolar and bipolar disorder. For example, they highlight that major depressive episodes often occur with hypomanic traits that fail to meet the full criteria for a hypomanic episode, however such an episode is currently neglected in the DSM-IV and ICD-10 systems. They also argue for a third type of bipolar disorder (BDIII) in which hypomania arises due to antidepressant use. Furthermore, they propose that the cut-off of four days (for hypomania) is too restrictive and neglects the high prevalence of hypomanic episodes that last 1-3 days. With regards to rapid cycling bipolar disorder these authors argue that this merely represents a *'transient phase'* of bipolar disorder rather than a distinct category, presenting evidence to suggest that most rapid cycling episodes return to a baseline level of cyclical mood in a 2-4 year period. They propose that the use of antidepressants play a crucial role in the onset of this subtype. The division between

BDI and BDII is also contentious, as is the divide between mania and hypomania and the criteria for mixed episodes. Although many authors agree with this argument for a wider spectrum, others oppose this suggesting that such a system would be misleading (Baldessarini, 2000).

The difficulties described above contribute significantly to the misdiagnosis and diagnostic delays in bipolar disorder. Research suggests that as many as 40% of bipolar disordered patients are given a diagnosis of unipolar depression initially (Ghaemi, Boiman & Goodwin, 2000; Ghaemi, Sachs, Chiou, Pandurangi & Goodwin, 1999). Mantere, Suominen, Leppamaki, Valtonen, Arvilommi and Isometsa (1993) report a median of 7.8 years delay from the first episode to diagnosis. In another study, one third (34%) of patients received more than 10 years of treatment before receiving a diagnosis of bipolar disorder (Hirschfield, Lewis & Vornik, 2003; Lish, Dime-Meenan, Whybrow, Price & Hirschfield, 1994). The classification difficulties also have serious implications for research for instance, the strict DSM-IV criteria result in the exclusion of many participants from studies and subsequently there are many gaps regarding our knowledge and understanding of this disorder (Akiskal, Bourgeois, Angst, Post, Moller & Hirschfeld, 2000; Wittchen, Muhlig & Pezawas, 2003).

Having examined the difficulties with the classification of bipolar disorders and provided the definition of these used in the current study, the following section goes on to describe the epidemiological literature associated with bipolar disorders.

1.3.2 Epidemiology

The debate and complexity surrounding the classification of bipolar disorders, contributes to the wide variance reported in the epidemiological literature. Angst (1998) proposes that this literature is likely to present underestimates with regards to the prevalence of bipolar disorders given the exclusion of many subtypes from the research. Most studies report prevalence rates between 1% and 5% in the general population. Wittchen, Muhlig and Pezawas (2003) for example, estimate a lifetime prevalence rate of 1–2% for BDI and 5% for BDII. A large scale study reported a prevalence rate of 6.3% when subthreshold symptoms are included (Placidi, Signoretta, Liguori, Gervasi, Maremanni & Akiskal, 1998). The World Health Organisation reports that at any one time of the 870 million people living in Europe, 4 million are suffering from bipolar disorder (WHO, 2005). It is estimated that approximately 20% of those diagnosed with bipolar disorder experience the rapid cycling subtype (Akiskal, Bourgeois, Angst, Post, Moller & Hirschfeld, 2000) and that this is more commonly related to BDII than BDI (Coryell, Endicott, & Kendler, 1992).

The age of onset also varies in the literature. Early studies report that the typical age of onset is between 28-33 years old (Mantere, Suominen, Leppamaki, Valtonen, Arvilommi & Isometsa, 2004). However more recent research suggests an earlier onset between late adolescence and early adulthood (Angst, 1988; Ramana & Bebbington, 1995). Bipolar disorder tends to be equally prevalent in males and females. Research from family, twin and adoption studies suggest that there is a strong genetic component associated with bipolar disorder (Goodwin & Jamison,

1990). There is also mounting evidence to suggest that environmental factors play a critical role in the onset and course of bipolar disorder. A study conducted by Johnson, Cueller, Ruggero, Winett-Perlman, Goodnick, White, *et al.* (2008) for instance, found that goal attainment life events predict increases in manic symptoms over time. Other studies have found that negative life events predict depressed episodes within bipolar disorder (Johnson, Winett, Meyer, Greenhouse & Miller, 1999). Social support and self-esteem appear to be the strongest predictors of bipolar depression however these are unrelated to mania (Johnson, Meyer, Winnett & Small, 2000). In terms of social class, Goodwin and Jamison (1990) suggest that bipolar disorder is more prominent in middle to upper social classes with the working classes being more likely to gain a diagnosis of schizophrenia. Mantere, Suominen, Leppamaki, Valtonen, Arvilommi and Isometsa's (2004) study of 191 patients, found that individuals with bipolar disorder were twice as often divorced; and despite similar education, were more often unemployed; and four times as often pensioned than the general population of the same age. Increased levels of stress and poor interpersonal relationships also correlate with the recurrence of bipolar disorder (Hammen, Henry & Daley, 2004). Furthermore, reproductive events are associated with a higher risk of mood disturbance in women (Freeman, Wosnitzer Smith, Freeman, McElroy, Kmetz, *et al.,* 2002). In summary, this research indicates the bipolar disorders are highly prevalent. Having considered the epidemiological research, the next section examines the course of these disorders.

1.3.3 Course

Bipolar disorders are recurring mental illnesses. Goodwin and Jamison (1990) report that as many as 80-90% of the bipolar population experience recurring episodes. Research suggests that the rate of recurrence increases over time with most estimates reporting that the risk of relapse and recurrence in the first year is 40-48% rising to 73-81% over a period of 4-7 years (Gitlin, Swendsen, Heller & Hammen, 1995; Keller, Klerman & Hirschfeld, 1986). Bipolar disorder is also associated with a high risk of suicide. Guze and Robins (1970) report the suicide rate in bipolar disorder to be 30 times higher than that of the general population. Other studies estimate a 15-20% risk of suicide (Iometsa, 1993; Simpson & Jamison, 1999). The majority of patients with bipolar disorders require a life long course of drug treatment (primarily lithium) to prevent future episodes, which in turn requires frequent monitoring and blood tests. Some data suggest that over 60% of the bipolar population require hospitalisation during the course of the disorder (Andlin-Sobocki & Wittchen, 2005). However, there is some evidence to suggest that a significant proportion of BDII and BDI patients are never hospitalised (Mantere, Suominen, Leppamaki, Valtonen, Arvilommi & Isometsa, 2004).

The duration and number of episodes varies greatly between individuals. The majority of patients have at least three episodes over a 20 year period (Wittchen, Muhlig & Pezawas, 2003). In a secondary care sample, Macqueen, Young, Robb, Marriott, Cooke and Joffe (2000) report a median of 5 lifetime episodes and 2-5 distinct phases (medians 2–5) and predict that this would be higher in more chronic patients. Research suggests that the mean length of episodes in hospitalised patients

ranges from 2-5 months (Angst & Sellaro, 2000). However, other studies suggest a longer duration for mixed episodes (Cassidy & Carroll, 2001). Angst and Sellaro (2000) found no evidence for decreasing cycle length over time. In between episodes, patients often describe subsyndromal levels of symptoms which are reported to last twice that of full blown episodes. For example, Paykel, Abbott, Morriss, Hayhurst and Scott (2006) report a pattern whereby patients are asymptomatic 50% of the time, subsyndromal 15% of the time and episodic 12% of the time. Similar rates are reported by Judd, Akiskal, Schettler, Endicott, Maser, Solomon *et al*'s (2002) longitudinal study. These findings are similar to the course of unipolar depression described previously in section 1.2.3.

The pattern of depressed, (hypo)manic and mixed episodes also varies greatly between individuals. However, depressed episodes and symptoms tend to dominate the longitudinal course of bipolar disorder (Judd & Akiskal, 2003). Joffe, MacQueen, Marriott and Young (2004) found that over the course of 1 year, patients were euthymic 50% of the time, depressed 41% of the time and manic 6% of the time. Wittchen, Muhlig, and Pezawas's (2003) literature review found that in clinical samples, the risk of mania and hypomania switching to depression is relatively high (17-30% and 29% respectively), however the risk of depression turning into (hypo)manic episodes over ten years is low (10%). Mixed states have been found to occur in over 40% of patients and in fact, Goodwin and Jamison (1990) report that most manic episodes actually represent mixed episodes. Some research suggests that the type of episode at onset affects the course of bipolar disorder. For instance, Perugi, Micheli, Akiskal, Madaro, Socci, Quilici, *et al.*'s (2000) systematic analysis

of a large sample of participants found that patients who are depressed at onset, are more likely to develop rapid cycling bipolar disorder and furthermore, are more likely to be suicidal and present with psychotic symptoms than those with mixed or manic episodes at onset.

Although the prevalence of bipolar disorder is similar in males and females, some research has found that gender affects the course of the illness. For example, Rasgon Bauer, Grof, Gyulai, Elamn, Glenn, & Whybrow (2005) analysed computerised daily diaries of 80 patients (35 males, 45 females) with bipolar disorder. They found that men reported to be depressed 17% of the time, euthymic 74 % of the time and manic 5.6% of the time; whereas women felt depressed 28.3%, euthymic 65.2% and manic 7.5% of the time. They concluded that women reported depression and large mood fluctuations more frequently than males. The finding that women are more often depressed than men has been replicated in many studies (Angst & Sellaro, 2000; Wittchen, Muhlig & Pezawas, 2003). On the otherhand, the studies report equal rates of manic and mixed episodes between the sexes. Ageing does not appear to affect the course of bipolar disorder. Angst and Weiss (1967) for instance, found that over a 20-year period, the pattern of bipolar disorder remains stable. Similarly, Sato, Bottlender, Sievas, Schroter, Hecht and Moller (2003) conducted a longitudinal study over a 20-year period and found that presentations of mania remained stable over the course of bipolar disorder. So far this section has considered the course of bipolar disorders, the following section outlines the co-morbidity associated with bipolar disorders.

1.3.4 Co-morbidity

In both clinical as well as epidemiological surveys, a very high degree of comorbidity and multi-morbidity has been confirmed for BDI however this finding is less consistently reported for BDII. Particularly high associations are evident for substance misuse, anxiety disorders, PTSD and OCD, both within as well as between episodes (Wittchen, Muhlig & Pezawas, 2003). In fact, this study suggests that BDI is almost never a pure disorder.

Otto, Perlman, Wernicke, Reese, Bauer and Pollack's (2004) review concluded that the mean rate of PTSD in bipolar disordered individuals of 16.0%, twice that of the general population. They also found that rates of PTSD differed between inpatient (11-40%), outpatient (7-19%) and community samples (39%). Furthermore, the rates of PTSD tend to be higher in BDI than BDII. Anxiety disorders other than PTSD are also commonly associated with bipolar disorder. Simon, Otto, Wisniewski, Fossey, Sagduyu, Frank, *et al* (2004) report a lifetime prevalence of at least one anxiety disorder in 51% of clinic samples. Elevated rates of panic disorder (10.6% to 62.%), social anxiety disorder (7.8% to 47.2%), generalised anxiety disorder (7% to 32%) and obsessive-compulsive disorder (3.2%-35%) have also been reported in the literature (Otto, Perlman, Wernicke, Reese, Bauer & Pollack, 2004). A longitudinal UK study revealed that anxiety disorder comorbidity was associated with the estimated loss of 39 days well, a lower likelihood of timely recovery from depression, risk of earlier relapse, lower quality of life and diminished role function over a 1-year period relative to those patients without comorbid anxiety (Otto,

Simon, Wisniewski, Miklowitz, Kogan, Reilly-Harrington, *et al.,* 2006). These effects were consistent for both BDI and BDII.

Personality disorders are also commonly associated with bipolar disorder. Avoidant and dependent personality disorders appear to be particularly common however, obsessive and borderline features are also evident in the literature (Wittchen, Muhlig & Pezawas, 2003). George, Miklowitz, Richards, Simoneau and Taylor's (2003) study of 52 bipolar disordered patients found that cluster B (dramatic, emotionally erratic) and cluster C (fearful, avoidant) personality disorders were more common than cluster A (odd, eccentric) personality disorders. This study concluded that less than one in three bipolar disorder patients suffer from a co-morbid Axis II disorder. Some research suggests that increased neuroticism predicts depressive symptomatology over time; while increased conscientiousness, particularly achievement striving, predicts mania (Lozano & Johnson, 2001). Participants with comorbid personality disorders differed in the severity of residual symptoms even in remission.

Other common comorbities with bipolar disorder include substance misuse, ADHD and psychotic disorders (Marneros & Brieger, 2002). It is estimated that between 6% and 10% of manic episodes and bipolar disorder turn into psychotic disorders such as schizoaffective disorder and schizophrenia (Winocur, Coryell, Akiskal, Endicott, Keller & Mueller, 1994).

To summarise, this research indicates that psychiatric comorbidity is high in bipolar disorders particularly with respect to anxiety disorders (such as PTSD), personality disorders and substance misuse. So far this chapter has drawn on the literature to illustrate that unipolar depression and bipolar disorders are highly prevalent severe mental illnesses, which can have a debilitating impact on individuals and their families and/or carers. Given the severity and prevalence of these disorders, the following section of this chapter reviews the biopsychosocial models that seek to provide accounts for these disorders.

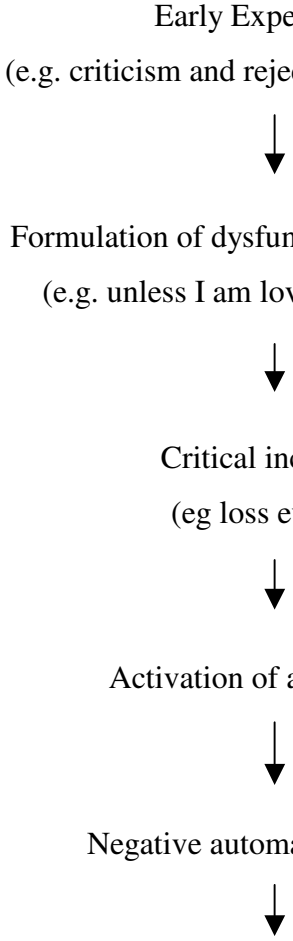
1.4 How do psychological models account for unipolar depression and bipolar disorder?

Five models and their applications to unipolar depression and bipolar disorder, will be presented in turn. The strengths and weaknesses of each model will also be addressed in this section.

1.4.1 Cognitive model of bipolar disorder

Beck, Rush, Shaw and Emery's (1979) cognitive therapy model was originally designed to account for depression. A summary of this model is illustrated in Figure 1.

Figure 1. Beck, Rush, Shaw and Emery's (1979) cognitive therapy model of depression.



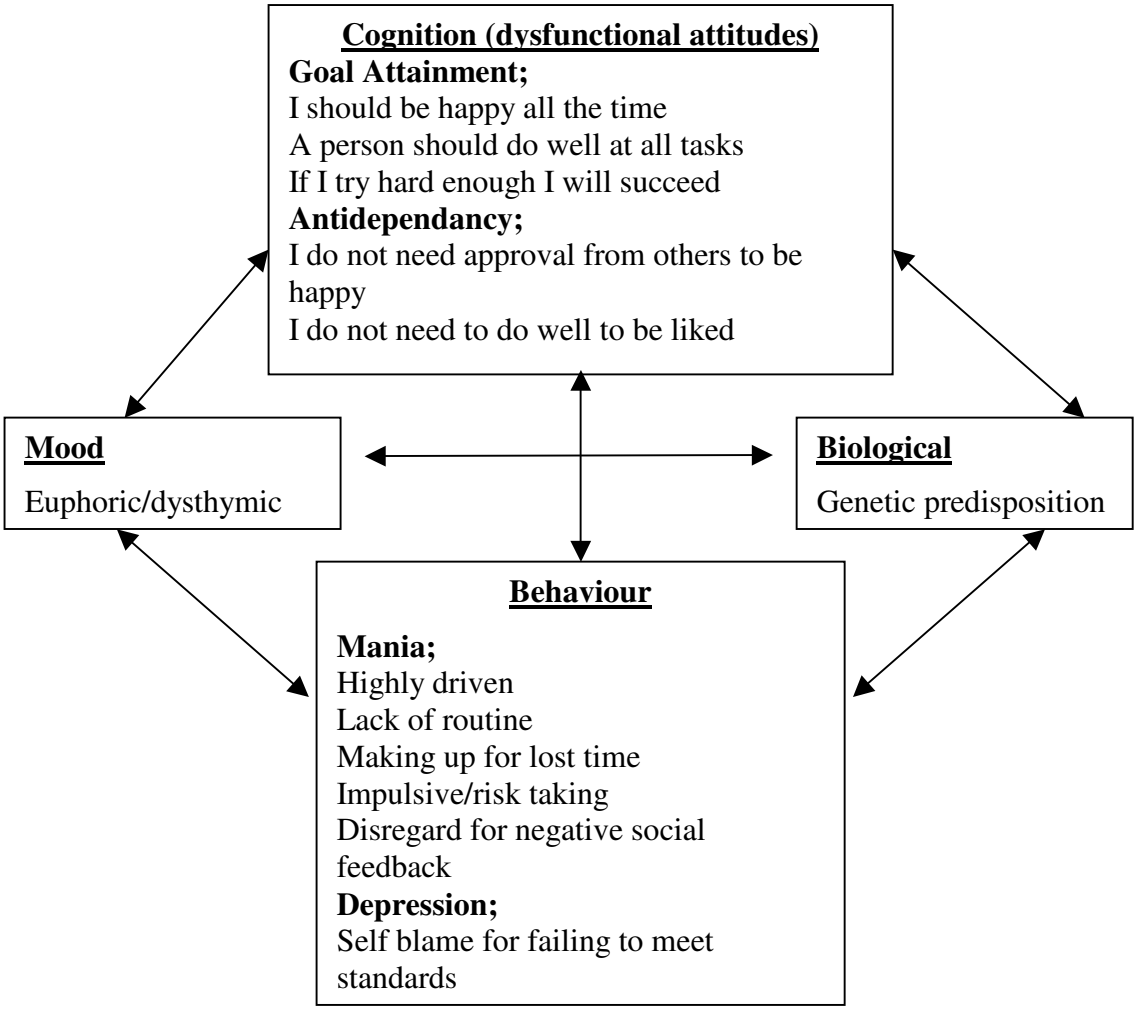
This model proposes that adverse and negative early experiences result in a cognitive vulnerability to depression and the development of dysfunctional beliefs. According to this theory, depression is characterised by a negative cognitive triad in which cognitions relating to the self, the world and the future become distorted and negatively biased. It is proposed that these cognitions typically revolve around themes of failure, loss and inadequacy. These negative cognitions are later activated

by critical experiences such as bereavements, which ultimately lead to depressed mood and depressed behaviours (e.g. withdrawal from activities). The cognitive therapy model therefore postulates that emotion (such as depression) is caused by negative dysfunctional cognitions. This model has been influential in psychological literature and clinical practice. It resulted in the development of Cognitive Behaviour Therapy (CBT) and has since been applied to a range of psychiatric disorders including anxiety disorders, schizophrenia, personality disorders and bipolar disorders.

The original adaptation of this model to bipolar disorders came from Beck (1983) who viewed mania as the polar opposite of depression. Beck (1983) conceived of mania in terms of positively biased cognitions and a positive cognitive triad. Such cognitions were proposed to result in selective attention to positive events and experiences, which in turn reinforced and maintained manic thoughts, mood and behaviour. Beck (1983) also noted that while manic, there is a tendency for individuals to move towards autonomy, however during depression, individuals tended to demonstrate a dependency on others. There are several gaps in this model. Firstly, it fails to account for mixed episodes. Secondly, it fails to describe the similarities or differences in dysfunctional beliefs in bipolar and unipolar depression. Furthermore, it fails to address the role of life events and to distinguish those that may be specific to mania as opposed to depression. Subsequently, a more recent adaptation has been made for bipolar disorders by Lam, Jones, Haywood & Bright (1999). This model is based on the original cognitive therapy model with the key

differences being the specific types of dysfunctional attitudes exhibited in bipolar disorders. Lam, Jones, Haywood and Bright's (1999) model is illustrated in Figure 2.

Figure 2. Lam, Jones, Haywood and Bright's (1999) cognitive model of bipolar disorder.



The above model was tested in a study comparing the dysfunctional attitudes of 143 patients with BDI and 109 patients suffering from unipolar depression (Lam, Wright & Smith, 2004). Initially, the study found no significant differences in the dysfunctional beliefs reported by these two groups. However, when subjects who

were likely to be in a major depressed episode were excluded, and residual symptoms of depression were controlled for, the bipolar group achieved higher scores than the unipolar group on the "goal attainment" and "antidependancy" factors of the Short Version of the Dysfunctional Attitudes Scale (DAS-SV; Power, Katz, McGuffin, Duggan, Lam & Beck, 1994). The goal attainment subscale captures attitudes such as, "If I try hard enough I should be able to excel at anything I attempt" and "I must be happy all of the time". Beliefs relating to goal attainment are postulated to precipitate both mania and depression in this model. For instance, in the case of mania such extreme beliefs are thought to contribute to high risk behaviours, pleasure seeking or over working. In turn, these behaviours lead to the disruption of circadian rhythms (e.g. disturbed sleep patterns) and therefore, increase the likelihood of a manic episode. Successful completion of the goal is considered to maintain and further exacerbate manic symptoms. On the other hand, when the effort (e.g. over working) is perceived to be unsuccessful, beliefs relating to self-blame, themes of failure, inadequacy or loss are thought to develop contributing to depressed mood. The antidependancy subscale captures Beck's (1983) concept of autonomy and includes items such as, "I do not need approval from others to be happy". Such beliefs were found to be a constant feature of bipolar disorder in the study, with euthymic bipolar participants scoring more highly on this subscale than euthymic depressed participants. This was the case in both sets of results (whether or not the patients in a major depressive episode were included). This study proposes that that these beliefs may interact with the illness itself and a biological vulnerability to it, serving to increase the likelihood of a more severe and prolonged course.

In a review Power (2005) argues that the cognitive therapy model is too simplistic in its account of bipolar disorders. For instance, the core premise of Lam, Jones, Haywood and Bright's (1999) model is the idea, from the original model, that dysfunctional beliefs cause emotion. Power (2005) argues that this theory is too simplistic and overly focussed on cognition at the expense of emotion. Furthermore, this review proposes that the model does not account for all of the features of bipolar disorder, such as the extreme shifts in self esteem observed between manic and depressed episodes and questions the processes by which the content of a single dysfunctional cognition could change from positive to negative. The underlying argument in this review is that more complex, emotion based, theoretically driven models are needed to account for bipolar disorders.

1.4.2 The behavioural activation system (BAS) model

The BAS model was first described by Gray (1976, 1982) and represents a neuropsychological model that aims to outline the relationship between cognition, emotion, conscious experience and the brain. Gray's original model proposed that two systems are involved in emotion; the behavioural activation system (BAS) and the behavioural inhibition system (BIS). The model was initially applied to anxiety and emphasised the role of the BIS (Gray, 1982). More recently, this model has been applied to the mood disorders and has emphasised the role of the BAS.

The BAS is concerned with approach behaviour and is activated by the presence of positive stimuli or reward. Activating processes of the BAS include incentive motivation and motor programmes related to approach. As such, high levels of BAS

activity are associated with high levels of arousal, positive emotion and engagement in goal directed behaviour. Low levels of activation on the other hand, are associated with low levels of arousal and disengagement from reward seeking activities. Depue, Krauss and Spoont (1987) and Goplerud and Depue (1985) have suggested that bipolar disorder results form an inability of the BAS to regulate mood and return it to baseline. They propose that high and low levels of activation in the BAS correspond to (hypo)manic and depressive symptomatology. There is evidence to suggest that activation of the BAS not only correlates with manic symptoms, but also predicts them (Meyer, Johnson & Winters, 2001). While, activation of the BAS in the presence of reward is normal, it is argued that in bipolar disorder there is an oversensitivity to reward in mania (Wright & Lam, 2004). In summary, (hypo)mania and depression are conceived in terms of over/underactivity in the BAS which in turn leads to hypersensivity to reward/non-reward and engagement or withdrawal from approach goals respectively. Figures 3 and 4 summarise the BAS model for (hypo)mania and bipolar depression respectively.

Figure 3. BAS model for the development of (hypo)mania. (In Wright & Lam, 2004).

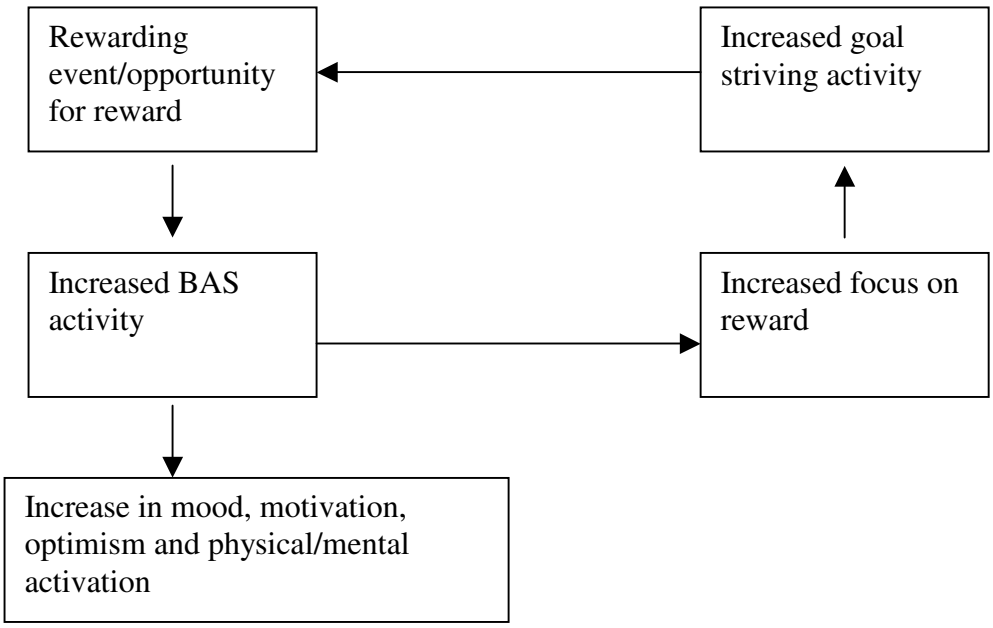
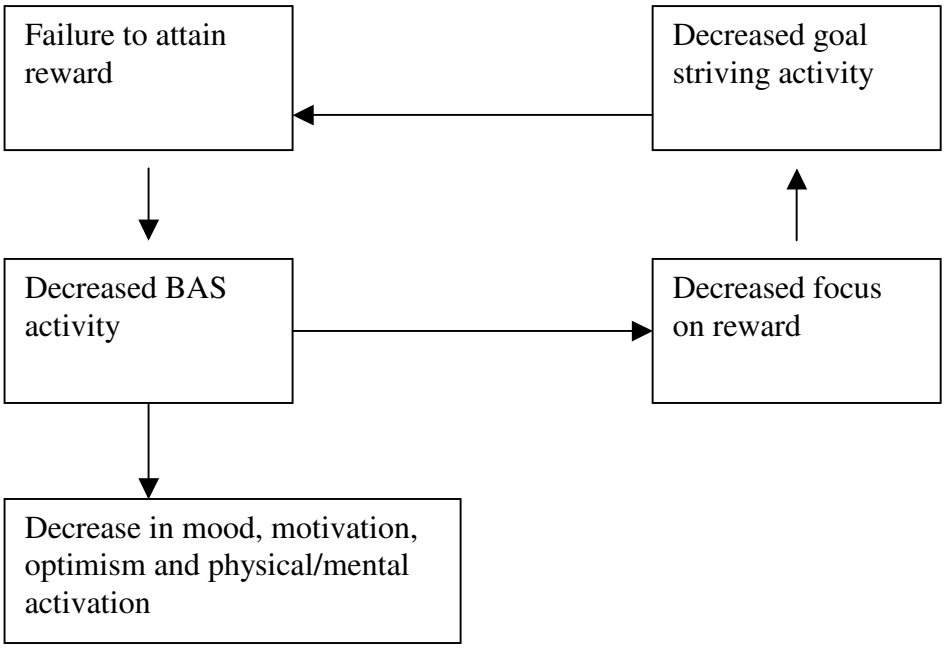


Figure 4. BAS model for the development of bipolar depression. (In Wright & Lam, 2004).



Power's (2005) paper also reviews the BAS account of bipolar disorders as with the cognitive therapy model, this review concludes that Gray's model provides a simplified account of emotion and fails to explain some of the key features in bipolar disorder.

1.4.3 Interpersonal and Social Rhythm Therapy approach (IPSRT)

IPSRT described by Frank, Swartz and Kupfer (2000) is based on the interpersonal therapy (IPT; Klerman, Weissman, Rounsaville & Chevron, 1984) model for unipolar depression. Before detailing IPSRT and its application to bipolar disorder, a brief description of IPT for depression is presented. IPT emphasises the interaction between biological vulnerability, life events, interpersonal relationships and psychosocial functioning in the onset of depression. The aim of this approach is to alleviate the symptoms of depression by facilitating the development of more effective coping strategies (such as increased engagement in interpersonal relationships and the use of a support network). The application of this approach to unipolar depression has gained much credit and subsequently, IPT is recommended by the NICE (2004) guidelines for the treatment of depression.

Like the cognitive therapy model described previously, IPSRT is primarily a treatment model. It draws upon IPT and the instability model proposed by Goodwin and Jamison (Ehlers, Frank & Kupfer, 1988). IRSRT proposes that stressful life events and psychosocial factors (such as interpersonal difficulties) interact with disrupted circadian rhythms resulting in recurring bipolar symptomatology. The goals of treatment are to help patients to regulate circadian rhythms by engagement

in routine and regular patterns of eating, sleeping and exercise and to improve their interpersonal functioning and relationships. One of the weaknesses of the IPSRT model is that due to the fact that its goal is primarily to inform treatment, it lacks in theory (Power, 2005). As with the cognitive therapy model, IPSRT can therefore be applied to several disorders. Furthermore, it does not account for all of the features of bipolar disorder such as changes to the self concept and the processes that are involved in this (Power, 2005).

Although the models presented so far in this chapter have provided a useful starting point for the application of biopsychosocial models to bipolar disorder, they all comprise of a single level of information processing leading some authors to argue that they are too simplistic in their account of the relationship between cognition and emotion (Power, 2005; Teasdale, 1999). Two multi level theories of emotion have therefore been applied to bipolar disorders the Integrating Cognitive Subsystems model (ICS; Teasdale & Barnard, 1993) and the Schematic, Propositional, Analogical and Associative Representation Systems (SPAARS; Power & Dalgleish, 1997). Multi level theories of emotion attempt to provide a detailed account of the relationship between cognition and emotion. They propose that information pertaining to different aspects of experience or events are represented at qualitatively distinct and separate levels within the mind. Furthermore, they propose that these different levels of information vary in their relationship to emotion. Therefore, multi level theories of emotion are more complex than the uni-level models presented previously, in that they consider different levels of cognition/information and their interaction with emotion separately, as opposed to in the uni-level theories which

treat information of the mind as a unitary concept under the term 'cognition'. There is a wide consensus within the literature that multi-level theories offer the most coherent and detailed account of cognition and emotion due to the fact that they can account for complex interactions that uni-level theories are unable to account for (Teasdale, 1999). Teasdale (1999) argues that it is important to adopt these models given that this approach is normative within cognitive psychology. The following two sections of this report describe the ICS and SPAARS models and their application to bipolar disorders.

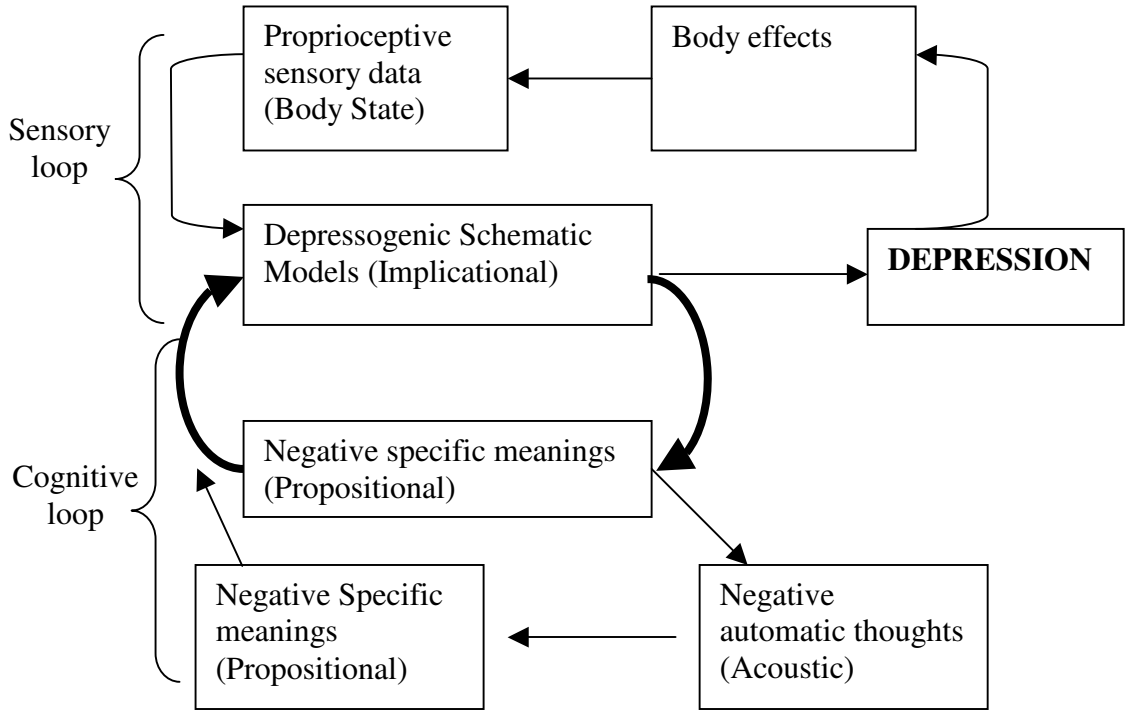
1.4.4 The interacting cognitive subsystems (ICS) model

In ICS (Barnard, 1985; Barnard & Teasdale, 1991; Teasdale & Barnard, 1993) information is arranged across nine cognitive subsystems in the mind, each of which is specialised to process a particular kind of information code. These include; the Sensory related subsystems (comprising of the Acoustic and Visual subsystems); the Central subsystems (comprising of the Morphonolexical, Propositional, Implicational and Object subsystems); and the Affector subsystems (including the Articulator, Body State and Limb subsystems). Information in each subsystem is stored separately in memory and is processed both sequentially and in parallel. The two levels related to the generation of emotion are the Propositional and Implicational. The propositional level comprises of the smallest semantic units. Propositional code is referred to as speech level code (Teasdale, 1999). It represents specific and explicit meanings that can be conveyed in a sentence in language, for instance, 'Gordon Brown is Prime Minister'. The Implicational level however, comprises of higher-level semantic representations referred to as 'schematic models'. Schematic models

are generic, holistic and implicit meanings, representing the largest semantic units. They are not easily conveyed in language because they are implicit. Information from the eight remaining subsystems feeds into the Implicational level and is integrated into schematic models. As a result, schematic models are particularly susceptible to thematic semantic content for instance themes such as, 'globally negative view of self', or 'hopeless, highly aversive uncontrollable situation that will persist indefinitely' (Teasdale, 1999). ICS proposes that emotion is generated directly via the Implicational subsystem when implicational codes are processed. Although Propositional codes influence emotion by feeding into the Implicational subsystem, there is no direct route to emotion at the Propositional level. In summary, the ICS model proposes that there is one route to emotion, directly via the schematic models. Therefore, ICS differs from the cognitive therapy model described previously, proposing that emotion is not the result of a specific appraisal, but instead is the result of a variety of information, drawn from all of the cognitive subsystems and integrated at the Implicational subsystem.

ICS has been applied to unipolar depression (Teasdale & Barnard, 1993; Teasdale, 1996). The key process in depression is the 'interlocking' of the subsystems which plays a major role in the maintenance of depression. Figure 5 illustrates this process.

Figure 5. The ICS 'depressive interlock'. (In Teasdale, 1999).



This figure highlights the Propositional and Implicational levels involved in emotion, the fact that the Implicational level combines information received from other subsystems and that emotion is generated directly via the Implicational level. While processing in the ICS model is usually dynamic, the bold arrows at the centre of the diagram below illustrate that sensory and cognitive feedback loops can develop within the system that 'lock' the subsystems into a configuration that maintains depression. Therefore it is proposed that in depression, information processing is less dynamic and that there is a low rate of exchange in the content of implicational information so that the system becomes locked, continually regenerating negative propositions. This process is linked to ruminative thought where the focus is on specific propositions rather than the inter-relationships between schematic models.

Depression is therefore postulated to occur when the processing of information between subsystems becomes locked and the schematic models become fixed on negative propositions.

ICS has also been applied to bipolar disorders (Lomax, Barnard & Lam, 2009; Palmer & Barnard, 2003). Bipolar depressed states involve the same processes as described above in unipolar depressed states. However, mania in ICS represents the opposite process from those in depression. In mania, it is thought that there is a high rate of exchange in implicational content and positive or mixed schematic models are processed. Implicational content is therefore exchanged so rapidly in mania that decreased attention is paid to the inter-relationships between specific propositions and as such, schematic models are unreflective and unevaluated. Discrepancies between the models are in turn, unevaluated. In summary, ICS proposes therefore that bipolar disorders and schizophrenia occur when the rate of exchange is so fast that schematic and propositional information becomes disintegrated and out of sync with each other.

The ICS model overcomes the limitations of the uni-level models presented previously, in that it describes a complex theory of cognition and emotion. Power (2005) outlines that it is primarily a theory of cognition and is less focussed on emotion. However, given that the application of ICS to bipolar disorders is fairly recent, more research is needed before conclusions can be drawn as to its usefulness with regards to these disorders.

1.4.5 The Schematic, Propositional, Analogical and Associative Representation Systems (SPAARS) Model

The SPAARS model is a biopsychosocial model which draws upon psychological and philosophical theory to explain the link between cognition and emotion. It attempts to account for both normal, everyday emotional experience and for the emotional disorders. Since, this model is at the core of the current study, it will be the focus of the following six sections of the chapter. A summary of the SPAARS theory of emotion and cognition is outlined respectively in the next two sections. The architecture and structure of the model is then presented and the final three sections describe the specific application of this model to the emotional disorders, unipolar depression and bipolar disorder. This discussion of SPAARS will be concise and focussed on the aspects relevant to the current study (for a fuller discussion see Power & Dalgleish, 2008).

1.4.5a Background to emotions in SPAARS

The SPAARS model draws upon philosophical theory to make four key points about emotions. The first is that emotions are functional. The second is that emotions comprise of several key components including; an event, an interpretation of the event, a subsequent appraisal of the interpretation in relation to goals, which then causes a physiological response, and an action potential. The most important of these components in terms of emotion, is the appraisal. Thirdly, each emotion comprises of its own unique appraisal and it is only on the basis of this appraisal that emotions can be meaningfully distinguished from each other. SPAARS argues that appraisals occur in relation to the roles and/or goals that are meaningful to the individual. The

final point SPAARS makes about emotions is that there are five basic emotions, which form the basis for all emotional experience (both normal and disordered). The basic emotion are; sadness, happiness, anger, fear and disgust. Table 1 below illustrates these emotions and their associated appraisals.

Table 1. Five basic emotions and their associated appraisals (Power & Dalgleish, 2008).

Basic emotion	***Appraisal***
Sadness	Loss or failure of a valued role or goal
Happiness	Successful movement towards a valued role or goal
Anger	Blocking or frustration of a valued role or goal
Fear	Physical or social threat to self or a valued role or goal
Disgust	A person, object or idea repulsive to self, and to valued roles and goals

None of the ideas presented above is completely new. SPAARS is grounded heavily in philosophical and psychological theory. The concept of basic emotions was first developed by Descartes (1649, 1989) and later by Darwin (1872, 1965). However, the idea of basic emotions is contentious within the literature. Some authors reject the notion of basic emotions (Ortony & Turner, 1990; Russell, 1994). Even those who concur with the concept of basic emotions disagree about how many there are.

The term ‘basic emotions’ refers to a small set of innate, universal emotions found across all cultures. The evidence for basic emotions focuses on specific components of emotion. For instance, Ekman proposes six basic emotions on the basis of universal facial expressions (Ekman, 1999); James (1884) argues for four basic emotions on the basis of universal physiology; Arnold (1960) argues for eleven basic emotions distinguished on the basis of the associated action potential; other authors have distinguished between basic emotions on the basis of universal antecedent events (Boucher, 1983 as cited in Ekman, 1999). The five basic emotions suggested in SPAARS were triangulated from this evidence and empirically tested using the Basic Emotions Scale (Power, 2006). This study provided support generally for the basic emotions approach and more specifically for the five basic emotions proposed in SPAARS.

Although SPAARS adopts the notion of basic emotions, it emphasises the role of appraisals as the core component of emotion and makes a theoretical proposition that ‘basic’ emotions are those that are associated with universal appraisals. The central arguments made by SPAARS with regards to basic emotions are firstly that these are distinguished in terms of appraisals which are heavily related to an individual’s roles and goals; and secondly that all emotional experience (whether normal or disordered) can be derived from these five emotions. This latter point forms a key hypothesis of the current study, which aims to investigate how manic and depressed states of bipolar disorder can be derived from the basic emotions.

1.4.5b Background to cognition in SPAARS

As noted above, SPAARS proposes that there are five universal appraisals[5]. There is much debate in the literature regarding cognition and emotion and the relationship between them. SPAARS's position is that cognition and emotion are integral and inseparable entities. SPAARS is a theory of mind as much as it is a theory of emotion and based on psychological literature, it makes a number of points regarding the content, organisation and format of mental representations. With regards to content, SPAARS proposes that information pertaining to four aspects are important in relation to emotion including; information about self, information about others, information about the world and information relating to the roles and goals of self and others.

This information is thought to be organised into three domains; the domain of self, other and the world. Information about goals is held within the domains of self and other and is organised hierarchically within SPAARS with higher order goals (such as self preservation) at the top, and smaller more transient goals (such as going to the cinema) at the bottom. Relationships between goals may be facilitatory or inhibitory, and furthermore, goals may be contradictory. In addition some goals may be dependant on the successful completion of others. Domains of knowledge and goals are key concepts in SPAARS because it is argued that individuals interpret and appraise situations on the basis of information held within these three domains. SPAARS proposes that emotional disorder can occur when an individual over invests in one domain (e.g. the domain of other) at the expense of other domains (e.g. the

[5] See Table 1 on page 52.

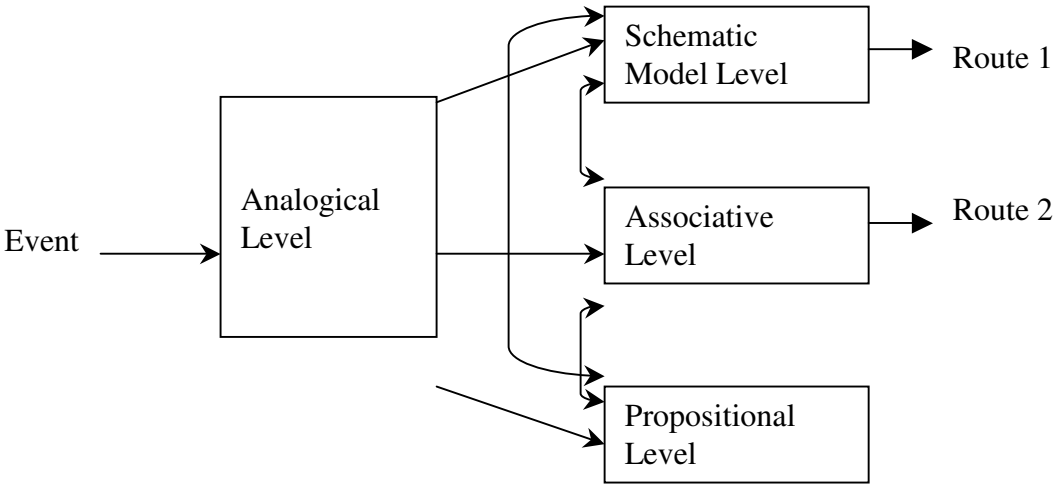
domain of self). With regards to the format of cognitions, SPAARS considers that mental representations are held in four formats or levels including; analogical, associative, propositional and schematic models. These are discussed in more detail in the following section.

In SPAARS, while it is possible to be consciously aware of the physiology of emotions, it is also possible to be unaware of the associated appraisal and interpretation. In addition, it is possible to hold two interpretations of event simultaneously and to be unaware of one but conscious of the other. Furthermore, interpretations can be appraised consciously resulting in one emotion whilst at the same time being appraised in another way resulting in an alternative emotion. When these two interpretations, appraisals and emotions are conflicting, a complex emotional experience and/or emotional disorder may ensue. The notion of unconscious and conscious systems provides an explanation for the conflictual aspects of emotional experience that the uni-level models presented previously fail to explain. For example, in phobias where an individual has an intense phobia of spiders, appraising these as threatening or dangerous, whilst at the same time rationally acknowledging that they are not harmful.

1.4.5c The structure and architecture of SPAARS

SPAARS is a multi level model comprising of four levels of mental representation; Schematic, Propositional, Analogical and Associative. Figure 6 below illustrates the SPAARS architecture.

Figure 6. The SPAARS model (Power & Dalgleish, 1997)



As illustrated above, initial processing of stimuli occurs at the analogical level via sensory specific systems such as, visual, auditory, tactile, proprioceptive and olfactory systems. Analogical representations are connected to a particular sense modality and include images, smells or sounds, for example. Output from this level may then feed into three semantic representation systems which operate in parallel. The lowest level of representation is termed the propositional level. This is similar to the propositional level described in the ICS model. It represents ideational content of the mind and are abstract entities such as ideas, beliefs, objects or concepts. In themselves, they are non-language specific however, their meaning and context can be expressed in spoken language. As in ICS, there is no direct route to emotion via the propositional level and it is proposed that output from this level feeds into either of the other two levels. The associative route represents the intermediate level in SPAARS. At this level information from the analogical and propositional levels can results in the automatic generation of emotion. The schematic model level is the highest level of representation. Again, these are similar to those described in the

implicational level of the ICS model described previously. Schematic representations cannot be fully expressed in natural language. These include schemas or models about self, other and the world.

The key difference between ICS and SPAARS is that SPAARS proposes two routes to emotion, either indirectly via the schematic route or directly via the associative route. At the schematic route, emotion is generated through effortful interpretations and appraisals of goal related events. So for instance, fear is generated at the schematic level where there is an interpretation or appraisal of threat either to self or to a valued role or goal. At the associative route, the schematic model level is bypassed and emotion is generated automatically via association. This involves processes that are similar to those involved when learning a new skill such as riding a bike in that eventually the skill becomes automatic. The idea is therefore that emotion can be generated automatically and without effortful appraisal or conscious awareness if, for instance, an event becomes associated with an emotion through the repeated pairing of event-emotion sequences (Power & Dalgleish, 1999). Phobias present a good example of the associative route when rationally (and at the schematic level) an individual may be aware that the object or event is non-threatening, but at the associative level the object or event is processed as anxiety-provoking.

Emotions generated at either route are referred to as ‘modules’ and act as reconfigurations of the SPAARS system. Facilitatory or inhibitory processes maintain or suppress these reconfigurations. Two or more emotions may be generated simultaneously in SPAARS and it is possible for these emotions to be

contradictory. Feedback loops can occur both within and between modules therefore emotions can become coupled. It is postulated in SPAARS, that such processes can give rise to emotional disorder.

1.4.5d SPAARS and emotional disorder

SPAARS proposes that the emotional disorders can be derived from the same five basic emotions that underlie normal, everyday emotional experience (Power & Dalgleish, 1997) and arise due to coupling or interlocking of one or more basic emotions. Table 2 indicates the basic emotions involved in some of the emotional disorders.

Table 2. Basic emotions and the associated emotional disorders (In Power & Dalgleish, 2008).

Basic emotion	*Coupled emotion*	*Emotional disorder*
FEAR	Disgust	• Panic • Phobias • OCD • GAD • PTSD • PTSD
SADNESS	Anger	• Pathological Grief • Traumatic Grief (PTSD)
	Disgust	• Depression
ANGER		• Pathological Anger • Morbid jealousy • Destructive envy • PTSD
HAPPINESS	Anger/Fear	• Polyannaism/ • pathological optimism • Hypomania • Mania • Love Sickness • De Clerambault's Syndrome
DISGUST Fear		• OCD • Suicide • Eating Disorders • PTSD

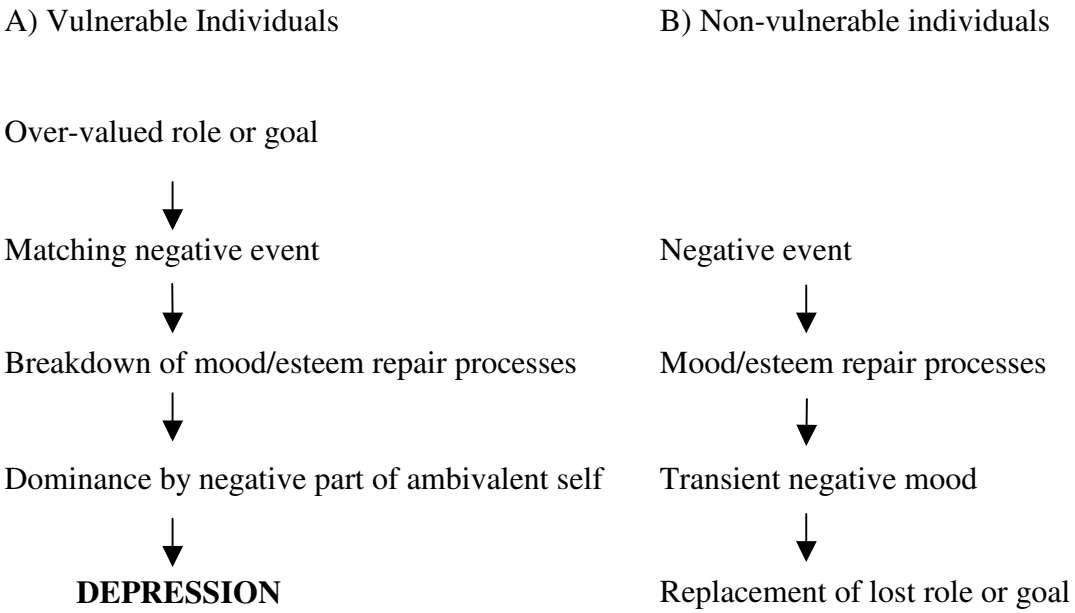
The current study aims to tests the prediction outlined in the table above, that mania is derived from an emotional coupling between happiness and anger/fear, while bipolar depressed states comprise of a coupling between sadness and disgust. Unlike the uni-level models discussed previously in the chapter, SPAARS argues that life events and vulnerability in themselves do not have a direct role in the development

of emotional disorder. Instead SPAARS highlights the importance of the appraisal and interpretation, arguing that it is the way in which an individual makes sense of these concepts that often leads to emotional disorder. Life events are therefore considered to be, *'a function of the individuals' models, goals and appraisals about themselves, the world, and others'* (In Power & Dalgleish, 2008, p134). Inhibitory processes that occur both between conscious and unconscious systems, and between and within different levels within the system, are also considered to play a key role in the development of emotional disorders. So far the last four sections of this report have considered the theory of SPAARS in relation to cognition and emotion, the architecture of the model itself and the SPAARS theory of emotional disorder generally. The next two sections of this thesis will outline briefly the application of SPAARS firstly to unipolar depression, and secondly to bipolar disorder.

1.4.5e SPAARS and unipolar depression

The SPAARS model of unipolar depression draws upon Champion and Power's (1995) model of depression as summarised in the Figure 7.

Figure 7. Champion and Power's (1995) model of depression



Champion and Power (1995) propose that vulnerable individuals tend to over invest in one role/goal at the expense of others. A longitudinal study found that the tendency to over-invest in a particular role or goal delays recovery and increases the likelihood of relapse in recovered individuals (Lam, Green, Power & Checkley, 1994, 1996). When this role or goal is being successfully pursued the individual has a sense of self-worth and is protected by depression. However, when the role or goal is threatened or lost, the matching negative event results in a breakdown of the processes that usually protect the individual. As a result, the individual becomes susceptible to self-negativity and negative self aspects become dominant over the positive. The argument in SPAARS is that this then leads to the generation of sadness and self-disgust. Although in normal individuals sadness at the loss of a valued role or goal will also occur, these individuals are less likely to experience

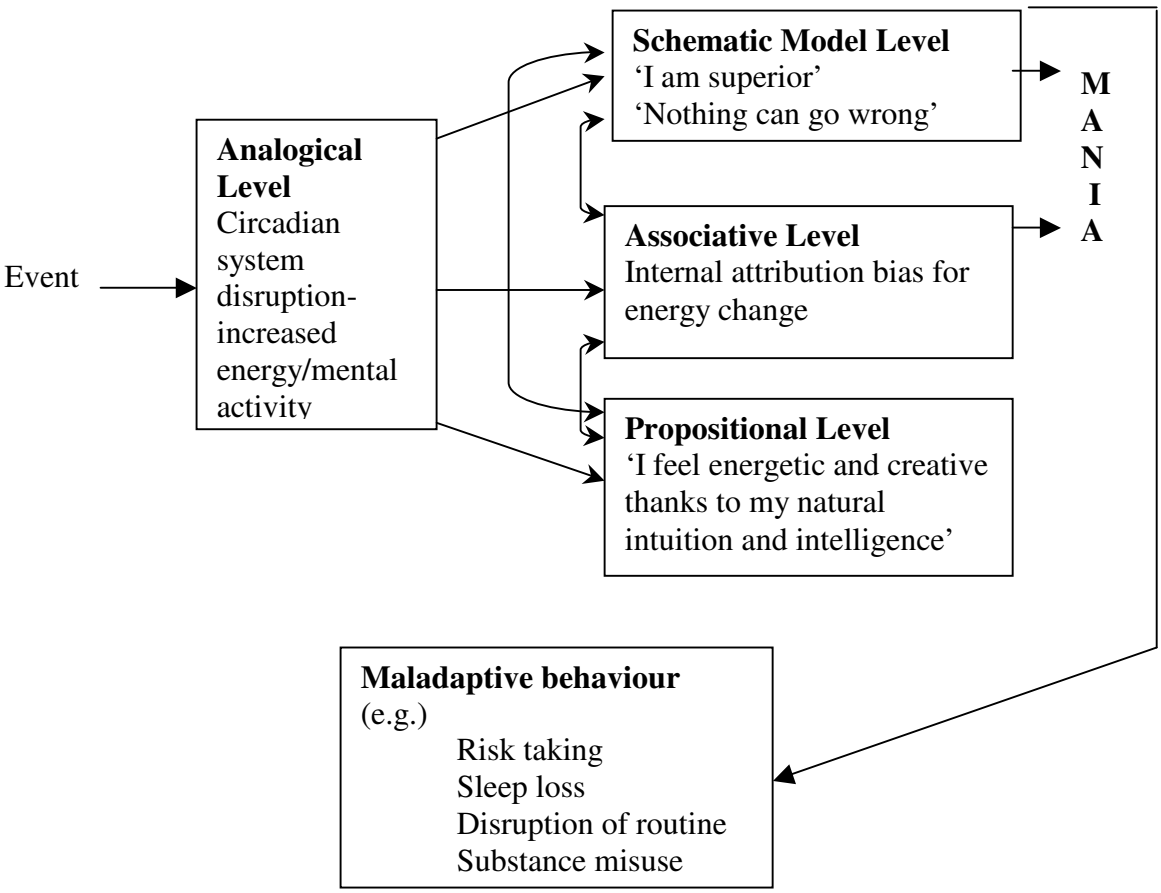
disgust and more likely to replace the lost role or goal. SPAARS proposes that this loss does not have to involve the actual loss of a role or goal but that the loss could be imagined or even result from the successful completion of a goal.

1.4.5f SPAARS and bipolar disorder

Jones (2001) has outlined a specific application of SPAARS to bipolar disorder. This model proposes that mania is initially triggered by life events relating to disrupted circadian rhythms (e.g. working longer hours and/or disruption to sleep pattern). These are processed at the analogical level producing physiological, cognitive and proprioceptive effects such as increased energy, alertness and stamina. Mania is generated at the schematic route when these changes are attributed (at the propositional level) to internal characteristics resulting in positive propositional cognitions such as, "I feel energetic and creative thanks to my natural intuition and intelligence" or "I am full of energy and ready to take on the world" (Jones, 2001). Positive information from other levels is then integrated at the schematic level, resulting in the development of positive schematic models such as, the self as powerful, other as inferior and the world as producing an endless supply of opportunities. These appraisals subsequently result in the generation of positive and elated mood (Jones, 2001). At the associative route, Jones proposes that mania is produced when circadian-emotion links are well established, bypassing the need for input from the schematic level. It is argued that this accounts for the finding that over time, bipolar episodes are triggered by less severe life events (Jones, 2001). In this adapted version of SPAARS, mixed states occur when conflicting emotions are produced at different levels within the system for example, when elated mood is

produced at the associative route while irritability/anger and/or depression is produced at the schematic model level. Jones' model of mania is summarised in Figure 8.

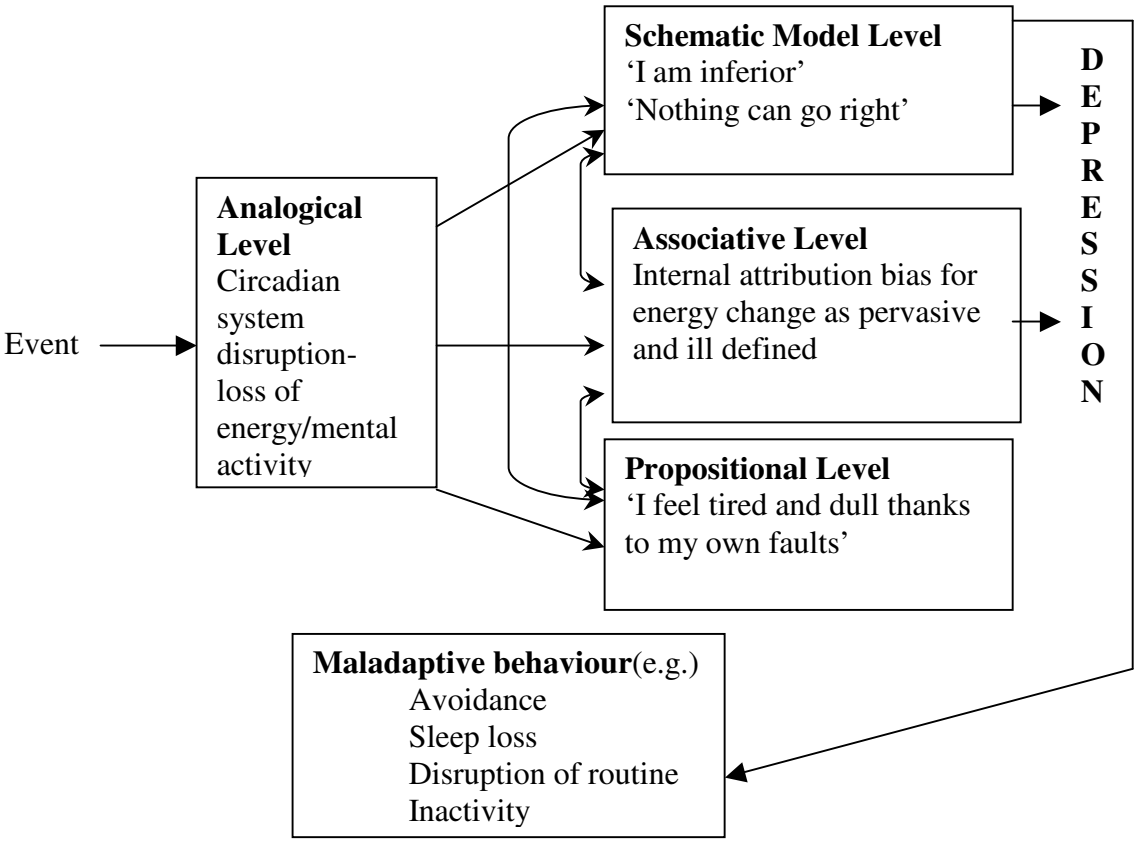
Figure 8. Adapted SPAARS model for mania (Jones, 2001)



With regards to bipolar depression, Jones (2001) proposes that the severity of the disruption to circadian rhythms determines whether or not the mood change will result in depression or mania. It is argued that more substantial environmental changes are associated with increased levels of energy and mania, while lower level

circadian disruption is associated with dysphoria and depression. In depression, it is potulated that the event triggers cognitive, proprioceptive and physiological changes at the analogical level relating to fatigue and lethargy. These changes are then attributed to negative internal characteristics of the individual at the propositional level for example, "I feel tired and dull thanks to my own faults" resulting in a negative bias. At the schematic model, negative information from other levels is integrated resulting in negative schematic models relating to self, other and the world. This negative event-emotion links if repeated over time, may result in negative affect or depression being produced at the associative level. These processes are illustrated below in Figure 9.

Figure 9. Adapted SPAARS model for bipolar depression (Jones, 2001).



Power (2005) makes several points with regards to Jones' (2001) model. The first point relates to the coupling of basic emotions, a core feature of the original SPAARS model (Power, 2005). Power argues that although Jones' (2001) model provides valuable insight, it ignores the role that the coupling of basic emotions plays in emotional disorder. For example, Power and Dalgleish (2008) postulate that depression represents the coupling of sadness with disgust, while mania is primarily a disorder of happiness which most prominently combines with anger. Mixed states are derived from combinations of happiness with sadness and dysphoric mania is derived from combinations of happiness with anxiety. The coupling of one or more basic emotions in mania is evidenced in recent factor analytic studies of mania. These are discussed in more detail in the following section of this thesis. According to Power (2005) these emotional couplings are not addressed in Jones' (2001) model.

The second point relates to the organisation of the self concept in bipolar disorder (Power, de Jong & Lloyd, 2002). Models of bipolar disorder need to account for the occurrence of rapid and frequent shifts in self esteem. For instance, mania is characterised by a positive self concept that revolves around themes of grandeur and invincibility while depression is characterised by a self loathing and themes of failure and worthlessness (Goodwin & Jamison, 1990). According to Power (2007) the self is not a unitary concept, but instead represents multiplicity of self or 'multiple selves'. In other words, the self concept comprises of multiple selves, or self aspects any of which may be active at any one time. These aspects can comprise of emotions, roles or goals, memories, beliefs, attitudes etc. In 'normal' individuals both positive

and negative self aspects are held together so that the self is 'integrated'. Furthermore, the individual invests equally in both positive and negative self aspects so that neither become dominant over the other. Self concepts organised in this manner remain flexible and adaptable in light of new, incongruent information.

Two studies carried out be Power, deJong and Lloyd (2002) found that in bipolar disorder, the self concept organised either entirely organised around extreme positive self aspects or extreme negative aspects. Power (2007) terms this process 'modularisation' and states that it lead to an 'Ambivalent' self. In these states, positive and negative self aspects are held separately so that when a negative aspect is activated, all other negative aspects in that domain will be activated as well and vice versa. In turn, this has significant consequences for self-esteem (Showers, 1992). For instance, low self-esteem may occur when positive self aspects are excluded because they are not valued as important and in turn, negative self aspects become overly dominant. The opposite effect will occur if positive self aspects are overly valued or dominant. In addition and as a result of this, the self concept becomes rigid and inflexible and is unable to adapt to new or incongruent information further contributing to shifts in self esteem. As such, individuals become 'immersed' in emotion and lose the ability to self reflect. It is postulated that the organisation of the self concept in bipolar disorder further exacerbates and maintains mood episodes (Power, deJong & Lloyd 2002). Power, deJong and Lloyd's (2002) second study concluded that compartmentalisation or modularisation of the self concept may, *'represent part of the recurring vulnerability of bipolar disorder'*. This finding has also been supported in two other studies (Reilly-Harrington, Alloy,

Fresco & Whitehouse, 1999; Zaretsky, Segal &Gemar, 1999). SPAARS accounts for these findings and the shifts in self concept found in bipolar disorder in terms of schematic models and emotional couplings. Power proposes that emotional couplings occur rapidly and frequently change so that different schematic models come to dominate and regulate the system at any one time. As such, an individual's mood and sense of self frequently shifts from positive to negative depending on which emotional couplings have occurred and through which routes in SPAARS, and in turn which schematic models are dominant at any one particular time (Power, 2007).

In summary, although several models have been developed for bipolar disorders, the SPAARS model is a multi-level theory of emotion, which is considered to provide the most comprehensive account of the cognitions, emotions and changes in self-concept observed in bipolar disorders. Having outlined the SPAARS theory of emotion and cognition and presented its application to unipolar depression and bipolar disorders, the first aim of the current study is to test the predictions of the SPAARS model in relation to these disorders. However before doing so, the next section of this chapter examines the literature previously done on the experience of emotion in unipolar depression and bipolar disorders.

1.5 What emotions are experienced in unipolar depression and bipolar disorder?

The literature for key studies on the subjective experience of emotion in unipolar depression, bipolar depression and (hypo)mania is examined in this section of the chapter.

1.5.1 Basic emotions and unipolar depression

In terms of the emotions experienced in unipolar depression, the DSM-IV criteria for a major depressive episode indicates that feelings of sadness and/or emptiness, being tearful and inappropriate feelings of guilt characterise depression. However, the SPAARS model postulates that depression derives from a combination of sadness and disgust, rather than guilt. Power and Tarsia (2007) conducted a study to test this theory. Participants were allocated to one of four groups; a depressed, anxious, mixed and control group and assessed using the using the Basic Emotions Scale (BES; Power, 2006). The results for the depressed group found that the three most common basic emotions in this group were sadness, fear and anger closely followed by disgust, and therefore provided empirical support for this hypothesis. The authors concluded that disgust as opposed to guilt was the defining emotion in depression. Although the SPAARS model acknowledges that self-conscious emotions such as, guilt, shame and embarrassment play a role in depression, it considers that these emotions are primarily are derived from disgust. Furthermore, depression is argued to arise in SPAARS when disgust is turned towards the self. The current study seeks to expand on these findings by comparing the emotions experienced in unipolar and bipolar depression.

1.5.2 Basic emotions and bipolar depression

The literature comparing unipolar and bipolar depression has found more similarities than dissimilarities, in fact some authors have proposed that it is impossible to distinguish between them (Cuellar, Johnson & Winters 2005). Furthermore, bipolar

depressed episodes are classified as 'major depressed episodes' in the DSM-IV and use the same criteria as those in unipolar depression (or MDD)[6]. Many of the models of bipolar depression have also used those in unipolar depression as a framework. Although the literature suggests that there are specific dysfunctional assumptions relating to goal attainment and dependency are unique in bipolar disorder (Lam, Jones, Haywood & Bright, 1999)[7], it is argued in the current study that as in unipolar depression, bipolar depressed episodes are likely to be derived from sadness and disgust.

1.5.3 Basic emotions in (hypo)mania

The current study also seeks to explore how (hypo)manic episodes of bipolar disorders are derived from the basic emotions. Despite early suggestions in the literature that mania is polar opposite of depression, the wide variations in presentations of mania have been reported since Kraeplin (1921). Although mania is primarily characterised by elevated mood, the DSM-IV indicates that irritability and anger may also be present. The SPAARS model primarily views mania as a disorder of happiness and predicts that mania will predominantly result from a combination of happiness and anger. However SPAARS also acknowledges the variations in mania and proposes that happiness and anxiety may underlie dysphoric mania. Recent factor analytic studies have demonstrated clusters of symptoms in mania. For instance, Mansell and Pedley (2008) conducted a literature review of seven large scale factor analytic studies which sought to identify the symptoms of mania, common prodromes of mania and the psychological processes associated with

[6] See Appendix 1 for the DSM-IV criteria for a major depressed episode
[7] As shown in the cognitive therapy model in Figure 2

bipolar disorders with a particular emphasis on mania. An overview of these studies and their findings is presented in Table 3.

Table 3. Overview of large factor analytic studies of mania (In Mansell & Pedley, 2008).

FACTORS	STUDIES AND SAMPLES OF EACH						
	Cassidy et al (1998) ***237 manic/mixed***	***Dilsaver et al (1999)*** ***105 manic/mixed***	***Rossi et al (2001)*** ***124 manic***	***Swann et al (2001)*** ***162 manic/mixed***	***Sato et al (2002)*** ***576 manic/mixed***	***Akiskal et al (2003)*** ***104 manic***	***Gonzalez-Pinto et al (2003)*** ***103 manic/mixed***
Depression/ anxiety	1	1	2	2. Anxious pessimism	1	6	1
Psychomotor agitation	2	3. Manic activation	1. Activation-euphoric	1. Impulsivity 3. Hyperactivity	5. Pure manic 6. Emotional lability/ agitation	1. Disinhibition-instability	5
Hedonic tone	4					5. Elation-euphoria 7. Hypersexulaity	3
Irritability/ aggression/ hostility	3	4. Irritability/ paranoia	4	5	2	2. Paranoia-hostility	2
Psychosis	5		_	6	7	4. Grandiosity-psychosis	4
Sleep disturbance	_	2	5	_	3	_	_
Psychomotor retardation	_	_	3	4. Distressed appearance	4	_	_
Deficit	_	_	_	_	_	3	_
TOTAL FACTORS	**5**	**4**	**5**	**6**	**7**	**7**	**5**

As demonstrated in Table 3, these studies suggest between four and seven independent factors of mania. The different methodologies used means that factors are split differently in each of the studies however the results, particularly of Cassidy, Murray, Forest & Carroll's (1998) study, have been largely replicated. In the review, depression and anxiety accounted for the greatest variance of the symptoms in mania. Mansell and Pedley's (2008) review also included studies which investigated whether individuals fall into clusters of symptoms. Based on the findings of these studies they concluded four hypothetical clusters of mania; 'depressive mania' (characterised by symptoms of depression and anxiety), 'pure mania' (characterised by elevated hedonic tone), 'dysphoric mania' (characterised by irritability and aggression) and 'psychotic mania' (characterised by psychotic symptoms). Psychomotor agitation is a common factor in all of these subgroups.

A more recent factor analytic study included ninety-eight who were in a purely manic episode (Picardi, Battisti, de Girolamo, Morosini, Norcio, Bracco, *et al.,* 2008). The findings were largely consistent with previous studies however, they also found an additional factor termed 'disorganisation' that corresponded to symptoms such as disorientation, emotional withdrawal, self-neglect and motor-retardation. The data for this factor were positively skewed, with most patients being free of such symptoms and others showing varying degree of severity. This study also differed from those in Mansell and Pedley's (2008) review in that they failed to find a depression factor. The authors attributed this to the fact that their sample comprised only of manic patients. An interesting study reported by (Sato, Bottlender, Sievas,

Schroter, Hecht & Moller, 2003), found that factors in mania remain relatively stable across presentations over a 20-year period.

With regards to the current study, this research suggests that any one of the basic emotions; sadness, fear, disgust, happiness or anger may be experienced in (hypo)manic episodes of bipolar disorder. The current study seeks therefore aims to investigate this in more detail using the Basic Emotions Scale (BES; Power, 2006). Having considered the literature regarding the emotions experienced in unipolar and bipolar disorder. The following section examines how these emotions are regulated in these disorders before moving on to outline the explicit hypotheses of the current study.

1.6 What coping strategies are used to regulate emotion in unipolar depression and bipolar disorder?

As well as testing the predictions of the SPAARS model with regards to the basic emotions in mania, unipolar and bipolar depression, the current study also aims to investigate the strategies used to regulate these emotions. In order to address this aim the literature was examined to identify key research in this area. The results of this literature review are presented in this section of the chapter.

1.6.1 Definition of emotion regulation

Emotion regulation can be viewed as a subcategory of the more inclusive concept of coping. The term 'coping' comes from the psychoanalytic tradition and refers to changing cognitive and behavioural efforts to manage demands that are perceived as

taxing (Lazarus & Folkman, 1984). These authors identified two key functions of coping; the first is termed 'problem-focussed coping' and refers to efforts that aim to manage or alter the problem; the second is termed 'emotion-focussed coping' and refers to attempts to regulate emotional responses to the problem. In other words coping refers to the ways in which an individual responds to emotion. Functional/adaptive coping strategies are those that enable emotions to be processed, dysfunctional/maladaptive coping strategies are those that prevent the processing of emotion. The link between emotion dysregulation, and the use of maladaptive coping strategies, and impaired functioning is well established within the literature. In fact, references to dysfunctional emotion regulation are made in over half of the DSM-IV Axis I disorders and all of the DSM-IV Axis II disorders (Gross, 1999).

Phillips and Power (2007) developed the Regulation of Emotion Questionnaire (REQ), a self-report questionnaire designed to empirically measure coping strategies (i.e. the ways in which an individual responds to emotion). This measure distinguishes between four types of emotion regulation strategies; 'internal-dysfunctional', 'external-dysfunctional', 'internal-functional' and 'external functional'. Based on the SPAARS theory that emotions are functional, Phillips & Power (2007) argue that 'functional' emotion regulation strategies are those that are based on the information provided by the emotion and that allow the emotion to be processed or 'held', therefore contributing to the development of goal directed behaviour. Subsequently, dysfunctional strategies therefore may include blocking or rejecting the emotion and ultimately lead to an escalation of emotion in the long term. Internal strategies are those that utilize personal or internal resources, while

external strategies are those that draw upon environmental or external resources (Phillips & Power, 2007). Phillips & Power's (2007) study assessed the link between dysfunctional emotional regulation and emotional distress in adolescents. As expected, the results found that dysfunctional coping strategies were associated with increased health difficulties and decreased quality of life. More specifically, internal dysfunctional strategies were linked to internalising problems and emotional symptoms, while external dysfunctional strategies were linked with externalising difficulties such as conduct disorder. Frequent use of dysfunctional strategies was associated with increased severity of difficulty. Given that dysfunctional coping strategies have a negative impact on functioning and the development of psychopathology, the current study sought to investigate the strategies used in unipolar and bipolar disorder. The following two sections outline the relevant research in these areas.

1.6.2 Emotion regulation in unipolar depression

Cognitive behavioural models of unipolar depression have demonstrated that maladaptive coping strategies such as self-blame, catastrophising, rumination, reduced activity and social withdrawal are common in unipolar depression. Rumination is particularly common in depression and has been associated with increased severity and duration of depressed episodes (Nolen-Hoeksema, 1991). Similarly, a study by Thomas and Bentall (2002) also found that rumination in a student sample was strongly associated with depression. Functional strategies such as distraction, problem solving and engaging in pleasurable activities on the other hand, improve depressed mood and are at the core of cognitive behaviour therapy. Based

on the literature presented, the current study hypothesises that unipolar depressed participants will more frequently use maladaptive emotion regulation strategies than participants in the control group.

1.6.3 Emotion regulation in bipolar disorder

Joyce (1985) identified non-compliance and an inability to recognise and respond to early symptoms of bipolar disorder as important factors in the maintenance of depression. The majority of the more recent research conducted in this area however, suggests that patients with bipolar disorder are able to reliably recognise and report prodromes (early symptoms leading up to an episode) (Lam & Wong, 1997). Lam and colleagues have conducted much of the work in this area. In one study, Lam and Wong (1997) outlined that the functioning of individuals with bipolar disorder (in terms of work, marital relationships, parenting abilities, social presentations etc) is strongly related to the strategies they use to deal with prodromes. Subsequently, the study distinguished between adaptive and maladaptive strategies for dealing with manic and depressed prodromes. The results are illustrated in Tables 4 and 5.

Table 4. Ten most frequently endorsed strategies for dealing with mania (In Lam & Wong, 1997).

Ten most frequently endorsed strategies for dealing with manic prodromes	Good coping group n=21 (%)	Poor coping group n=15 (%)
Modifying excessive behaviour	62	0
Engaging in calming activities	48	13
Extra time to rest	43	0
Seeing a doctor	29	7
Medication adherence	19	7
Enjoying the high	5	20
Continue to move about	0	27
Do nothing	0	27
Spend more money	0	20
Find more to do	0	20

Table 5. Seven most frequently endorsed strategies for dealing with depression (In Lam & Wong, 1997).

Seven most frequently endorsed strategies for dealing with depressed prodromes	Good coping group n=17 (%)	Poor coping group n=12 (%)
Get oneself organised and keep busy	53	0
Get social support and meet people	29	0
Distract myself from negative thoughts	24	8
Recognise and evaluate negative thoughts	24	0
Stay in bed and hope it will go away	6	53
Take extra medication without prescription	6	17
Do nothing	0	25

As illustrated in Table 4, maladaptive strategies for dealing with prodromes of mania include; modifying excessive behaviour, engaging in calming activities, taking time to rest and seeing a doctor. On the other hand maladaptive coping strategies for manic prodromes include; enjoying the 'high', continuing to take on more, going out and spending more money and doing nothing. In terms of depressive prodromes, Table 5 illustrates that adaptive strategies include; being organised, seeking social support, distracting oneself from negative thoughts by doing things, and recognising and evaluating negative thoughts. While maladaptive strategies for depressed prodromes include; staying in bed and wishing the problem would go away, taking extra medication without prescription and doing nothing.

In a later study, Lam, Wong & Sham (2001) investigated the coping strategies that were related to reduced relapse and good functional outcomes. With regards to mania, they concluded that behavioural coping strategies such as prioritising and reducing tasks to a realistic amount resulted in a reduced rate of manic relapse over 18 months. However strategies such as engaging in arousing activities, taking on too many tasks were associated with relapse. Similar results were found for depression for instance; 'organising oneself' and 'sorting out worries' were found to be effective in reducing relapse. While drinking alcohol or using other passive strategies when depressed resulted in relapse. In summary, this section has outlined the literature reviewed for emotion regulation and coping strategies in unipolar depression and bipolar disorder. The current study seeks to expand on this literature by investigating the ways in which individuals with bipolar disorder respond to the basic emotions

experienced in manic and depressed states. Having reviewed the literature and provided a rational for the current study based on this literature, the final section of this chapter details the experimental hypotheses for the current study.

1.7 Experimental Hypotheses

The overarching aims of the current study are twofold; firstly, to test the predictions made by the SPAARS model in relation to basic emotions and bipolar disorder. This research is important given the lack of theoretical models, which can adequately account for bipolar disorders (Power, 2005). Secondly, given the important role that effective coping strategies have in reducing relapse in bipolar disorder, this study also seeks to determine how these emotions are regulated in bipolar disorder. More specifically, there are three principle aims. The first is to investigate the basic emotions experienced in the manic phase of bipolar disorder. The second is to investigate and compare the basic emotions experienced in bipolar and unipolar depression. The final aim is to investigate and compare the strategies used to regulate the emotions experienced in unipolar depression and bipolar disorder compared to a control group. The three hypotheses of the study correspond to these aims and are outlined below.

1) The emotional profiles of mania will reveal elevated levels of happiness coupled with anger and/or fear.
2) The emotional profiles of bipolar and unipolar depression will reveal elevated levels of sadness coupled with disgust and/or fear and will not differ significantly from each other.
3) The clinical groups will more frequently use dysfunctional strategies to regulate negative and positive emotion than the control group.

CHAPTER 2 - METHODS

This chapter details the methodology used in the current study to test the hypotheses. The chapter comprises of five main sections describing the research design, participants, measures used, ethical considerations and procedures adopted in the study.

2.1 Design

Data was collected from a semi-structured clinical interview, a clinician rated questionnaire and self report questionnaires. Quantitative methods were used to analyse the data both within and between the groups. There were three participant groups; a bipolar group (comprising of participants with a diagnosis of BDI or BDII), a unipolar group (comprising of participants who were diagnosed with Major Depressive Disorder, MDD) and a control group (comprising of NHS catering and domestic staff). A cross sectional design was used in the current study and hypotheses were tested using a mixture of within and between subjects designs as discussed below.

2.1.1 Design: Hypothesis one

A within subjects design was employed in order to compare the emotional profiles of participants in the bipolar group across general, manic and depressed states.

2.1.2 Design: Hypothesis two

A between subjects design was employed to compare the emotional profiles of depressed states between the bipolar and unipolar groups.

2.1.3 Design: Hypothesis three

A between subjects design was employed in order to compare the coping strategies used to regulate positive and negative emotion between the bipolar, unipolar and control groups.

2.1.4 Design: Additional analyses

Additional analyses included a between subjects design which compared the emotional profiles of general states between the bipolar, unipolar and control groups.

2.2 Participants

This section details the methods used to recruit participants in each group. It then goes on to provide descriptions of each group in terms of demographics (such as age, gender, marital status etc) and current mood state before outlining the exclusion/inclusion criteria adopted in the study.

2.2.1 Recruitment of participants

Participants in the study were recruited from local hospitals and outpatient mental health services in the North East of Scotland.

2.2.1a Recruitment: Clinical groups

Participants in the clinical groups were recruited via a lithium clinic and staff members within two Community Mental Health Teams. These will be discussed in turn. The majority of patients with a diagnosis of bipolar disorder, and a proportion with major depressive disorder, are prescribed lithium. Patients on this medication attend a lithium clinic usually three monthly in order to receive blood tests. These clinics are run on a weekly basis. Participants in the clinical groups were recruited via these clinics. They were identified and informed of the study by the lead clinician responsible for their care. The researcher attended these clinics on a weekly basis and participants who expressed an interest to take part in the study were referred to her and provided with a pack containing a letter of invitation[8], information sheet[9] and consent form[10]. Participants were also recruited outwith the lithium clinics via two Community Mental Health Teams. These multi disciplinary teams comprised of psychiatrists, community psychiatric nurses (CPNs), psychologists, occupational therapists, art therapists, social workers etc. Team members were informed of the study by the researcher and information packs (as described above) were provided to them. Team members were asked to inform patients, who met the exclusion/inclusion criteria, about the study. Those who expressed a wish to participate were provided with information packs by the team member responsible for their care.

After being given information packs, potential participants were invited to provide their contact details and were informed that the researcher would contact them within

[8] See Appendix 5 for the letters of invitation for the three groups
[9] See Appendix 6 for the information sheets for the three groups
[10] See Appendix 7 for the consent form

a one week period in order to determine whether or not they still wished to take part in the study, after having read the information sheet. This provided sufficient time for participants to make an informed decision as to their participation in the study. The voluntary nature of participation was highlighted and participants were invited to contact the researcher in the event that they needed any further information. The above methods of recruitment were considered to be the most appropriate for the clinical groups due to the fact that they were familiar with the location and with the staff members who initially approached them. A total of fifty-eight participants with bipolar disorder were invited to take part (forty-five were recruited from the lithium clinic and thirteen were recruited from the teams). Of the fifty-eight who were invited, thirty-five agreed to take part, fourteen were unable to be contacted and nine did not wish to participate. For the unipolar group, a total of fifteen participants with major depressive disorder were invited to take part, all of whom agreed.

2.2.1b Recruitment: Control group

Participants in the control group were recruited from a local hospital. The researcher contacted the managers of the catering and domestic staff and provided them with information about the study. Four hundred packs were then posted out to staff in these departments via the internal mail service at the hospital. These packs contained a letter of invitation[11], information sheet[12] and consent form[13] which detailed the rationale for the study and contact details for the researcher. The packs also

[11] See Appendix 5 for the letters of invitation for the three groups
[12] See Appendix 6 for the information sheets for the three groups
[13] See Appendix 7 for the consent form

contained a demographics sheet[14], five self report questionnaires (described later in section 2.5) and a stamped, addressed envelope enabling participants to return forms. These participants were given the same information regarding the study as participants in the clinical groups. The main difference in recruitment was the method of delivery. Posting the questionnaires was considered to be the most appropriate and convenient method for this participant group for two main reasons. Firstly, it was not necessary for the researcher to meet these participants due to the fact that this group were not tested with the semi-structured interview[15]. Secondly, this method also ensured the anonymity of these participants was upheld[16]. Of the four hundred people invited to take part in this group, fifteen returned completed questionnaires to the researcher.

2.2.2 Description of participants

This section of the methods chapter, provides details as to the demographics and current mood state of participants in the three groups. These will be discussed in turn.

2.2.2a Description: Bipolar group

A total of thirty-five participants in the bipolar group agreed to take part in the study, however one participant was excluded from the research on the basis that he acquired bipolar disorder following a substantial head injury. Diagnoses were confirmed using

[14] See Appendix 8 for the demographics sheet
[15] See section 2.5.2 for a discussion on the procedure used for testing this group
[16] See section 2.4 for a discussion on ethics and more detail on anonymity

the Structured Clinical Interview for DSM-IV diagnoses (SCID-I).[17] Based on these criteria, of the 34 participants who were included in the study, 23 met the criteria for BDII and 11 met the criteria for BDI. Furthermore, 15 participants reported a first episode of depression, 18 reported a first episode of (hypo)mania and one participant described recurrent manic episodes in the absence of depression.

Of the thirty-four participants in this group, 9 were male and 25 were female. The mean age of this group was 46.03 years (SD=10.87; range 24-64). The mean number of years spent in education for this group was 13.73 (SD=2.91; range 9-20). With regards to employment status; 9 participants were employed, 16 were unemployed and 6 were retired. For marital status; 6 participants reported that they were single, 17 were married, 4 were divorced, 2 were widowed and 5 were cohabiting. The mean number of previous psychiatric admissions for the group was 2.72 (SD=4.02; range 0-22). With regards to current mood state[18], the mean BDI-II score was 16.82 falling into the 'mild depression' category (SD=12.21; range 0-47), the mean STAI-State score was 38.70 (SD=13.78; range 20-70) and the mean STAI-Trait score was 48.79 (SD=13.22; range 28-75). Finally, the mean MAS score was 4.29 (SD=4.09; range 0-18).

2.2.2b Description: Unipolar group

A total of fifteen participants in this group agreed to take part, all of whom were included in the study. Diagnoses were confirmed using the Structured Clinical

[17] See Section 2.3.1 for a discussion about this measure and Appendix 9 for a sample
[18] See Section 2.3 for a full description of the measures used

Interview for DSM-IV diagnoses (SCID-I) and all of these participants met the criteria for recurrent major depressive disorder.

Of the 15 participants in this group, 6 were male and 9 were female. The mean age was 48.60 years (SD=8.45; range 36-60) and the mean number of years spent in education reported was 13.86 (SD=2.32; range 10-17). With regards to employment status; 6 were employed, 7 were unemployed and 2 were retired. The marital status for the group comprised of 2 participants who were single, 5 who were married, 3 were divorced/separated, 1 was widowed and 4 were co-habiting. The mean number of previous psychiatric admissions was 1.46 (SD=2.03; range 0-8). For current mood state, the mean BDI-II total score for the group was 21.14, which fell into the category for 'moderate depression' (SD=15.66; range 0-46). However data for 1 participant was incomplete and this participant was therefore excluded from analyses which used BDI-II scores. The mean STAI-State score was 42.82 (SD=15.81; range 21-76) and the mean STAI-Trait score was 52.33 (SD=14.66; range 27-35).

2.2.2c Description: Control group

The control group comprised of fifteen participants all of whom were included in the analyses. Of these participants, 4 were male and 11 were female. The mean age was 47.53 years (SD=11.17; range 25-62) and the mean number of years spent in education was 10.61 (SD=2.02; range 6-13), although two participants failed to provide this information and were subsequently excluded from these particular analyses. This group comprised of catering and domestic staff in a local hospital and

therefore all of the participants in this group were employed. With regards to marital status; 2 participants were single, 9 were married, 3 were divorced/separated, 1 was widowed and 1 was co-habiting. Only one participant reported a previous psychiatric admission. The mean BDI-II score for this group was 9.16 meeting the criteria for 'minimal depression' (SD=14.78; range 0-54). However missing data was found for 3 participants and they were subsequently excluded from the analyses done on this measure. The mean STAI-State score was 33.86 (SD=13.88; range 20-71) and the mean STAI-Trait score was 37.53 (SD=15.00; range 21-80).

2.2.3 Exclusion/Inclusion criteria

Participants outwith the ages of 18−65 years old were excluded from participating in the study. In terms of the bipolar group participants were required to meet the DSM-IV criteria for either BDI or BDII and for the unipolar group, participants had to meet the DSM-IV criteria for major depressive disorder. Participants with a diagnosis of schizoaffective disorder were excluded from the study. Participants who were unable to provide informed consent, and those who were currently in an acute episode, were also excluded from the study.

2.3 Measures

This section provides a description of the measures used in the study, as well as the validity and reliability of each. These will be discussed in turn.

2.3.1 Structured Clinical Interview for DSM-IV (Research Version, Patient edition with Psychotic screen) (SCID-I/P W/Psy Screen); First, Spitzer, Gibbon, & Williams, 1996)

The SCID[19] is a semi structured clinical interview designed to assist clinicians and researchers in making and confirming psychiatric diagnoses. There are several versions of the SCID, however the one outlined above was designed specifically for research purposes, with participants who are identified as psychiatric patients. It comprises of ten modules that assess each of the Axis I psychiatric disorders in the DSM-IV. However, in accordance with the recommendations made in the manual, the interview was customized in the current study to include only those modules relevant to major depressive and bipolar disorders. Those were the; overview, mood episodes module, mood disorders module and the psychotic screen. The items in the interview are based directly on the DSM-IV criteria and are rated by the clinician on a four point scale where; '?' means inadequate information was provided, '1' means that the symptoms are absent, '2' means that the symptoms are present at subthreshold level and '3' means that the symptoms meet the criteria. In the current study the SCID-I was used to confirm diagnoses and to assign participants to either the unipolar or bipolar group.

Few studies have been done on the reliability and validity of the SCID in comparison to other measures. However, some authors consider the SCID to be the 'gold standard' for making psychiatric diagnoses (Shear, Greeno, Kang, Ludewig, Frank, Swartz *et al.,* 2000; Steiner, Tebes, Sledge, Sledge & Walker, 1995). Excellent

[19] See Appendix 9 for a sample of the SCID

interrater reliability has been illustrated for the SCID in three studies, which report Kappa coefficients of; 0.85 overall for the SCID-I (Ventura, Liberman, Green, Shaner & Mintz, 1998), 0.84 for mood disorder diagnoses (Schneider, Maurer, Sargk, Heiskel, Weber, Frolich, *et al.,* 2004) and 0.80 for the diagnosis of a major depressed episode (Zanarini, Skodol, Bender, Dolan, Sanislow, Schaefer, *et al.,* 2000). Test retest data for the SCID are also good and consistent across studies with three raters (Zanarini, Skodol, Bender, Dolan, Sanislow, Schaefer, *et al.,* 2000) and in a major multi site comparison study (Williams, Gibbon, & First, 1992). Similar findings are reported in cross cultural studies including; Norwegian (Skre, Onstad, Targersen & Kringlen, 1991) and Brazilian populations (Del Ben, Rodrigues & Zuardi, 1996).

The SCID-I has also been found to be a reliable measure for making bipolar diagnoses. One study reports Kappa coefficients of 1.0 for sensitivity, 0.94 for specificity and 0.96 for agreement (Fennig, Craig, Lavelle, Kovasznay & Bromet, 1994). Furthermore, it has been used frequently used in studies of bipolar disorder (Goldberg, Gerstein, Wenze, Welker & Beck, 2008; Lam, Watkins, Hayward, Bright, Wright, Kerr, *et al.,* 2003; Lam, Wright & Smith, 2004). The latter study also reported good-excellent reliability of the SCID for bipolar diagnoses (Kappa coefficient = .84). In summary, research on the SCID suggests that it is a valid and reliable tool for most Axis I disorders, including major depressive disorder and bipolar disorder. Based on this research, it was used in the current study to confirm diagnoses in the clinical groups and assign participants to either the unipolar depressed or the bipolar disordered groups.

2.3.2 Basic Emotions Scale (BES; Power, 2006)

The BES is a self-report measure that was originally designed to assess the basic emotions experienced generally over the last week. It comprises of 20 emotion terms which are rated on a 7−point scale from "not at all" to "all of the time". These items map onto one of five subscales that correspond to each of the basic emotions (i.e. happiness, sadness, anger, fear and disgust) and a total score is calculated for each (Power, 2006). The scale has been found to have good internal reliability and discriminant group validity in a clinical sample of anxious and depressed outpatients (Power & Tarsia, 2007). Three versions of the BES were therefore used in the current study; the original BES that asks about emotions generally, one that asks about emotions during manic episodes and another that asks about the emotions experienced when depressed[20].

2.3.3 Regulation of Emotion Questionnaire (REQ; Phillips & Power, 2007)

The REQ was originally designed to assess the frequency with which adolescents used functional and dysfunctional emotion regulation strategies, which draw upon internal and external resources. Participants are asked how often they use a list of strategies and the items are rated on a 5−point scale from "never" to "always". This measure comprises of 21 items relating to four subscales; "internal functional", "internal dysfunctional", "external functional" and "external dysfunctional." Phillips and Power's (2007) study reported evidence in support of the validity of this measure. Two versions of the REQ were used in the current study; one asking

[20] See Appendix 10 for each version used in the current study

participants about the strategies used to regulate positive emotion and another one asking about the regulation of negative emotion[21].

2.3.4 Beck Depression Inventory – II (BDI–II; Beck, Steer & Brown, 1996)

The BDI-II[22] is a self-report measure designed to assess the presence and severity of depressive symptoms. Based on the DSM-IV criteria it comprises of 21 items relating to the cognitive (e.g. "I feel I am a total failure as a person"), somatic (e.g. "I don't have enough energy to do anything") and behavioral (e.g. "It's hard to get interested in anything") aspects of depression. Participants are asked to rate each item, based on the last two weeks, on a 4-point Likert scale of severity ranging from 0 to 3. Cut off scores were applied according to the BDI-II manual (Beck, Steer & Brown, 1996) whereby total scores between 0-13 indicated 'minimal' depression, 14-19 indicated 'mild' depression, 20-28 indicated 'moderate' depression and 29-63 suggested 'severe' depression.

The BDI-II is one of the most widely used self-report measures of depression in both clinical practice and research. It has been found to be a highly valid and reliable measure of depression regardless of the population used. Beck, Steer and Garbin (1988) for instance, reported good internal consistency for the BDI-II in both psychiatric and nonpsychiatric populations (Cronbach's alpha was 0.86 and 0.81 respectively). Furthermore cross cultural studies have demonstrated the reliability and validity of the measure in German (Kuehner, Buerger, Keller & Hautzinger,

[21] See Appendix 11 for the versions used in the current study.
[22] See Appendix 12

2007), Spanish (Wiebe & Penley, 2005) African-American (Dutton, Jones, Bodenlos, Ancona & Brantley, 2004) and Turkish (Runa, Emine, Bedriye, Mert & Hakan, 2008) populations. Based on this research, the BDI-II was used in the current study to measure the extent to which depressed symptoms were present at the time of testing.

2.3.5 State–Trait Anxiety Inventory (STAI; Speilberger, Gorsuch & Lushene, 1983)

The STAI[23] comprises of 2 subscales; the first of these measures state anxiety and the second measures trait anxiety. Each subscale comprises of 20 items relating to the symptoms of anxiety. In the state subscale, participants are asked to rate each item on a 4-point frequency Likert scale ranging from 1 (i.e. 'not at all') to 4 (i.e. 'very much so') based on how they feel *'right now at this moment'*. The trait subscale asks participants to rate each item on a slightly different 4-point scale ranging, from 1 (i.e. 'almost never') to 4 (i.e. 'almost always'), according to how they feel *'generally'*. Total scores on each subscale range from 20-80. High state scores indicate that the individual is currently in an anxious state. High trait scores indicate that the individual is prone to reacting to situations in such a way that they easily become anxious.

According to Groth-Marnat (2003), the STAI is currently the most frequently used measure of anxiety and is used in over 8,000 studies. Research has consistently demonstrated the reliability and validity of this measure (Metzger, 1976; Rule &

[23] See Appendix 13

Traver, 1983; Smeets & Merckelbach, 1996; Speilberger, Gorsuch & Lushene, 1983). The STAI has also been used extensively in cross-cultural studies, for example Quek, Phil, Low, Razack, Loh and Chua (2004) reported its use in Malaysian samples, further evidencing its reliability and validity. Based on this research, the STAI was used in the current study to assess anxiety levels at the time of testing and to measure the extent to which this differs from the norm for each individual (i.e. anxious personality traits).

2.3.6 The Bech-Rafaelsen Mania Scale (MAS; Bech, Rafaelsen, Kramp & Bolwig, 1978)

The MAS[24] comprises of eleven clinician rated items which map onto the symptoms of mania described in the DSM-IV criteria. Each item is rated on a 5−point scale ranging from 0 (i.e. "not present") to 4 (i.e. "severe or extreme"). Higher scores on this measure indicate higher levels of (hypo)mania with total scores of 0-5 indicating 'no mania', 6-9 indicating hypo or 'mild mania', 10-14 indicating 'probable mania' and scores of 15 or more indicate 'definite mania'. The MAS has been widely used in clinical trials and other published research into mania. A review of the studies using the MAS concluded that it has good internal and external validity as well as high inter-rater reliability (Bech, 2002). Subsequently, the MAS was used in the current study, to estimate the severity of manic symptoms present at the time of testing.

[24] Appendix 14

2.4 Ethical Considerations

Ethical approval for the study was provided by the Tayside Committee on Medical Research Ethics[25]. Approval from the Research and Development Department of the local NHS area was also obtained. In order to protect the anonymity and confidentiality of participants, all of the data collected was stored using an anonymised numerical system. Codes were allocated and correlated with the participant's name on one data sheet that was stored separately from the rest of the data.

2.5 Procedure

This section details the procedure used to test participants in each group. In all cases participants were tested individually.

2.5.1 Clinical Groups

Participants who agreed to take part in the study after a one-week period were invited to meet the researcher at the outpatient clinic at an agreed time. Before starting the study, participants were given the information sheet and consent form. They were instructed again as to the nature of the study and asked if they still wished to participate. They were also given the opportunity to ask questions before being advised to complete the consent form. The relevant modules of the SCID-IV were then administered in order to confirm the clinical diagnoses. Administration of the SCID-IV took between 30-45 minutes to complete. No discrepancies were found with the diagnoses. Participants were then given a series of self report measures. For

[25] See Appendix 22 for a copy of the letter granting ethical approval for the study

each measure in turn, participants were asked to read the instructions before completing the measures. The measures provided to participants differed between the clinical groups and as such these will be discussed in turn.

Participants in the bipolar group were asked to complete three versions of the Basic Emotions Scale including the; BES-General, BES-Depressed and BES-Manic versions. Upon completion of these, they were also given two versions of the Regulation of Emotion Questionnaire; the REQ-Positive and the REQ-Negative. The BDI-II and the STAI-state and trait forms were then given to participants for completion in order to assess current mood state. Finally, the researcher administered the MAS. Participants in the unipolar group were asked to complete two versions of the BES; BES-General and BES-Depressed. The manic version of this questionnaire was omitted in this group, since these participants in this group had never suffered from mania. The positive and negative versions of the REQ were also administered as in the bipolar group. Finally, participants were asked to complete the BDI-II and STAI-State and trait forms. At the end, participants in both groups were thanked for taking part and were given the opportunity to ask questions.

2.5.2 Control Group

This group were not tested using the SCID-IV therefore, self report questionnaires were posted via the internal mail service at the hospital where they worked. The procedure for completing these measures did not differ from those used in the clinical groups. The only difference was the method of delivery. The verbal instructions on the questionnaires asked participants to read the instructions before

completing them. The packs posted to these participants included a covering letter, information sheet, consent form and demographics sheet as described previously. In addition the packs also included the BES-General version, two versions of the REQ (i.e. the REQ-Positive and Negative) as well as the BDI-II and the STAI-State and trait. Participants were asked to return the completed questionnaires using the stamped and self-addressed envelope provided. The contact details for the researcher were provided in the covering letter and information sheet, and participants were encouraged to contact the researcher in the event that they had any questions about the study.

CHAPTER 3 - RESULTS

3.1 Introduction to results

This chapter presents the results of the current study. It outlines and justifies the procedures used to analyse the data, and in addition, it provides the power calculations carried out before and after the analyses. Finally, the key results of the study are summarised.

3.2 Participant demographics

This section describes the participant demographics and explores whether the groups differ with respect to gender, age, education, employment, marital status, number of previous psychiatric admissions and current mood state.

3.2.1 Gender

All three groups were predominantly female (bipolar group; 73.5% female compared to 26.5% male; unipolar group 60% female compared to 40% male; control group 73.3% female compared to 26.7% male in the control group). Furthermore, the groups did not differ significantly with respect to gender ($\chi^2(2)=0.99$; $p=0.60$).

3.2.2 Age

The mean age of the bipolar group was 46.03 years (SD=10.87), for the unipolar group the mean age was 48.60 years (SD=8.45) and for the control group it was

47.53 years (SD=11.17). No significant differences were found between the groups for age (F(2,61)=0.34; p=0.71).

3.2.3 Education

Participants in the bipolar group spent a mean of 13.73 (SD=2.91) years in education. The unipolar group spent a mean of 13.86 (SD=2.32) years and the control group spent a mean of 10.61 (SD=2.02) years in education. However, two participants in the control group failed to provide this information and were excluded from this analysis. A Welch's F test, carried out due to the heterogeneity of variance, revealed that the groups differed significantly with respect to the years spent in education ($F'(2,30.79)=10.67$; $p<0.001$). A post hoc (Dunnett's C) test revealed that the bipolar and unipolar groups spent a significantly higher number of years in education than the control group.

3.2.4 Employment

The majority of participants in the bipolar group were unemployed (55.9% unemployed, 26.5% employed and 17.6% retired). In the unipolar group, 46% were unemployed, 40% were employed and 13.3% were retired. All of the participants in the control group were employed. When all three groups were included, significant differences were found between the groups (χ^2 (4)=22.98; $p<0.001$). However, when the control group were excluded, no significant differences were found between the clinical groups ($\chi^2(2)=0.90$; $p=0.63$).

3.2.5 Marital status

No significant differences were found between the groups for marital status ($\chi^2(2)=5.91$; p=0.82). 50% of the bipolar group were married, 17.6% were single, 11.8% were divorced and 14.7% were co-habiting. In the unipolar group 33.3% married, 26.7% were co-habiting, 13.3% were single or divorced, 6.7% were widowed and 6.7% were separated. In the control group 60% were married, 13.3% were single and 6.7% were divorced, widowed, co-habiting or separated.

3.2.6 Number of previous psychiatric admissions

The mean number of previous psychiatric admissions for the three groups comprised of 2.72 (SD=4.02) for the bipolar group, 1.46 (SD=2.03) for the unipolar group and 0.07 (SD=0.26) for the control group. A Welch's F test revealed that the groups differed significantly ($F'(2,26.78)=10.19$; p=0.001). A post hoc (Dunnett's C) test further indicated that the clinical groups had significantly more previous psychiatric admissions than the control group.

3.2.7 Screening measures

The means and standard deviations for the BDI-II total score, STAI state and trait and MAS scores in the three groups are shown in Table 6.

Table 6. Means and standard deviations of the BDI-II total score, STAI state and trait scores and MAS scores for the bipolar, unipolar and control groups.

Screening measure	Group					
	Bipolar		***Unipolar***		***Control***	
	Mean	***SD***	***Mean***	***SD***	***Mean***	***SD***
BDI-II total score	16.82	12.21	21.14 *[1]	15.66	9.16 *[2]	14.78
STAI_State	38.70	13.78	42.80	15.81	33.86	13.88
STAI_Trait	48.79	13.22	52.33	14.66	37.53	15.00

*[1] missing data for 1 participant in the unipolar group.
*[2] missing data for 3 participants in the control group.

No significant differences were found between the three groups for the BDI-II ($F(2, 57)=2.57$; $p= 0.08$) or STAI state scores ($F(2,61=1.46$; $p=0.23$). However, the groups differed significantly in the STAI trait scores ($F(2, 61)=4.80$; $p=0.01$). A post hoc (Scheffe) test revealed that the bipolar group obtained significantly higher scores on the STAI trait than the control group, as did the unipolar group in comparison to the controls.

In summary, the demographics of each group were relatively similar. However, the groups differed on four variables. Firstly, the clinical groups spent significantly more

years in education than the control group. Secondly, as expected, the clinical groups had significantly more previous psychiatric admissions than the control group. Thirdly, due to the recruitment procedure used, significant differences were found between the groups for employment. However, when the control group was excluded, the no significant differences were found between the clinical groups. Finally, the clinical groups obtained higher scores on the STAI trait indicating that these groups are more prone to interpreting situations in a way that means that they become more easily anxious than the controls. Thus, the chapter will now move on to outline the analysis procedure used in the study.

3.3 Analysis procedure

Data were analysed using the Statistical Package for Social Sciences (SPSS) Version 16 computer program. The two overarching aims of the statistical analyses were firstly, to analyse the differences in the emotional profiles both within the bipolar group and between the unipolar, bipolar and control groups. The second aim was to analyse the differences between the three groups in the coping strategies used to regulate negative and positive emotions. One way Analyses of Variance (ANOVAs) were considered the most appropriate tests to use for analysis for two reasons. Firstly, the aims and hypotheses of the study involved the analysis of differences between three groups or states (with the exception of hypothesis two which involved the comparison of two groups). Secondly, using ANOVAs enabled the researcher to control for current mood state as covariates. In the event of a significant ANOVA, post hoc Sheffe and LSD tests were carried out in order to investigate these differences further. Finally, ANCOVAs were carried out (with BDI-II and STAI state

scores as covariates) following the between subjects ANOVAs in order to investigate the impact of current mood state on the results.

However, prior to carrying out these tests, the data were explored in order to ensure the assumptions of the ANOVA were met. Where the data were found to violate the homogeneity of variance assumption[26], Welch's F tests and Dunnett's C post hoc tests were carried out in place of the ANOVA. Following the exploratory analyses, data were analysed in two main stages; firstly data were analysed with outliers removed and transformed fear data;[27] secondly, these analyses were repeated with outliers included and the original fear data in order to determine whether these procedures made any difference to the main conclusions drawn. The results of these stages will be outlined later in Sections 3.6 and 3.7, however the following section presents the power calculation carried out before data collection.

3.4 Statistical power

Following the decision to analyse the data using ANOVAs, a power calculation was conducted in order to determine the number of participants that were required in each group before meaningful interpretations could be drawn. In line with Cohen's (1988) convention, in a study with three groups when estimating a large effect size, a total of 66 participants (22 participants in each group) were needed to achieve power of 80% when alpha is 0.05. For a within subjects design, a total of 12 participants were needed to achieve a large effect with power of 80% using alpha 0.05[28]. Having

[26] See Section 3.4.4 for more detail
[27] A more detailed discussion of these procedures follows in Sections 3.4.1 and 3.4.3
[28] See Appendices 17-21 for effect sizes and power actually achieved in this study

considered the power calculation, the next section will detail the exploratory analyses.

3.4 Exploring the data

This section details the exploratory analyses carried out on the data. It also outlines the assumptions of the ANOVA and details the procedures used in the current study to ensure that these were met.

3.4.1 Outliers

Outliers are extreme scores in a data set. They can be caused by inaccurate data entry or can simply be a legitimate value that is extreme (Clark-Carter, 1997). Outliers can have a detrimental impact on statistical analyses due to the fact that they affect the mean and the variance, ultimately resulting in inaccurate and unreliable interpretations of results. Considering this, outliers can be legitimately removed from the data set (Clark-Carter, 1997). The raw data for the current study were analysed to identify outliers and these were removed from the data set in the initial stage of analysis.

The data for each dependant variable was analysed in turn. Four outliers were found in the BES general data; three in the happiness subscale (2 in the bipolar group and 1 in the control group) and one on the sadness subcale. In the BES depressed data, a total of 6 outliers were found; 1 in the bipolar group for fear, 2 in the bipolar group for disgust and sadness and 1 in the unipolar group for happiness. In the BES manic version, 2 outliers were found in the bipolar group for happiness and disgust. The

REQ negative data contained 5 outliers in the bipolar group for internal functional strategies, 1 was found for internal dysfunctional strategies in the unipolar group, 3 were found in unipolar group and control groups for external dysfunctional strategies and two were found in the unipolar group for external functional strategies. Finally, regarding the REQ positive data, for external dysfunctional strategies 3 were found in the bipolar group, 1 was found in the unipolar group and 3 were found in the control group. For internal dysfunctional and functional strategies 1 was found in the unipolar group. The tables in Appendix 15 illustrate the means and standard deviations of the variables containing outliers pre and post their removal from the data.

3.4.2 Assumptions of ANOVA

The use of an ANOVA requires three main assumptions about the nature of the data to be fulfilled. The first is that the scores for each condition (i.e. group or state) must be normally distributed. The second states that the variance of the scores in each condition must be the same. For a within subjects design, the variance between an individual's scores on each level of the independent variable must also be the same. This is termed 'sphericity' of data. Thirdly, the ANOVA assumes that the data for each condition must be separate/independent from each other. The last of these assumptions were met as indicated by the hypotheses, however the data were analysed in order to assess whether the first two assumptions were met as described in the following two subsections.

3.4.3 Normality of distributions

Histograms revealed that all of the data were normally distributed except the fear subscale of the BES. Subsequently, the data for this subscale was transformed for the first stage of analysis using an ln transformation. Appendix 16 shows the differences in the means for this data before and after the transformation.

3.4.4 Equality/sphericity of variance

Mauchly's tests were used to investigate the sphericity of data for the within subjects data and Levene's tests were employed to investigate the equality of variance in the between subjects data. A significant result in either of these tests indicates that this assumption has been violated. In the event of a significant result from Mauchly's test, the Greenhouse-Geisser correction was used. This conservative correction adjusts the degrees of freedom and therefore, is likely to avoid a Type 1 error (Clark-Carter, 1997). Where there was a significant Levene's test, a Welch's F′ test was employed rather than the ANOVA. This test is a modified version of the t test designed to address heterogeneity of variance by adjusting the degrees of freedom. The results of these tests are presented in the following section of this chapter, along with the results of the ANOVAs.

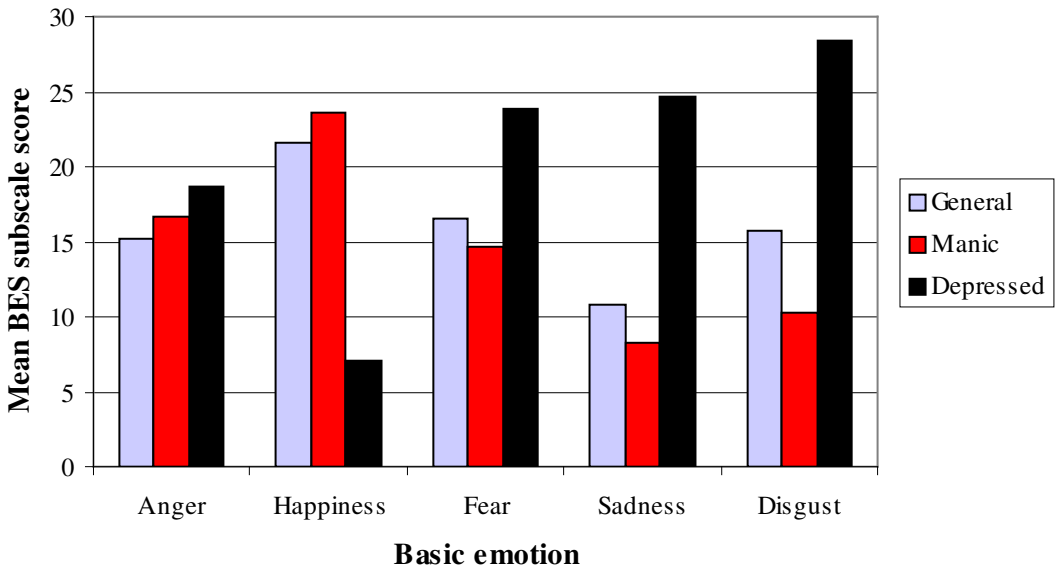
3.6 Results – 1st stage (with outliers removed and transformed fear

This section presents the results of the current study once outliers were removed and the data was transformed. The results for each hypothesis will be discussed in turn. Additional results are also described in this section.

3.6.1 Hypothesis 1: The emotional profiles of mania will reveal elevated levels of happiness coupled with anger and/or fear.

Figure 10 below presents the emotional profiles of general, depressed and manic states within the bipolar group[29].

Figure 10. Mean BES subscale scores for the five basic emotions in general, manic and depressed states within the bipolar group.



[29] The raw data for these results can be found in Appendix 17.

Sphericity of variance was confirmed for all of the data except for transformed fear (i.e. anger p=0.33; happiness p=0.34; transformed fear <0.001; sadness p=0.66; disgust p=0.52). The Greenhouse-Geisser correction was therefore used in this data. Significant differences were found in the emotional profiles of general, manic and depressed states of bipolar disorder (anger ($F(2,64)=3.39$; $p=0.04$); happiness ($F(2,56)=189.06$; $p<0.001$); transformed fear ($F(1.43,44.35)=22.12$; $p<0.001$); sadness ($F(2,58)=146.92$; $p<0.001$); disgust ($F(2,58)=96.71$; $p<0.001$))[30].

The post hoc (LSD) test for anger found that it was significantly elevated in depressed compared to general states (p=0.007). However, no significant differences were found between depressed and manic states (p=0.18) or manic and general states (p=0.26). The results of the post hoc (LSD) tests for fear, revealed elevated levels in depressed compared to general (p<0.001) and manic states (p<0.001). However, there were no significant differences between general and manic states (p=0.12). The post hoc (LSD) tests for happiness revealed three significant results with happiness significantly more elevated in general than depressed states (p<0.001) and in manic compared to general (p0.04) and depressed states (p<0.001). The posthoc LSD tests for sadness and disgust revealed that both of these were significantly elevated in depressed compared to general and manic states, and in general states compared to manic states (all comparisons for sadness and disgust p<0.01).

In summary, the emotional profiles significantly differed between general, manic and depressed states. Manic states comprised of elevated levels of happiness compared to

[30] See Appendix 17 for effect sizes and power.

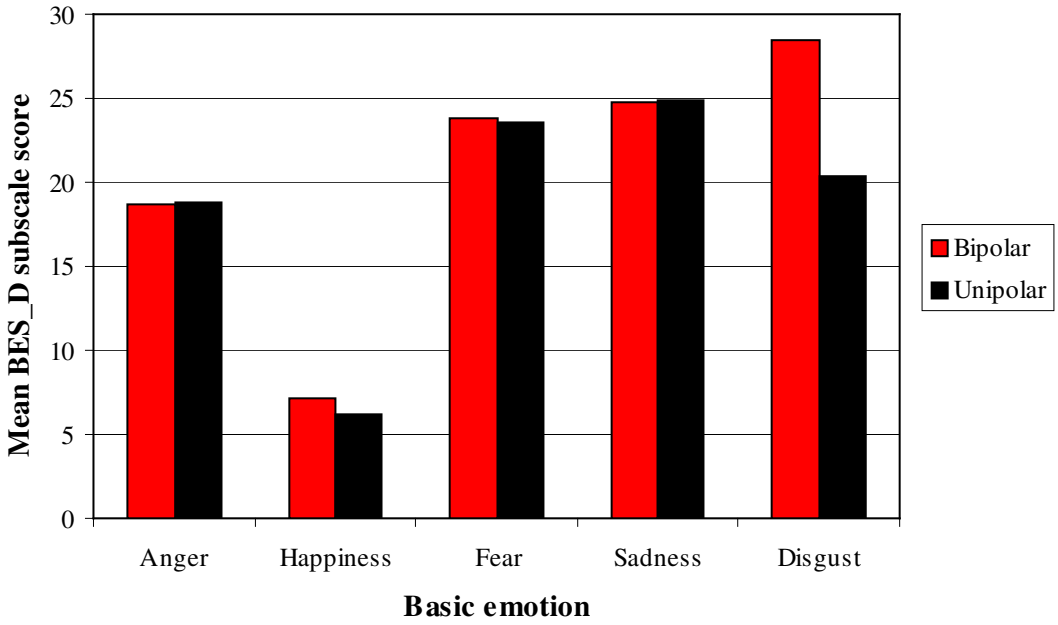
depressed and general states. Anger and fear were also elevated in manic states, however, these emotions were most elevated in depressed states. Furthermore, levels of anger and fear did not differ between general and manic states. The emotional profiles for depression revealed elevated levels of disgust, sadness, fear and anger. Compared to general and manic states, disgust, sadness and fear were significantly elevated in depressed states. Although anger was elevated in depressed states, there were no differences in anger between depressed and general states. Finally, the emotional profiles of general states in bipolar disorder revealed elevated levels of happiness, fear, disgust and anger. In comparison to depressed and manic states, intermediate levels of happiness, fear, sadness and disgust were reported in general states compared to manic and depressed states. Anger on the otherhand, was least frequently reported in general compared to depressed and manic states.

3.6.2 Hypothesis 2: The emotional profiles of bipolar and unipolar depression will reveal elevated levels of sadness coupled with disgust and/or fear and will not differ significantly from each other.

Figure 11 presents the emotional profiles for bipolar and unipolar depression.[31]

[31] The raw data are shown in Appendix18

Figure 11. Mean BES subscale scores for the five basic emotions in depressed states between the bipolar and unipolar groups.



A Levene's test revealed homogeneity for all data except for disgust (i.e. anger p=0.07; happiness p=0.06; transformed fear p=0.11; sadness p=0.56; disgust p=0.004). Furthermore, with the exception of disgust, no significant differences were found between the bipolar and unipolar groups in depressed states (anger (F(1,46)=0.00; p=0.93); happiness (F(1,45)=1.26; p=0.26); sadness (F(1,44)=0.02; p=0.87); transformed fear (F(1,45)=0.13; p=0.71))[32]. The Welch's F′ for disgust found that it was significantly more elevated in bipolar than unipolar depressed states (F′(1,19.22)=9.81; p=0.005). The same conclusions were drawn from the ANCOVAs (anger (F(1,43)=0.001; p=0.97); happiness (F(1,42)=1.48; p=0.23); fear (F(3,1)=0.02; p=0.88); sadness (F(3,1)=0.01; p=0.91); disgust (F(3,1)=12.22;

[32] See Appendix 18 for effect sizes and power calculations.

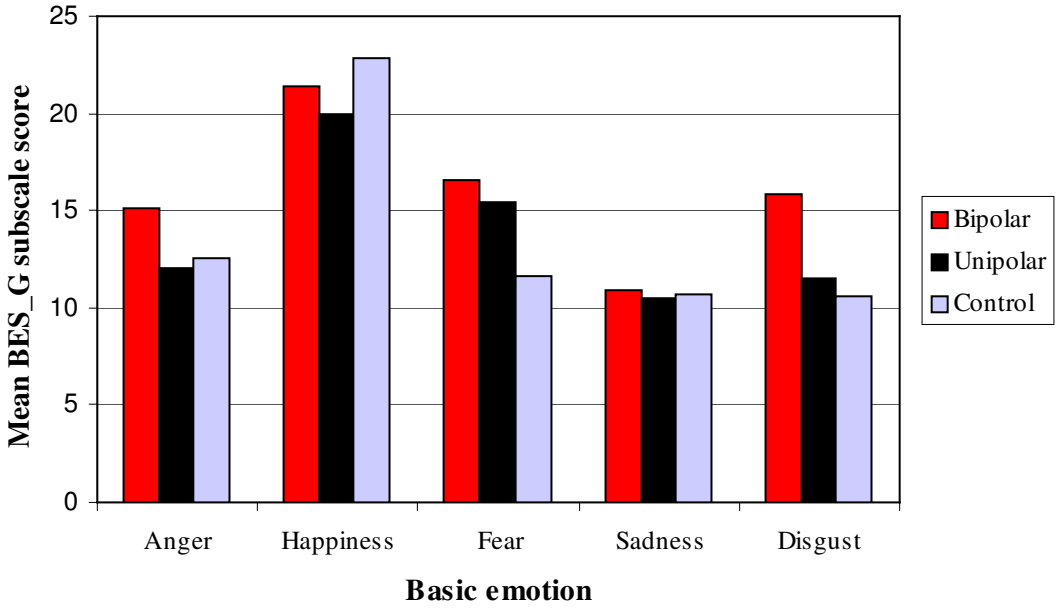
p=0.001). In summary, no significant differences were found between bipolar and unipolar depressed states with the exception of disgust, which was found to be more elevated in bipolar depressed states. These results remained even when current mood state was controlled for.

3.6.3 Additional analysis: Are there any differences in the emotional profiles of general states between the three groups?

Figure 12 below presents the emotional profiles of general states between the bipolar, unipolar and control groups[33].

[33] The raw data for these results are illustrated in Appendix 19

Figure 12. Mean BES subscale scores for the five basic emotions in general states between the bipolar, unipolar and control groups.



Homogenetity of variance was confirmed for all of the data (i.e. anger p=0.23; happiness p=0.05; transformed fear p=0.15; sadness p=0.23; disgust p=0.32). No significant differences were found between the groups for happiness (F(2,58)=2.54; p=0.08) or sadness (F(2,60)=0.04; p=0.96)[34]. However, significant differences were found between the three groups for anger (F(2,61)=3.81; p=0.02), fear (F(2,61)=6.21; p=0.003) and disgust (F(2,61)=6.15;p=0.004).

Post hoc (Scheffe) tests failed to find any significant differences between the groups for anger, however the differences between the bipolar and unipolar groups just missed significance (p=0.06). Post hoc (LSD) tests on the other hand revealed that

[34] See Appendix 19 for effect sizes and power calculations.

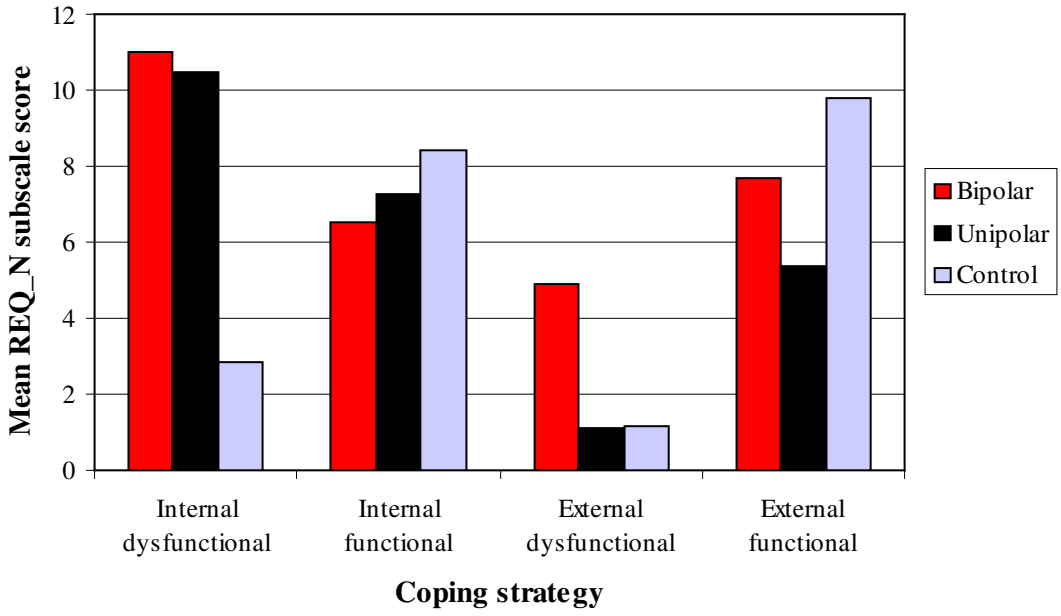
the bipolar group were more frequently angry than the unipolar (p=0.01) and control groups (p=0.05) in general states. No significant differences were found for anger between the unipolar and control groups (p=0.72). With regards to fear, post hoc (Sheffe) tests concluded that the bipolar group were significantly more fearful than the controls (p=0.004) in general states. LSD tests further revealed that the unipolar group were also significantly more fearful than the control group (p=0.02) in general states. Finally, post hoc (Sheffe and LSD) tests found that disgust was more frequently experienced in the bipolar group compared to the unipolar (Sheffe p=0.04; LSD p=0.01) and control groups (Sheffe p=0.01; LSD p=0.003). No significant differences were found in either of the post hoc tests for disgust in the unipolar compared to the control group (Sheffe p=0.89; LSD p=0.64). The ANCOVAs confirmed that these results remained even when current mood state was accounted for (anger ($F(4,2)=3.40$; p=0.04; happiness ($F(4,2)=1.00$; p=0.32; fear ($F(4,2)=4.36$; p=0.01; disgust ($F(4,2)=5.12$; p=0.009 and sadness ($F4,2)=0.27$; p=0.75).

In summary, all three groups reported elevated levels of happiness and low levels of sadness in general states. However, the levels of anger, fear and disgust experienced generally differed significantly between the groups, in that the bipolar group experienced significantly elevated levels of disgust and anger generally than the other two groups. Fear was also more elevated in both clinical groups than the control group. Overall, general states in the unipolar and control group were predominantly characterised by happiness, fear and anger. For the bipolar group these states were predominantly characterised by happiness, fear, disgust and anger. These results remained constant even when current mood state was controlled for.

3.6.4 Hypothesis 3: The clinical groups will more frequently use dysfunctional strategies to regulate negative emotion than the control group.

Figure 13 presents the coping strategies used by the bipolar, unipolar and control groups for dealing with negative emotion[35].

Figure 13. Means for the use of internal dysfunctional, internal functional, external dysfunctional and external functional coping strategies by the bipolar, unipolar and control groups for dealing with negative emotion.



Levene's tests revealed heterogeneity of variance for the data on external dysfunctional ($p<0.001$), internal dysfunctional ($p=0.005$) and internal functional

[35] The raw data for these results are illustrated in Appendix 20.

(p<0.001) coping strategies. However, homogeneity was confirmed for external functional strategies (p=0.05). Welch's F′ tests found that the groups differed significantly in their use of dysfunctional coping strategies in general when regulating negative emotion (external dysfunctional (F′(2, 34.40)=10.34; p<0.001); internal dysfunctional (F′(2,30.43)=55.14; p<0.001)[36]. The ANOVA also revealed significant differences between the groups (F(2,59)=3.79; p=0.02) for external functional strategies. However, no significant differences were found between the groups for internal functional strategies for negative emotion (F′(2,1.81)=23.11; p=0.18).

Post hoc tests (Dunnett's C) revealed that the bipolar group more frequently used external dysfunctional strategies for dealing with negative emotion than the unipolar group (p=0.05) and the control group (p=0.05). Furthermore, these tests indicated that the clinical groups more frequently used internal dysfunctional strategies for dealing with negative emotion than the control group (for both groups p=0.05). Post hoc (Sheffe and LSD) tests confirmed that the control group more frequently used external functional strategies for regulating negative emotion than the unipolar group (Sheffe p=0.02; LSD p=0.008). ANCOVAs confirmed these results for dysfunctional coping strategies (external dysfunctional (F(4,2)=7.53; p=0.001); internal dysfunctional (F4,2)=19.63; p<0.001)) and internal functional (F(4,2)=1.21; p=0.30)). However, the ANCOVA for external functional strategies just missed significance when current mood state was controlled for (F(4,2)=2.86; p=0.06);

[36] See Appendix 20 for effect sizes and power calculations.

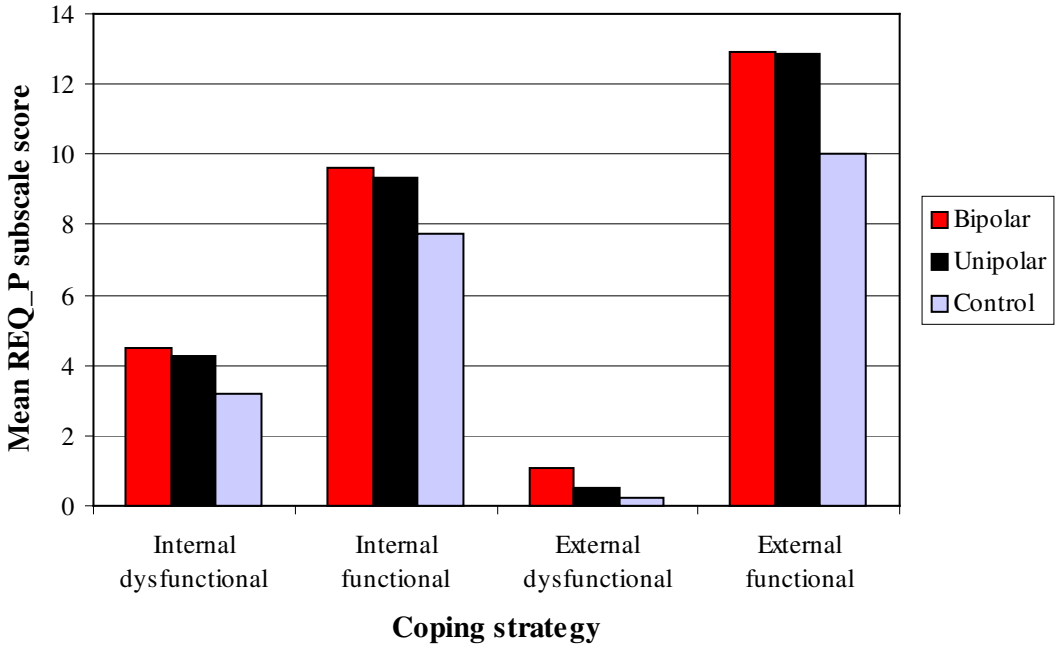
In summary, there are three important findings in these results. Firstly, the control group more frequently used external functional strategies for managing negative emotion than the unipolar group. Although no significant differences were found between the control and bipolar group for these strategies, a trend in the results indicated that they were more frequently used in the control group. With regards to internal functional strategies, a trend in the results suggested that the control groups more frequently used external functional strategies than the clinical groups. However, no significant differences were found. The second important finding is that the clinical groups more frequently used internal dysfunctional strategies than the control group. Thirdly, that the bipolar group more frequently used external dysfunctional strategies than the other groups for regulating negative emotion. With the exception of external functional strategies, these results remained when current mood state was controlled for.

3.6.5 Hypothesis 3: The clinical groups will more frequently use dysfunctional strategies to regulate positive emotion than the control group.

Figure 14 presents the means the coping strategies used by the bipolar, unipolar and control groups for dealing with positive emotion[37].

[37] The raw data for these results can be found in Appendix 21

Figure 14. Means frequency for the use of internal dysfunctional, internal functional, external dysfunctional and external functional coping strategies by the bipolar, unipolar and control groups for dealing with <u>positive emotion.</u>



The results from the Levene's tests found heterogeneity of variance for all data (external dysfunctional (p=0.07), external functional (p=0.52) strategies, internal dysfunctional (p=0.74) and internal functional (p=0.06)). With the exception of external dysfunctional strategies (F(2,55)=3.68; p=0.03), no significant differences were found between the groups for the coping strategies used to manage positive emotion (internal functional (F(2,60)=1.51; p=0.22, internal dysfunctional (F(2,60)=1.70; p=0.19); external functional (F(2,61)=2.50; p=0.09))[38].

[38] See Appendix 21 for effect sizes and power calculations.

Post hoc (Dunnett's C) tests revealed that the bipolar group more frequently use external dysfunctional strategies to regulate positive emotion than the control group. The ANCOVAs also found no significant differences between the groups for their use of external functional ($F(4,2)=2.19;p=0.12$), internal functional ($F(2,54)=1.81$; $p=0.17$) or internal dysfunctional ($F(2,54)=1.54$; $p=0.22$) coping strategies. However, a significant effect of group was revealed for external dysfunctional coping strategies ($F2,51)=3.21$; $p=0.04$). A post hoc LSD test found that the bipolar group used external dysfunctional coping strategies significantly more often than the control group ($p=0.05$) to manage positive emotions when current mood state is accounted for.

In summary, although there are differences in the means shown in Figure 15, on the whole, no significant differences were found between the groups in the strategies they use to regulate positive emotion. The exception is external dysfunctional strategies which were more frequently used by the bipolar than the control group. However, it is worth noting that the results for external functional strategies just missed significance. These results remained even when current mood state was accounted for. This section has outlined the results following the 1st stage of the analyses where outliers were removed and the data for fear was transformed. Results were presented for each hypothesis in turn and additional findings were outlined. The next section presents the 2nd stage of the analyses.

3.7 Results - 2nd stage

In this final stage, the analyses described previously in Section 3.6 were repeated on the whole data set with outliers and the original BES fear data included (prior to the ln transformation) in order to determine whether these procedures made any differences to the main conclusions drawn.

Regarding the BES data, the ANOVAs carried out on the complete data set (with outliers included) confirmed the conclusions described above. This is also true for the fear data before and after transformation. With regards to the REQ data for negative emotion, the ANOVAs carried out on the complete data set also confirmed the conclusions presented above for all of the coping strategies, except for external functional coping strategies. When the outliers were removed, the results found that the control group used significantly more external functional strategies for managing negative emotion then the unipolar group. However, when all of the data was used, no significant differences were found between the groups. Finally, the ANOVA results for the REQ positive data also differed when the analyses were repeated on the complete data set. When the outliers were removed, the bipolar group was found to use external dysfunctional strategies significantly more frequently than the control group. However, no significant differences were found between the groups for any of the strategies used to manage positive emotion. A possible explanation for these findings is that although these results were significant when outliers were removed, they were not as strongly significant as the other results. Therefore, the significance was lost when the covariates were included. The last two sections of this chapter have reported the results of the analyses carried out on the data. A power calculation

was then preformed in order to assess the effect size. This is detailed in the next section.

3.8 Effect size

Calculating the effect size is important because it informs the researcher as to how powerful the tests were. There are several different methods for calculating effect size, in the current study partial eta squared (n^2) was used. This is found by calculating the sum of squares for the treatment by the total sum of squares. As set out by Clark-Carter (1997) partial eta squared can be converted into Cohen's (1988) statistic, therefore the cut off for a small effect size is an n^2 of 0.01, for a medium effect size n^2 is 0.05 and for a large effect size n^2 is 0.138. Using these cut offs, data in the current study were converted into Cohen's (1988) statistic in order to determine whether the effect size was large, small or medium (see Clark-Carter, 1997). Appendices 17-21 illustrate the effect size and power for all of the analyses carried out in the 1st stage, with outliers excluded. The thesis will focus on the results for the first stage given that they are likely to represent the findings more accurately[39]. These tables show that a large effect size was found for all of the ANOVAs where there were significant results.

3.9 Summary

To summarise, this chapter presented the results of the current study. Regarding the experience of the basic emotions, there are three key findings. Firstly, significantly different basic emotions are experienced between general, manic and depressed

[39] As described previously in Section 3.4.1

states in bipolar disorder. Manic states are predominantly characterised by happiness with anger and fear. General states are predominantly characterised by happiness, fear, disgust and anger. Depressed states on the other hand, are predominantly characterised by disgust, followed by sadness, fear and anger. Secondly, no significant differences were found in the emotional profiles of unipolar and bipolar depressed states with the exception of disgust, which is more often experienced in bipolar depressed states than unipolar. Thirdly, general states in the unipolar and control group were predominantly characterised by happiness, fear and anger. For the bipolar group these states were predominantly characterised by happiness, fear, disgust and anger.

With regards to coping strategies for negative emotion, although a trend in the results indicated that the control group used more functional strategies either of the clinical groups – this was only statistically significant for external functional strategies. Dysfunctional strategies were more frequently used to manage negative emotion in the clinical groups than the control groups however this was only statistically significant for the use of internal dysfunctional strategies. Furthermore, the bipolar group more frequently used external dysfunctional strategies than the other two groups. Finally with regards to coping strategies used for positive emotion, external dysfunctional strategies were more frequently used in the bipolar compared to the control group however, on the whole the groups manage positive emotions in similar ways.

CHAPTER 4 - DISCUSSION

4.1 Introduction to discussion

The following chapter discusses the results of the current study in the context of the theoretical rationale. The chapter addresses the results for each hypothesis in turn and the clinical implications of these are explored. The limitations and strengths of the study are also outlined in this chapter.

4.2 Summary of research

Despite the increased attention bipolar disorder has received in the psychological literature in the last decade, there remains a lack of theoretical models, which can adequately account for the key features of both mania and depression (Power, 2005). The current study sought to address this gap by testing the predictions made by the SPAARS model. This model proposes that all emotional experience (normal and disordered) can be derived from couplings between five basic emotions (i.e. happiness, sadness, fear, anger and disgust). The first aim of the current study was to explore the emotional couplings experienced in mania as well as those experienced in bipolar depression compared to unipolar depression. Using the BES, the results of the current study provided support for the proposals set out by the SPAARS model.

Previous research has also emphasised the crucial role that coping strategies play in the severity and duration of psychopathology (Nolen-Hoeksema, 1991). Furthermore emotional dysregulation is implicated in the DSM-IV criteria for half of the Axis I disorders and all of the Axis II disorders (Gross, 1999). Subsequently, the second

aim of the current study was to compare the coping strategies used in bipolar, unipolar and control groups for managing negative and positive emotion with the aim to expanding on the literature previously carried out in this area. The results found that dysfunctional strategies were frequently used by the clinical groups to regulate emotion, in particular external dysfunctional strategies were more frequently used to regulate both positive and negative emotion significantly more frequently than in the unipolar and control group.

4.2.1 Hypothesis one

The emotional profiles of mania will reveal elevated levels of happiness coupled with anger and/or fear.

The SPAARS model predicted that (hypo)mania was primarily a disorder of happiness coupled with anger and/or fear. The results of the study confirmed this hypothesis. Happiness was the most frequently reported basic emotion in mania. Furthermore, happiness was significantly more elevated in manic states than general and depressed states and was therefore a distinguishing feature of mania. Anger was the second most frequently reported emotion in manic states, however the levels reported in mania did not differ significantly from those in general and depressed states. In fact, anger was most often reported in depressed states and was significantly more elevated in these states than general states. Despite the fact that anger did not distinguish mania from general or depressed states in bipolar disorder, it was a common feature of mania, providing support for the SPAARS proposal that mania is derived predominantly from an emotional coupling of happiness and anger.

The finding that anger/irritability is a predominant feature of mania has been replicated in previous studies (Dayer, Aubry, Roth, Ducrey & Bertschy, 2000; Goodwin & Jamison, 1990; Perugi, Akiskal, Micheli, Musetti, Paiano, Quilici, Rossi & Cassano, 1997). In addition, Mansell & Pedley's (2008) review of large scale factor analytic studies[40] concluded four clusters of mania one of which was characterised by elevated levels of irritability. The DSM-IV criteria for (hypo)manic episodes also outlines irritability as a possible feature.

The current study also found that fear was commonly experienced in mania, however as with anger, no significant differences were found between the levels of fear reported in general and manic states. Fear was also most frequently reported in depressed states and was significantly more elevated in depressed than in general and manic states. Nonetheless, the results suggested that fear was an important feature of mania and this finding has also been replicated in previous large scale factor analytic studies (Mansell & Pedley, 2008).

In the current study, disgust and sadness were the least frequently reported emotion in mania. These emotions were significantly more elevated in depressed states than general and manic states and in addition, they were more elevated in general than manic states. This finding differs slightly from those described in Mansell & Pedley's (2008) review. These authors indicated that depressive symptoms were commonly found in mania and proposed that 'depressive mania' formed another cluster. This finding was not replicated in the current study. One possible explanation

[40] See Table 2 for a summary of the results of this review

is the difference in the samples used between the studies. The majority of the factor analytic studies reviewed by Mansell & Pedley (2008) comprised of participants who met the criteria for either manic or mixed states. In the current study, although it is possible that participants may have recalled a mixed episode, given that recall was retrospective in the current study, the researcher controlled for this by using the SCID-IV to ensure that the recalled episodes met the DSM-IV criteria for hypomanic or manic episodes. Therefore the difference may be explained by the absence of a mixed episode group in the current study. This is evidenced by a recent factor analytic study in which the sample used was similar to that used in the current study so that participants were in a purely manic episode (Picardi, Battisti, de Girolamo, Morosini, Norcio, Bracco, & Biondi, 2008). The findings concurred with those from the current study, failing to find a depressed factor.

In summary, the current study found support for the proposal in SPAARS that mania primarily occurs through a combination of happiness with anger/fear. Power & Dalgleish (1997, 2008) argue that the coupling of the basic emotions provides the basis for emotional disorders and that descriptions of the emotional disorders should begin with the identification of the basic emotions involved. The idea in SPAARS is that there are two routes to emotion and that two or more of the basic emotions can be processed in parallel via the schematic and associative routes. Findings from the current study therefore suggest that in mania, while happiness may be generated via the schematic route (involving effortful appraisal regarding the successful movement towards a valued role or goal), anger or fear may be generated at the same time via the associative route, perhaps due to previous experiences in a manic state where the

individual has learned, or is aware at some subconscious level, that they may indeed be unwell and therefore feels frustrated or fearful that the illness may present an obstacle to achievement of the valued role or goal.

4.2.2 Hypothesis two

The emotional profiles of bipolar and unipolar depression will reveal elevated levels of sadness coupled with disgust and/or fear and will not differ significantly from each other.

There are two parts to this hypothesis firstly, whether or not the emotional profiles reveal elevated levels of sadness and disgust as predicted in SPAARS and secondly, whether or not bipolar and unipolar depressed states differ significantly from each other. These will be discussed in turn.

The results of this study found support for the proposal in SPAARS that depression (in both unipolar and bipolar groups) comprises of an emotional coupling between sadness and disgust. The emotional profiles revealed that disgust is the most frequently experienced emotion in bipolar depression followed by elevated levels of sadness, fear and anger. While in unipolar depression, sadness is the most frequently experienced emotion followed by fear, disgust and anger. A previous study conducted by Power & Tarsia (2006) compared the emotional profiles of four groups (anxious, unipolar depressed, mixed (anxiety and depression) and a control group). They found that the emotional profiles of the unipolar depressed group comprised of elevated levels of sadness, fear and anger followed closely by disgust. The authors

concluded that sadness and disgust rather than guilt, as proposed in the DSM-IV criteria, were predominant features of unipolar depression. The results of the current study replicated and expanded these on findings indicating that bipolar depression is predominantly comprised of the same emotional coupling between sadness and disgust. The elevated levels of fear in both unipolar and bipolar depression may be accounted for by the high rate of comorbid anxiety that occurs with depressive disorders.

Partial support was found for the second part of the hypothesis – that bipolar and unipolar depression do not differ significantly from each other. No significant differences were found between bipolar and unipolar depression in the levels of sadness, fear, anger or happiness. However, disgust was significantly more elevated in bipolar depressed stated than unipolar depressed states. Some previous research has suggested that unipolar and bipolar depressed states are indistinguishable (Cuellar, Johnson & Winters, 2005). Previous research regarding dysfunctional cognitions has also found that many of the dysfunctional cognition observed in unipolar depression are also found in bipolar depression (Mansell & Scott, 2006). Furthermore, the DSM-IV criteria are the same for a depressed state in bipolar and unipolar depression. Overall, the results of the current study agree with those in previous studies that unipolar and bipolar depressed states do not differ significantly in the emotional profiles. However the findings do suggest that disgust is more elevated in bipolar depression than unipolar depression.

In summary, the current study found support for the proposal in SPAARS that depression predominantly comprises of a combination of sadness with disgust. Although fear and anger are also elevated, it is argued that it is disgust that plays a key role in the onset of depression. The idea in SPAARS is that the depressed individual may feel sadness due to effortful appraisal regarding the loss of a valued role or goal (resulting in the generation of loss at the schematic level). Disgust is generated at the same time via the associative route due to the individual's perception, possibly via repeated exposure to depressed episodes, that they are inadequate or have failed in their efforts to achieve a valued role or goal. As a result the self is viewed in terms of negative self aspects resulting in shame, guilt and low self esteem which SPAARS proposes are all derived from the basic emotion of disgust.

4.2.3 Hypothesis three

The clinical groups will more frequently use dysfunctional strategies to regulate negative and positive emotion than the control group.

4.2.3a The regulation of negative emotion between groups

Partial support was found for hypothesis three with respect to negative emotion. Internal dysfunctional strategies were the most frequently used strategies to regulate negative emotion by the clinical groups. Furthermore, the clinical groups used these strategies significantly more often than the control group to regulate negative emotion. Internal dysfunctional strategies are strategies which inhibit the processing of emotion and which draw upon internal resources (e.g. rumination "I dwell on my

thoughts and feelings", self mutilation "I harm or punish myself in some way" or "I keep the feeling locked up inside"). The finding that such strategies are used frequently in depression has been documented in the literature. Thomas and Bentall (2002) for example found that depression was strongly linked to rumination. In a later study of bipolar depressed patients, they found that rumination was the most frequently used response style in depression however, interestingly rumination was more evident in bipolar remitted group than the bipolar depressed group (Thomas, Knowles, Tai & Bentall, 2002). Nolen-Hoeksema and colleagues have also found that rumination is evident in depressed samples and furthermore that it predicts the duration and severity of depressed episodes (Nolen-Hoeksema, 1991; Nolen-Hoeksema, McBride & Larson, 1997).

In comparison to the other strategies, external dysfunctional strategies were the least frequently reported strategy by all three groups for regulating negative emotion, however while they were rarely used by the unipolar and control groups; they were reported significantly more often in the bipolar group. Therefore, the findings for external dysfunctional strategies do not support the hypothesis because all three groups rarely used these strategies to regulate negative emotion. However, while it is important to note that these strategies were rarely used, it is interesting that the bipolar group used them significantly more often than the unipolar and control groups. Within the external dysfunctional subscale, the most frequently used strategy was "I take my feelings out on others verbally (i.e. shouting, arguing)" with 60% of the sample reporting that they do this 'often', 'very often' or 'always'. Previous research has found an association between anger/aggressive behaviour and bipolar

disorder. A study by Perlis, Smoller, Fava, Rosenbaum, Nierenberg & Sachs (2004) for example, compared a sample of 50 participants with major depressive disorder and 29 participants with bipolar disorder who were currently in a purely depressed episode and found that anger attacks were twice as common in the bipolar group. The authors concluded that anger attacks might be a feature of bipolar depression. Similarly, Garno, Gunawardane & Goldberg (2008) found that both manic and depressed symptoms significantly predicted trait aggression in bipolar disorder.

The control group was found to use external functional strategies significantly more frequently than the unipolar group however, no significant differences were found between the bipolar and control group or between the clinical groups for these strategies. Furthermore, no significant differences were found at all between the groups for internal functional strategies and negative emotion. Although no significant differences were found for internal functional strategies or between the control and bipolar group for external functional strategies, a trend in the results indicated that the control group more frequently used these strategies. Therefore some support was found in support of the hypothesis. The failure to find some of these significant differences may be explained by the fact that many of the participants in the clinical groups had long standing diagnoses. They were recruited from a lithium clinic and from members of staff within a Community Mental Health Team. Therefore, these participants had received many years of medical (as well as probable psychological treatment at some point) and subsequently may have learned to develop more adaptive ways of regulating negative emotion. However, although this may have had some bearing on the results, this appears to have been minimal

given that significant differences were found between the control group and unipolar group for external functional strategies and that the clinical groups still used more dysfunctional strategies than the control group.

In summary, the results for the coping strategies between the groups for regulating negative emotion found that by far, internal dysfunctional strategies were the most frequently used strategy by both clinical groups. While external dysfunctional strategies were rarely used, they more often used by the bipolar group than the unipolar or control groups. Furthermore, there is some evidence in the current study to suggest that the control groups more frequently use functional strategies than the clinical groups. Taken together these results provide support for hypothesis three.

4.2.3b The regulation of positive emotion between groups

Hypothesis three was partially supported by the results with regards to positive emotion. External functional strategies were the most frequently used strategy to regulate positive emotion by all three groups followed by internal functional strategies. Internal dysfunctional strategies were the third most frequently used strategies by all groups with external dysfunctional strategies rarely used by any other groups. With the exception of external dysfunctional strategies, no significant differences were found between the groups in the use of any strategies for regulating positive emotion. However, a trend in the results indicated that the clinical groups used both types of functional strategies more frequently than the control group and furthermore, that these groups also used dysfunctional strategies more frequently than the control group. Again, this finding may be explained by the fact that many of

the participants in the clinical groups had long standing diagnoses and were recruited from a lithium clinic and from members of staff within a Community Mental Health Team and subsequently may have learned to develop more adaptive ways of regulating emotion.

The finding that the bipolar group used external dysfunctional strategies significantly more often than the control group is interesting (although it must be noted that these strategies were very rarely used by any of the groups). When the individual frequencies were analysed, the item "I take my feelings out on others verbally (i.e. shouting, arguing)" was the most frequently reported item in this subscale with 57% of the sample reporting that they use this strategy seldom or often. The REQ positive[41] asks participants about positive emotion generally rather than specifically about manic states for instance it asks about happiness and other complex emotions derived from it (such as joy and excitement). The current study shows that happiness is predominant in both manic and general states[42] and furthermore that in each of these states, anger is also elevated. Therefore, it is possible that the elevation of anger in these states may account for the finding that external dysfunctional strategies are significantly more elevated in the bipolar group.

4.2.4 Additional findings

In addition to testing the experimental hypotheses, this study also revealed significant differences in the emotional profiles between bipolar, unipolar and control

[41] See Appendix 11
[42] The emotional profiles of general states are discussed in more detail in section 4.2.4.

groups in general states (i.e. the emotions they experience generally, outwith episodes). The emotional profiles of the unipolar and control group were markedly similar. With the exception of fear, no significant differences found between these groups. The elevation of fear in this group may represent the high rate of comorbidity between anxiety and mood disorders. In these groups, general states are predominantly characterised by happiness, with anger, fear, disgust and sadness reported less frequently. Although generally the same pattern was found in the bipolar group, levels of fear, disgust and anger were also elevated in this group. In fact, disgust and anger were significantly more elevated in the bipolar group than the unipolar and control group. A trend in the results indicated that the same was true for fear, however the bipolar group only differed significantly for fear in comparison to the control group.

One possible explanation for the finding that negative emotions such as anger, fear and disgust are elevated in general states of bipolar disorder, and that fear is elevated in general states of the unipolar group, is the presence of subsyndromal symptoms between episodes. For instance, there is considerable evidence to suggest that in the course of unipolar depression patients frequently experience lower level symptoms in between full blown major depressed episodes (Kennedy, Abbott & Paykel, 2004; Judd, Akiskal, Schettler, Endicott, Maser, Solomon, *et al.,* 1998). In a large scale (n=253) longitudinal study of bipolar patients over a period of 18 months, participants were asymptomatic for a mean of 47% of the time and experienced mild symptoms 20% of the time, subsyndromal symptoms 23% of the time and major symptoms 10% of the time (Paykel, Abbott, Morriss, Hayhurst & Scott, 2006).

Furthermore, this study found that subsyndromal symptoms were twice as likely in bipolar disorder than major depressive disorder and that subsyndromal depressive symptoms were three times as likely as manic symptoms. Judd, Akiskal, Schettler, Endicott, Maser, Solomon, *et al.'s* (2002) study of 146 patients with bipolar disorder reports similar findings. It may have been the case in the current study that the elevated levels of fear experienced generally in the unipolar group, and the elevated levels of anger, fear and disgust experienced generally in the bipolar group, represent subsyndromal depressive symptoms.

Another possible explanation is that the emotions become coupled in general states as described previously (see section 4.2.1) in the manic state so that in between episodes of bipolar disorder the individual experiences happiness generated via the schematic route, and the appraisal that they are moving successfully towards a role or goal, but at the associative route may feel anger, fear or disgust due to the bipolar condition that they have and their perception that this may stand in the way of them achieving valued role or goals .

4.3 Clinical implications of the study

The results of the current study have three important clinical implications. Firstly, the results found support for the proposal in SPAARS that emotional disorders (as well as normal emotional experience) can be derived from five basic emotions (happiness, sadness, fear, anger and disgust) and that couplings between two or more of these emotions provide the basis for emotional disorders. Mania was found to predominantly comprise of a combination of happiness and anger, while sadness and

disgust predominantly characterised bipolar depressed states. Furthermore the study found that while happiness is predominant in general states, fear, disgust and anger are also elevated. These findings suggest that this model has clinical validity and is applicable to bipolar disorder. In terms of the applicability of these findings to clinical practice, these findings indicate that one of the goals of therapy may be to better understand the unique emotional profile of the individual's manic and depressed episodes and to attempt to disentangle these emotions from each other (Power & Schmidt, 2004).

Secondly, the study also it revealed important results particularly with regards to the role that disgust may play in bipolar disorder. Interestingly, not only was disgust a key feature in depressed episodes, but it was also a key feature of bipolar disorder generally (outwith manic and depressed episodes). Furthermore, the results suggested that the high level of disgust experienced in bipolar depression may distinguish it from unipolar depression. In agreement with Power & Tarsia's (2007) study, these results have important clinical implications for the DSM-IV criteria which currently emphasise the role of guilt. It is argued that guilt is derived from the basic emotion of disgust and in depression, the key issue in depression is that disgust is turned against the self so that some aspects of the self are considered to be repulsive and should be eliminated (Power & Schmidt, 2004). It is proposed therefore that it is disgust not guilt that plays a key role in the onset of depression.

In turn, the findings regarding disgust also have important clinical implications for the self concept and therapeutic work. Power and Dalgleish (2008) have suggested

that disgust may play an important role in some cases of suicide and parasuicide. It is possible that there is an association between the levels of disgust experienced in bipolar disorder and the high rate of suicide in this population. Some empirical findings have suggested that the self concept is organised differently in bipolar disorders so that the self is modularised around either positive or negative self aspects (Power, de Jong & Lloyd, 2002). Rather than these being integrated as they are in normal individuals they are modularised so that the self is defined entirely by positive or negative characteristics. Therefore in depressed states, positive aspects are ignored and in manic states negative self aspects are ignored resulting in extreme shifts in self esteem between manic and depressed states (hence why disgust was rarely experienced in manic states). As such part of the aim of clinical work should be to integrate these aspects into the self concept so that both negative and positive self aspects are considered (Power & Schmidt, 2004). This would involve enabling the individual to become more aware of and to experience the particular aspects that are perceived to be repulsive so that the emotion of disgust can be processed in a safe way. This is especially since the longitudinal course of bipolar disorder is dominated by depressed episodes (Judd & Akiskal, 2003) therefore suggesting that the experience of disgust may be more frequent as the illness progresses.

Thirdly, the current study revealed important results regarding the regulation of emotion. As in previous research, the clinical groups frequently used internal dysfunctional strategies (such as rumination and self mutilation) to regulate negative emotion. Such strategies have been found to increase the duration and severity of episodes (Nolen-Hoeksema, 1991). The results also revealed that external

dysfunctional strategies (particularly shouting and arguing with others) were used in the bipolar group as a means of regulating both positive and negative emotion. These findings bear clinical relevance in that they suggest that along with the common dysfunctional coping strategies present in mania (such as spending, risk taking and pleasure seeking) and depression (such as withdrawal from activities and social isolation), verbal aggression may also be an important focus of therapy.

4.4 Limitations of the research

Four limitations of the research were identified. Firstly, the methodology employed in the current study relied on retrospective recall from participants who had a longstanding diagnosis and who had experienced multi-episodes. While it is acknowledged that this is a limitation of the study, it would have been difficult to gain ethical approval to recruit patients currently in an acute episode of an illness given the impact that this may have had on informed consent. Furthermore, the current study employed the same methodology as the literature reviewed for example many of the studies reviewed in Mansell & Pedley's (2008) paper also relied on retrospective recall other studies include the work from Lam and colleagues (e.g. Lam, Wright & Smith, 2004).

A second limitation concerns the diagnosis of bipolar disorder. Bipolar disorder is historically diagnosed, in other words a diagnosis is made retrospectively once (hypo)manic or mixed episodes have been identified. Therefore, it is impossible for participants to recover from bipolar disorder. Furthermore, their current levels of functioning are not taken into account by the diagnosis so while a participant may

have been diagnosed as BDI initially, after treatment they may meet the criteria more accurately for BDII. In the current study participants were recruited from a lithium clinic and were therefore receiving medical treatment. Furthermore, many of the participants had long standing diagnoses. This is a limitation generally with the diagnosis of bipolar disorder and it is hard to see how this could have been overcome in the current study. Diagnoses in the current study were confirmed using the SCID[43] (a semi structured interview for DSM-IV diagnoses). Currently, ICD-10 and DSM-IV are considered the 'gold standard' for psychiatric diagnoses however, both carry the same issue of historical diagnosis for bipolar disorder.

The third limitation relates to the difficulty measuring emotion regulation strategies used. The current study relied on a self report measure however, some research has highlighted the difficulty with this approach given that the regulation of emotion is often unconscious. However, self report measures are a well established method of collecting data and are frequently used in psychological research. Furthermore the particular measure used in the current study (i.e. REQ) has been used in previous research which revealed that it has good internal reliability and consistency and was therefore a valid tool for the measurement of the regulation of emotion (Phillips & Power, 2007).

The fourth limitation with the study concerns the issue of multiple testing. Given that this study uses a series of ANOVAs, it is possible that there was an increased likelihood of finding a significant result. With regards to the multiple post hoc tests

[43] See Appendix 9

used in the study, a mixture of lenient and more conservative tests were used in order to ensure that significant results were not missed.

4.5 Strengths of the research

Despite the limitations described above, several strengths were also identified for the current research. Firstly, the study makes valuable contributions to the literature on bipolar disorder. Until recently bipolar disorders were rarely studied in the psychological literature. Although this has changed in the last ten years there has remained a lack of adequate theoretical models which can explain the complex features of bipolar disorder (Power, 2005). As argued in the introduction, existing models have either been too simplistic in their account of bipolar disorder, or they have been older models adapted specifically to bipolar disorders or they have focussed on one aspect such as cognition at the expense of emotion. The current study has contributed to the literature and helped to address this gap by testing the predictions made by the SPAARS model. This model is unique in that it attempts to account both for normal, everyday emotional experience as well as for the emotional disorders. In addition, it made several proposals regarding the key features of bipolar disorders (including mood fluctuations and shifts in self esteem). To date these were largely based on theory and given that the model itself is relatively new and research was needed to test its predictions and validate it. The current study found support for the predictions this model made therefore suggesting that the SPAARS model is clinically valid.

A second strength of the study is the finding that disgust plays a key role in bipolar disorder. Previous research had suggested that unipolar and bipolar depressed episodes were indistinguishable. Although the results of the current study largely supports this finding, one of the differences between the two was that disgust was more elevated in bipolar depressed states. The study also found that disgust was also elevated generally in bipolar disorder. The role that disgust plays in emotional disorders appears to have been overlooked in the literature. In line with SPAARS, the current study begins to address this gap and highlights the importance that this emotion may play not only in bipolar disorder but other forms of psychopathology as well. Finally, the current study relates to the large effect size found. The number of participants recruited in the study meant that large effect sizes were achieved indicating that the results of the study are therefore powerful and meaningful.

There are two other factors which contribute to the power of the results in the current study. Firstly, the fact that with the exception of the results for the use of external functional strategies when regulating negative emotion, all of the results in the current study remained the same when current mood state was controlled for. With regards to external functional strategies when regulating negative emotion, the original results (when mood state was not included) found that there was a significant difference between the groups however when mood state was controlled for there were no significant differences between the groups for this variable. This chance is attributed to chance because chance would predict that at least one of the results would change when mood state was controlled for. Furthermore, in comparison to the other significant results for the strategies with negative emotion, the p value for

external functional strategies was less significant (p0.02) and so this might indicate that although there was a difference the effect was too weak when current mood state was included.

Secondly, there were no significant differences between the groups for age, gender, marital status or current mood state which in turn also contributes to the power of the study. Although the control group were all employed due to the location of recruitment there were no significant differences between the clinical groups, and furthermore although the clinical groups were more educated than the control group, there were no differences between the clinical groups. As a result, the similarities between the groups on these variables also added to the power of the results.

4.6 Future research

This study tested the predictions that the SPAARS model made regarding the emotional couplings experienced in bipolar disorder. Previous research has investigated the emotional couplings in major depressive disorder and anxiety disorders (Power & Tarsia, 2006). Future research is needed to test the predictions that SPAARS model makes regarding the emotional couplings experienced in other psychiatric disorders[44].

The results from the current study and the study described concur with the proposal in SPAARS that disgust may play a central role not only in bipolar disorder, unipolar

[44] See Table 2 on page 61 for a summary of the predicted emotional couplings in the emotional disorders

depression and anxiety, but also in other psychiatric disorders. For instance, Power & Dalgleish (2008) suggest that disgust may also play a significant role in obsessive-compulsive disorders and eating disorders. Although research is increasingly recognising the role of shame in emotional and psychiatric disorders, the role of disgust has been overlooked in the literature and warrants more investigation in order to determine it role in other disorders.

4.7 Conclusions

The first aim of the current study was to investigate the basic emotions experienced in manic states, bipolar compared to unipolar depressed states and generally in a bipolar group compared to a unipolar and control group. The study used the Basic Emotions Scale (BES; Power, 2006) in order to address this aim. The results found that manic states were predominantly characterised by an emotional coupling between happiness and anger/fear. Bipolar and unipolar depressed states were both characterised by an emotional coupling between sadness and disgust although fear and anger were also elevated in both group in depressed states. Furthermore, the bipolar group experienced elevated levels of disgust significantly more frequently than the unipolar group in these states. The emotions experienced generally in the bipolar group differed from the unipolar and control group in that levels of disgust and anger were also significantly more elevated generally in this group. Furthermore, fear was more elevated in the clinical groups generally than in the control group. These results supported the predictions made by the SPAARS model that there are five basic emotions and that coupling between these emotions forms the basis for the

emotional disorders. The results also suggested that disgust plays a key role in bipolar disorder.

The second aim of the study was to compare the coping strategies frequently employed by a bipolar, unipolar and control group when regulating negative and positive emotions. The study used self report measures in the form of the Regulation of Emotion Questionnaire (REQ; Phillips & Power, 2007) to address these aims. The research found that the clinical groups more frequently use internal dysfunctional strategies to regulate negative emotion than the control group as found in previous research. Furthermore, the results indicated that the bipolar group use external dysfunctional strategies (particularly "I take my feelings out on others verbally (i.e. shouting and arguing)" significantly more frequently than the unipolar and control group. With regards to positive emotion, the results indicated that overall the three groups regulate positive emotion in similar ways however as with negative emotion, the bipolar group use external dysfunctional strategies (particularly "I take my feelings out on others verbally (i.e. shouting and arguing)") significantly more frequently than the control group. A trend in the results indicated that they also used these more than the unipolar group however this difference was not statistically significant.

The results of the current study are important because they offer further insights into the psychological approaches involved in bipolar disorder in two key ways. Firstly, the results have contributed to the search for a theoretical model that can account for bipolar disorder by testing the predictions made by SPAARS. Secondly, the results

suggest that disgust plays in a key role in bipolar disorder. This particular emotion has been overlooked in the literature but some research suggests that disgust may play a key role in other emotional disorders as well as in suicide and parasuicide (Power & Dalgleish, 2008). More research is needed to investigate the role of disgust in other emotional disorders. In conclusion, this study provides a valuable contribution to the literature suggesting that the SPAARS model has clinical validity in its application to bipolar disorder. However, this model is still relatively new and further research is needed to test the predictions that it makes in relation to other emotional disorders.

REFERENCES

Akiskal, H. S. (1986). A developmental perspective on recurrent mood disorders: A review of studies in man. *Psychopharmacology Bulletin, 22, (3), p579–586.*

Akiskal, H. S., Azorin, J. M., & Hantouche, E. G. (2003). Proposed multidimensional structure of mania: Beyond the euphoric–dysphoric dichotomy. *Journal of Affective Disorders, 73, p7–18.*

Akiskal, H. S., Bourgeois, M. L., Angst, J., Post, R., Moller, H. J. & Hirschfeld, R. (2000). Re-evaluating the prevalence of and diagnostic composition within the broad clinical spectrum of bipolar disorders. *Journal of Affective Disorders, 59, S5–S30.*

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders*. (4th ed). Washington, DC: APA.

Andlin-Sobocki, P. & Wittchen, H. U. (2005). Cost of affective disorders in Europe. *European Journal of Neurology, 12, (Supplement 1), p 34–38.*

Angst, J. (1988). Clinical course of affective disorders. In Helgason, T., & Daly, R. J. (Eds.), *Depressive Illness: Prediction of Course and Outcome* (pp. 1–48). Berlin: Springer.

Angst, J. (1998). The emerging epidemiology of hypomania and bipolar II disorder. *Journal of Affective Disorders, 50, p 143–151.*

Angst, J. & Sellaro, R. (2000). Historical perspectives and natural history of bipolar disorder. *Biological Psychiatry, 48, p 445–457.*

Angst, J. & Weiss, P. (1967). Periodicity of depressive psychoses. In Wittchen, H-U., Muhlig, S. & Pezawas, L. (2003). Natural course and burden of bipolar disorders. *International Journal of Neuropsychopharmacology, 6, p145–154.*

Arnold, M. (1960). *Emotion and personality.* New York Columbia: University Press.

Ayuso-Mateos, J. L., Vazquez-Barquero, J. L., Dowrick, C., Lehtinen, V., Dalgard, O. S., Casey, P., Wilkinson, C., Lasa, L., Page, H., Dunn G., Wilkinson, G., & the ODIN group. (2001). Depressive disorders in Europe: Prevalence figures from the ODIN study. *British Journal of Psychiatry, 179, p308-316.*

Baldessarini, R. J. (2000). A plea for integrity of the bipolar concept. *Bipolar Disorders, 2, p3-7.*

Barnard, P. (1985). Interacting cognitive subsystems: A psycholinguistic approach to short-term memory. In A. Ellis (Ed.), *Progress in the psychology of language* (Vol 2). London: Lawrence Erlbaum Associates Ltd.

Barnard, P. J. & Teasdale, J. D. (1991). Interacting cognitive subsystems: A systemic approach to cognitive-affective interaction and change. *Cognition and Emotion, 5, p 1-39.*

Bebbington, P. (2004). Classification and epidemiology of depression. In Power, M. J. (Eds). (2004). *Mood disorders: A handbook of science and practice.* Chichester: Wiley & Sons.

Bebbington, P., Hurry, J., Tennant, C., Sturt, E. & Wing, J. K. (1981). The epidemiology of mental disorders in Camberwell. *Psychological Medicine, 11, p561-580.*

Bech, P. (2002). The Bech-Rafaelsen Mania Scale in clinical trials of therapies for bipolar disorder: A 20-year review of its use as an outcome measure. *CNS Drugs, 16, (1), p47-63*

Bech, P., Rafaelsen, O. J., Kramp, P. & Bolwig, T. G. (1978). The mania rating scale: scale construction and inter-observer agreement. *Neuropharmacology, 17, (6), p430-1.*

Beck, A. T. (1983). Cognitive Therapy of Depression: New perspectives. In Power, M. J. (Eds). (2004). *Mood disorders: A handbook of science and practice.* Chapter 12. Chichester: Wiley & Sons.

Beck, A. T., Rush, A. J., Shaw, B. & Emery, G. (1979). *Cognitive Therapy of Depression.* Guilford Press, New York.

Beck, A. T., Steer, R. A. & Brown, G. K. (1996). *Manual for the Beck Depression Inventory-II.* San Antonio, Tex: Psychological Corporation.

Beck, A. T., Steer, R. A. & Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8*, p77-100.

Bentall, R. P., Claridge, G. S. & Slade, P. D. (1989). The multidimensional structure of schizotypal traits: A factor-analytic study with normal subjects. *British Journal of Psychology, 28, p363-375.*

Brown, G. W., & Harris, T. O. (1989). *Life events and illness.* New York: Guilford Press.

Brown, T., Campbell, L. A., Lehman, C. L., Grisham, J. R., & Mancill, R. B. (2001). Current and lifetime comorbidity of the DSM-IV anxiety and mood disorders in a large clinical sample. *Journal of Abnormal Psychology, 110, p585-599.*

Cassano, P. & Fava, M. (2002). Depression and public health: An overview. *Journal of Psychosomatic Research, 53, (4), p849–857.*

Cassidy, F. & Carroll, B. J. (2001). The clinical epidemiology of pure and mixed manic episodes. *Bipolar Disorders, 3, p 35–40.*

Cassidy, F., Murray, E., Forest, K. & Carroll, B. J. (1998). Signs and symptoms of mania in pure and mixed episodes. *Journal of Affective Disorders, 50, p187−201.*

Champion, L. A. & Power, M. J. (1995). Social and cognitive approaches to depression: Towards a new synthesis. *British Journal of Clinical Psychology, 34, p 485-503.*

Clark-Carter, D. (1997). *Doing quantitative research: From design to report.* Sussex: Psychology Press.

Cohen, J. (1988). *Statistical power for the behavioural sciences.* (2nd Ed.). Hillsdale, NJ: Erlbaum.

Coryell, W., Endicott, J. & Kendler, M. (1992). Rapid cycling affective disorder: Demographics, diagnosis, family history and course. *Archives of General Psychiatry, 49, p129-131.*

Coryell, W., Leon, A., Winokur, G., Endicott, J., Keller, M., Akiskal, H. & Solomon, D. (1996). Importance of Psychotic Features to Long-Term Course in Major Depressive Disorder. *American Journal of Psychiatry, 153, (4), p483-489.*

Craddock, N. & Owen, M. S. (2005). The beginning of the end of the Kraeplinian dichotomy. *British Journal of Psychiatry, 186, p364-366.*

Cuellar, A. K., Johnson, S. L. & Winters, R. (2005). Distinctions between bipolar and unipolar depression. *Clinical Psychology Review, 25, p307-339.*

Darwin, C. (1872/1965). *The expression of emotions in man and animals.* Chicago: Chicago University Press.

Das Gupta, R. & Guest, J. F. (2002). Annual cost of bipolar disorder to UK society. *British Journal of Psychiatry, 180, 227-233.*

Dayer, A., Aubry, J. M., Roth, L., Ducrey, S. & Bertschy, G. (2000). A theoretical reappraisal of mixed states: dysphoria as a third dimension. *Bipolar Disorders, 2, (4), p316– 324.*

Del-Ben, C. M., Rodrigues, C. R. C. & Zuardi, A. W. (1996). Reliability of the Portuguese version of the structured clinical interview for DSM-III-R (SCID) in a Brazilian sample of psychiatric outpatients. *Brazilian Journal of Medical and Biological Research*, *29, p1675 -1682.*

Depue, R. A., Krauss, S. P. & Spoont, M. R. (1987). A two-dimensional models of seasonal bipolar affective disorder. In Power, M. J. (Eds). (2004). *Mood disorders: A handbook of science and practice*. Chapter 12. Chichester: Wiley & Sons.

Descartes, R. (1649,1989). *The passions of the soul.* Indianapolis, IN: Hackett.

Dilsaver, A. C., Chen, Y. R., Shoaib, A. M. & Swann, A. C. (1999). Phenomenology of mania: evidence for distinct depressed, dysphoric, and euphoric presentations. *American Journal of Psychiatry, 156, p426−430.*

Dutton, G. R., Jones, G. N., Bodenlos, J., Ancona, M. & Brantley, P. J. (2004). Validation of the Beck Depression Inventory-II in a Low-Income African American Sample of Medical Outpatients. *Psychological Assessment, 17, (1) p110-114.*

Ehlers, C. L., Frank, E. & Kupfer, D. J. (1988). Social zeitgebers and biological rhythms: a unified approach to understanding the etiology of depression. *Archives of General Psychiatry, 45, p948-952*.

Ekman, P. (1999). Basic emotions. In (Eds.) T. Dalgleish, & M. J. Power. (1998). *Handbook of cognition and emotion.* Chichester: Wiley & Sons.

Ernst, C., & Angst, J. (1992). The Zurich Study XII: sex differences in depression: evidence from longitudinal epidemiological data. *European Archives of Psychiatry and Clinical Neuroscience, 241, p222-230.*

Fava, M. & Kendler, K. (2000). Major depressive disorder. *Neuron, 28, (2), p335–341.*

Fennig, S., Craig, T., Lavelle, J., Kovasznay, B. & Bromet, E. J. (1994). Best estimate versus structured interview – based diagnosis in first admission psychosis. *Comprehensive Psychiatry, 35, (5), p 341-348.*

First, M. B., Gibbon, M., Spitzer, R.L. & Williams, J. B. W. (2002). *Structured Clinical Interview for DSM-IV Axis I Disorders. Research version, Patient edition with psychotic screen. (SCID-I/P W/Psy Screen).* New York. Biometrics Research. New York State Psychiatric Institute.

Frank, E., Swartz, H. A. & Kupfer, D. J. (2000). Interpersonal and social rhythm therapy: managing the chaos of bipolar disorder. *Biological Psychiatry, 48, p 593-604.*

Freeman, M. P., WosnitzerSmith, K., Freeman, S. A., McElroy, S. L., Kmetz, G. F., Wright, R. & Keck Jr, P.E. (2002). The impact of reproductive events on the course of bipolar disorder in women. *Journal of Clinical Psychiatry, 63, (4), p 284-287.*

Garno, J. L., Gunawardane, N. & Goldberg, J. F. (2008). Predictors of trait aggression in bipolar disorder. *Bipolar Disorders, 10, p285–292.*

George, E. L., Miklowitz, D. J., Richards, J. A., Simoneau, T. L., & Taylor, D. O. (2003). The comorbidity of bipolar disorder and axis II personality disorders: prevalence and clinical correlates. *Bipolar Disorders, 5, p115–122.*

Ghaemi, S. N., Boiman, E. E. & Goodwin, F. K. (2000). Diagnosing bipolar disorder and the effect of antidepressants: a naturalistic study. *Journal of Clinical Psychiatry, 61, p804–808.*

Ghaemi, S. N., Sachs, G. S., Chiou, A. M., Pandurangi, A. K. & Goodwin, K. (1999). Is bipolar disorder still underdiagnosed? Are antidepressants overutilized? *Journal of Affective Disorders, 52 p135–144.*

Gitlin, M. J., Swendsen, J., Heller,T. L. & Hammen, C. (1995). Relapse and impairment in bipolar disorder. *American Journal of Psychiatry, 152, p1635-1640.*

Goldberg, J. F., Gerstein, R. K., Wenze, S. J., Welker, T. M. & Beck, A. T. (2008). Dysfunctional attitudes and cognitive schemas in bipolar manic and unipolar depressed outpatients: Implications for cognitively based psychotherapies. *Journal of Nervous and Mental Disease, 196, (3), p207-210.*

Gonzalez-Pinto, A., Ballesteros, J., Aldama, A., Perez de Heredia, J. L., Guiterrez, M., Mosquera, F. & Gonzalez-Pinto, A. (2003). Principal components of mania. *Journal of Affective Disorders, 76, p95-102*.

Goodwin, F. K. & Jamieson, K. R. (1990). *Manic depressive illness.* Oxford: Oxford University Press.

Goplerud, E. & Depue, R. A. (1985). Behavioural response to naturally occurring stress in cyclothymia and dysthymia. *Journal of Abnormal Psychology, 94, (2), p128-139.*

Gray, J. A. (1976). The behavioural inhibition system: A possible substrate for anxiety. In M. P. Feldman & A. M. Broadhurst (Eds.), *Theoretical and experimental bases of behaviour modification.* Chichester: Wiley.

Gray, J. A. (1982). *The neuropsychology of anxiety*. Oxford: Oxford University Press.

Gross, J. J. (1999). Emotion regulation: Past, present, future. *Cognition and Emotion*, *13, (5), p551–573.*

Groth-Marnat, G. (Ed.). (2003) (4th Edition). *Handbook of psychological assessment.* Wiley & Sons: New Jersey.

Guze, S. B. & Robins, E. (1970). Suicide and primary affective disorders. *British Journal of Psychiatry, 117, p437-438.*

Hammen, C., Henry, R. M., & Daley, S. E. (2004). Effects of stress and social support on the recurrence of bipolar disorder. *Journal of Affective Disorders, 82, 91, p143-147.*

Hasin, D. S., Goodwin, R. D., Stinson, F. S. & Grant, B. F. (2005). Epidemiology of Major Depressive Disorder: Results From the National Epidemiologic Survey on Alcoholism and Related Conditions. *Archives of General Psychiatry, 62, (10), p1097-1106.*

Hirschfeld, R. M., Lewis, L. & Vornik, L. A. (2003). Perceptions and impact of bipolar disorder: how far have we really come? Results of the national depressive and manic-depressive association 2000 survey of individuals with bipolar disorder. *Journal of Clinical Psychiatry, 64, p 161–174.*

Isometsae, E. T. (1993). Course, outcome and suicide risk in bipolar disorder: a review. *Psychiatrica Fennica, 24, 113-124.*

Jahoda, M. (1982). Employment and unemployment: A social psychological analysis. Cambridge: Cambridge University Press.

James, W. (1884). What is an emotion? *Mind, 9, p188-205.*

Jenkins, R., Bebbington, P. E., Brugha, T., Farrell, M., Gill, B., Lewis, G., Meltzer, H. & Petticrew, M. (1997). The National Psychiatric Morbidity Surveys of Great Britain-strategy and methods. *Psychological Medicine, 27, p765-744.*

Joffe, R. T., MacQueen, G. M., Marriott, M. & Young, L. T. (2004). A prospective, longitudinal study of percentage of time spent ill in patients with bipolar 1 or bipolar 2 disorders. *Bipolar Disorders, 6, p62-66.*

Johnson, S. L., Cueller, A. K., Ruggero, C., Winett-Perlman, C., Goodnick, P., White, R. & Miller, I. (2008). Life Events as Predictors of Mania and Depression in Bipolar I Disorder. *Journal of Abnormal Psychology, 117, (2), p268–277.*

Johnson, S. L., Meyer, B., Winett, C. & Small, J. (2000). Social support and self-esteem predict changes in bipolar depression but not mania. *Journal of Affective Disorders. 58, 921, p79-86.*

Johnson, S. L., Winett, C., Meyer, B., Greenhouse, W. & Miller, I. (1999). Social support and the course of bipolar disorder. *Journal of Abnormal Psychology, 108, p558–566.*

Jones, S. H. (2001). Circadian rhythms, multilevel models of emotion and bipolar disorder – An initial step towards integration? *Clinical Psychology Review, 21, (8), p 1193-1209.*

Jorm, A. F. (1987). Sex and age differences in depression: A quantitative synthesis of published research. *Australian and New Zealand Journal of Psychiatry, 21, p46-53.*

Joyce, P. R. (1985). Illness behaviour and rehospitalisation in bipolar affective disorder. *Psychological Medicine, 15, p521-525.*

Judd, L. L. & Akiskal, H. S. (2003). Depressed episodes dominate the longitudinal course of bipolar disorder. *Current Psychiatric Reports, 5, p 417-418.*

Judd, L. L., Akiskal, H. S., Schettler, P. J., Endicott, J., Maser, J., Solomon, D. A., Leon, A. C., Rice, J. A. & Keller, M. B. (2002). The long-term natural history of the weekly symptomatic status of bipolar I disorder. *Archives of General Psychiatry, 59, (6), p530-537.*

Kasanin, J. (1933). The acute schizoaffective psychoses. *American Journal of Psychiatry, 90, p97-126.*

Keller, M. B., Klerman, G. L. & Hirschfeld, R. M. A. (1986). Differential outcome of pure manic, mixed/cycling, and pure depressive episodes in patients with bipolar illness. *Journal of the American Medical Association, 255, p3138–3142.*

Kennedy, N., Abbott, R. & Paykel, E. S. (2004) Longitudinal syndromal and sub-syndromal symptoms after severe depression: 10-year follow-up study. *British Journal of Psychiatry, 184, p330-336.*

Kessler, R. C., Bergund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K . R., Rush, A. J., Walters, E. E., & Wang, P. S. (2003). The epidemiology of Major

Depressive Disorder: Results from the National Comorbidity Survey – Replication (NCS-R). *Journal of the American Medical Association, 289, p3095-3105.*

Kessler, R. C., McGonagle, K. A., Swartz, M., Blazer, D. G., & Nelson, C. B. (1993). Sex and depression in the National Comorbidity Survey, I: lifetime prevalence, chronicity and recurrence. *Journal of Affective Disorders, 29, p85-96.*

Klerman, G. L., Weissman, M. M., Rounsaville, B. J. & Chevron, E. S. (1984). *Interpersonal Psychotherapy of Depression.* Basic Books, Inc: New York.

Korczak, D. J. & Goldstein, B. I. (2009). Childhood onset major depressive disorder: Course of illness and psychiatric comorbidity in a community sample. *Journal of Pediatrics, 155, (1), p11-23.*

Kraeplin, E. (1921). *Manic Depressive Insanity and Paranoia.* (Ed. G. M. Robertson, Trans. R. M. Barclay). Edinburgh: Livingstone.

Kuehner, C., Buerger, C., Keller, F. & Hautzinger, M. (2007). Reliability and validity of the Revised Beck Depression Inventory (BDI-II). Results from German samples. *Nervenarzt, 78, p651-656.*

Lam, D. H., Green, B., Power, M. J. & Checkley, S. (1994). The impact of social cognitive variables on the initial level of depression and recovery. *Journal of Affective Disorders, 32, p75-83.*

Lam, D. H., Green B., Power, M. J., & Checkley, S. (1996). Dependency, matching adversities, length of survival and relapse in major depression. *Journal of Affective Disorders, 37, p81-90.*

Lam, D. H., Jones, S. H., Haywood, P. & Bright, J. A. (1999). *Cognitive Therapy for Manic Depression.* Chichester: Wiley.

Lam, D. H., Watkins, E. R., Hayward, P., Bright, J., Wright, K., Kerr, N., Parr-Davis, G., & Sham, P. (2003). A randomised controlled study of cognitive therapy for relapse prevention for bipolar affective disorder: outcome of the first year. *Archives of General Psychiatry, 60, (2), p145-152.*

Lam, D. H. & Wong, G. (1997). Prodromes, coping strategies, insight and social functioning in bipolar affective disorders. *Psychological Medicine, 27, p 1091, 1100.*

Lam, D. H., Wong, G. & Sham, P. (2001). Prodromes, coping strategies and course of illness in bipolar affective disorder – a naturalistic study. *Psychological Medicine, 31, p 1397-1402.*

Lam, D. H., Wright, K. & Smith, N. (2004). Dysfunctional attitudes: Extreme goal-attainment beliefs in remitted bipolar patients. *Journal of Affective Disorders, 79, p193-199.*

Lish, J. D., Dime-Meenan, S., Whybrow, P. C., Price, R. A. & Hirschfeld, R. M. (1994). The National Depressive and Manicdepressive Association (DMDA) survey of bipolar members. *Journal of Affective Disorders, 31, p281–294.*

Lazarus, R. S. & Folkman, S. (1984). *Stress, Appraisal and Coping*. New York: Springer.

Lomax, C. L., Barnard, P. J. & Lam, D. (2009). Cognitive processing in bipolar disorder conceptualized using the Interactive Cognitive Subsystems (ICS) model. *Psychological Medicine, 39, p773-783.*

Lozano, B.E. & Johnson, S. L (2001). Can personality traits predict increases in depression and mania symptoms? *Journal of Affective Disorders, 63, (1-3), p103-111.*

MacQueen, G. M., Young, L. T., Robb, J. C., Marriott, M., Cooke, R. G. & Joffe, R.T. (2000). Effect of number of episodes on wellbeing and functioning of patients with bipolar disorder. *Acta Psychiatrica Scandinavica, 101, p374–381.*

Mansell, W. & Pedley, R. (2008). The ascent into mania: A review of psychological processes associated with the development of manic symptoms. *Clinical Psychology Review, 28, p494-520.*

Mansell, W. & Scott, J. (2006). Dysfunctional beliefs in individuals with bipolar disorders. In S. H. Jones & R. P. Bentall. (Eds.) *The psychology of bipolar disorders.* Chapter 4. Oxford: Oxford University Press.

Mantere, O., Suominen, K., Leppamaki, S., Valtonen, H., Arvilommi, P., & Isometsa, E. (2004). The clinical characteristics of DSM-IV bipolar I and II disorders: baseline findings from the Jorvi Bipolar Study (JoBS). *Bipolar Disorders, 6, p395–405.*

Marneros, A. & Brieger, P. (2002). Prognosis of bipolar disorder: a review. In: M. Maj, H. S. Akiskal, J. J. Lopez-Ibor & N. Sartorius. (Eds.). *Bipolar Disorder*. Chapter 2. John Wiley and Sons.

McGuffin, P., Farmer, A. & Harvey, I. (1991). A polydiagnostic application of perational criteria in studies of psychotic illness. Development and reliability of the OPCRIT system. *Archives of General Psychiatry, 48, p764-770.*

Metzger, R. L. (1976). A reliability and validity study of the State-Trait Anxiety Inventory. *Journal of Clinical Psychology, 32, (2), p276-278.*

Meyer, B., Johnson, S. L. & Winters, R. (2001). Responsiveness to threat and incentive in bipolar disorder: Relations of the BIS/BAS scales with symptoms. *Journal of psychopathology and Behavioural Assessment, 23, p133-143.*

Mustafa, H. M., Rush, A. J., Sackeim, H. A., Wisniewski, S. R., McClintock, S. M., Craven, N., Holiner, J., Mitchell, J. R ., Balasubramani, G. K. & Hauger, R. (2005). Age-Related Characteristics of Depression: A Preliminary STAR*D Report. *American Journal of Geriatric Psychiatry, 13, (10), p852-860.*

National Institute for Clinical Excellence (NICE). (2004). *Depression: Management of depression in primary and secondary care. National Clinical Practice Guideline Number 23.* The British Psychological Society & The Royal College of Psychiatrists.

Nolen-Hoeksema, S. (1987). Sex Differences in Unipolar Depression: Evidence and Theory. *Psychological Bulletin, 101, (2), p259-282.*

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology, 100, p569–582.*

Nolen-Hoeksema, S., McBride, A. & Larson, J. (1997). Rumination and psychological distress among bereaved partners. *Journal of Personality and Social Psychology, 72, p855-862.*

Ortony, A. & Turner, W. (1990). What's basic about "basic" emotions? *Psychological Review, 97, p315-331.*

Otto, M. W., Perlman, C. A., Wernicke, R., Reese, H. E., Bauer, M. S. & Pollack, M. H. (2004). Posttraumatic Stress Disorder in patients with bipolar disorder: A review of prevalence, correlates and treatment strategies. *Bipolar Disorders, 6, p 470-479.*

Otto, M. W., Simon, N. M., Wisniewski, S. R., Miklowitz, D. J., Kogan, J. N., Reilly-Harrington, N. A., Frank, E., Nierenberg, A. A., Marangell, L. B., Sagduyu, K., Weiss, R. D., Miyahara, S., Thase, M. E., Sachs, G. S., & Pollack, M. H. (2006). Prospective 12-month course of bipolar disorder in out-patients with and without comorbid anxiety disorders . *British Journal of Psychiatry, 189, p20-25.*

Palmer, A. & Barnard, P. J. (2003). The immediate processing of schema discrepant meaning in bipolar disorder. *Bipolar Disorders Supplement, 5, (1), p 73.*

Paykel, E. S., Abbott, R., Morriss, R., Hayhurst, H., & Scott, J. (2006). Sub-syndromal and syndromal symptoms in the longitudinal course of bipolar disorder. *British Journal of Psychiatry, 189, p118 -12 3.*

Perlis, R. H., Smoller, J. W., Fava, M., Rosenbaum, J. F., Nierenberg, A. A. & Sachs, G. S. (2004). The prevalence and clinical correlates of anger attacks during depressive episodes in bipolar disorder. *Journal of Affective Disorders, 79, p291–295.*

Perugi, G., Akiskal, H. S., Micheli, C., Musetti, L., Paiano, A., Quilici, C., Rossi, L. & Cassano, G. B. (1997). Clinical subtypes of bipolar mixed states: validating a

broader European definition in 143 cases. *Journal of Affective Disorders, 43, (3), p169– 180.*

Perugi, G., Micheli, C., Akiskal, H. S., Madaro, D., Socci, C., Quilici, C. & Musetti, L. (2000). Polarity of the first episode, clinical characteristics, and course of manic depressive illness: A systematic retrospective investigation of 320 bipolar I patients. *Comprehensive Psychiatry, 41, p13–18.*

Phillips, K. F. V. & Power, M. J. (2007). A New Self-Report Measure of Emotion Regulation in Adolescents: The Regulation of Emotions Questionnaire. *Clinical Psychology and Psychotherapy, 14, p145–156.*

Picardi, A., Battisti, F., de Girolamo, G., Morosini, P., Norcio, B., Bracco, R., & Biondi, M. (2008). Symptom structure of acute mania: A factor study of the 24-item Brief Psychiatric Rating Scale in a national sample of patients hospitalized for a manic episode *Journal of Affective Disorders , 108 , p183–189.*

Placidi, G. F., Signoretta, S., Liguori, A., Gervasi, R., Maremanni, I., & Akiskal, H. S. (1998). The Semi-Structured Affective Temperament Interview (TEMPS-I): reliability and psychometric properties in 1010 14-26 year-old students. *Journal of Affective Disorders, 47, p1-10.*

Power, M. J. (2005). Psychological approaches to bipolar disorders: A theoretical critique. *Clinical Psychology Review, 25,(8), p1101-1122.*

Power, M. J. (2006). The structure of emotion: An empirical comparison of six models. *Cognition and Emotion, 20, p694–713.*

Power, M. J. (2007). The Multistory Self: Why the Self Is More Than the Sum of Its Autoparts. *Journal of Clinical Psychology: In Session, 63, (2), p187–198.*

Power, M. J. & Dalgleish, T. (1997). *Cognition and emotion: From order to disorder.* Hove: Psychology Press.

Power, M. J. & Dalgleish, T. (1999). Two routes to emotion: Some implications of multi-level theories of emotion for therapeutic practice. *Behavioural and Cognitive Psychotherapy, 27, p129-141.*

Power, M. J. & Dalgleish, T. (2008). *Cognition and Emotion: From order to disorder.* 2nd Edition. Sussex: Psychology Press.

Power, M. J., de Jong, F. & Lloyd, A. (2002). The organisation of the self concept in bipolar disorders: An empirical study and replication. *Cognitive Therapy and Research, 26, p 553-561.*

Power, M. J., Katz, R., McGuffin, P., Duggan, C. F., Lam, D. & Beck, A. T. (1994). The Dysfunctional Attitudes Scale (DAS): A comparison of forms A and B and

proposal for a new sub-scaled version. *Journal of Research into Personality, 28, p263-276.*

Power, M. J. & Schmidt, S. (2004). Emotion-focussed treatment of unipolar and bipolar mood disorders. *Clinical Psychology and Psychotherapy, 11, p44-57.*

Power, M. J., & Tarsia, M. (2007). Basic and Complex Emotions in Depression and Anxiety. *Clinical Psychology and Psychotherapy, 14, p19–31.*

Quek, K. F., Phil, M., Low, W. Y., Razack, A. H., Loh, C. S. & Chua, C. B. (2004). Reliability and validity of the STAI in a Malaysian population. *Medical Journal of Malaysia, 59, (2), p258-267.*

Ragson, N., Bauer, M., Grof, P., Gyulai, L., Elamn, S., Glenn, T. & Whybrow, P.C. (2005). Sex specific self reported mood changes by patients with bipolar disorder. *Journal of Psychiatric Research, 39, (1), p77-83*

Ramana, R. & Bebbington, P. (1995). Social influences on bipolar affective disorders. *Social Psychiatry and Psychiatric Epidemiology, 30, p152-160.*

Reilly-Harrington, N. A., Alloy, L. B., Fresco, D. M. & Whitehouse, W. G. (1999). Cognitive styles and life events interact to predict bipolar and unipolar symptomatology. *Journal of Abnormal Psychology, 108, p567–578.*

Rossi, A., Daleluzzo, E., Arduini, L., Di Domenico, M., Pollice, R. & Petruzzi, C. (2001). A factor analysis of signs and symptoms of the manic episode with Bech-Rafaelson Mania and Melancholia Scales. *Journal of Affective Disorders, 64, p267–270.*

Rossi, A., Marinangeli, M. G., Butti, G., Scinto, A., Di Cicco, L., Kalyvoka, A. & Petruzzi, C. (2001). Personality disorders in bipolar and depressive disorders. *Journal of Affective Disorders*, *65, (1), p3-8.*

Rule, W. R. & Traver, M. D. Test-Retest Reliabilities of State-Trait Anxiety Inventory in a Stressful Social Analogue Situation. *Journal of Personality Assessment, 47, (3), p276-277.*

Runa, I. U., Emine, G. K., Bedriye, O., Mert, U. & Hakan T. (2008). Psychometric Properties and Cut-off Scores of the Beck Depression Inventory-II in Turkish Adolescents. *Journal of Clinical Psychology in Medical Settings, 15, (3), p225-233.*

Russell, J. (1994). Is there a universal recognition of facial expression? A review of the cross-cultural studies. *Psychological Bulletin, 115, p102-141.*

Sartorius, N. (2001). The economic and social burden of depression. *Journal of Clinical Psychiatry, 6, (Supplement 15), p8–11.*

Sato, T., Bottlender, R., Kleindienst, N. & Moeller, H-J. (2002). Syndromes and phenomenological subtypes underlying acute mania: A factor analytic study of 576 manic patients. *American Journal of Psychiatry, 159, p968–974.*

Sato, T., Bottlender, R., Sievas, M., Schroter, A., Hecht, S., Moller, H-J. (2003). Long-term inter-episode stability of syndromes underlying mania. *Acta Psychiatrica Scandinavica, 108, p310–313.*

Schneider, B., Maurer, K., Sargk, D., Heiskel, H., Weber, B., Frolich, L., Georgi, K., Fritze, J. & Seider, A. (2004). Concordance of DSM-IV Axis I diagnoses by personal and informant's interview. *Psychiatric Research, 127, (1-2), p121-136.*

Shear, M. K., Greeno, C., Kang, J., Ludewig, D., Frank, E., Swartz, H. A. & Hanekamp, M. (2000). Diagnosis of nonpsychotic patients in community clinics. *American Journal of Psychiatry, 157, p 581-587.*

Showers, C. J. (1992). Compartmentalization of positive and negative self knowledge: Keeping bad apples out of the bunch. *Journal of Personality and Social Psychology, 62, p1036–1049.*

Simon, N. M., Otto, M. W., Wisniewski, S. R., Fossey, M., Sagduyu, K., Frank, E., Sachs, G. S., Nierenberg, A. A., Thase, M. E. & Pollack, M. H. (2004) Anxiety disorder comorbidity in bipolar disorder: data from the first 500 STEP-BD participants. *American Journal of Psychiatry, 161, p2222-2229.*

Simpson, S. G. & Jamison, K. R. (1999). The risk of suicide in patients with bipolar disorders. *Journal of Clinical Psychiatry, 60, (Suppl.2), p53–56.*

Simpson, H. B., Nee, J. C. & Endicott, J. (1997). First-Episode Major Depression: Few Sex Differences in Course. *Archives of General Psychiatry, 54, (7), p633-639.*

Singleton, N., Bumpstead, R., O'Brien, M., Lee, A. & Meltzer, H. (2001). *The prevalence of psychiatric morbidity amoung adults living in private households*. London: HMSO.

Skre, I., Onstad, W., Targersen, S. & Kringlen, E. (1991). High interrater reliability for the structured clinical interview for DSM-III-R Axis I (SCID-I). *Acta Psychiatrica Scandinavica, 84, p167-173.*

Smeets, G. & Merckelbach, H. (1996). Panic disorder and right hemisphere reliance. *Anxiety, Stress and Coping, 10, (3), p245-255.*

Spielberger, C. D., Gorsuch, R. L. & Lushene, R. (1983). *Manual for the State-Trait Anxiety Inventory: STAI (Form Y).* Palo Alto, CA: Consulting Psychologists Press.

Steiner, J. L., Tebes, J. K., Sledge, W. H., Sledge, W. & Walker, M.L. (1995). A comparison of the structured clinical interview for DSM-III-R and clinical diagnoses. *Journal of Nervous and Mental Disease, 183, (6), p365-369.*

Swann, A. C., Janicak, P. L., Calabrese, J. R., Bowden, C. L., Dilsaver, S. C., Morris, D. D., Petty, F. & Davis, J. M. (2001). Structure of mania: Depressive, irritable, and psychotic clusters with different retrospectively-assessed course patterns of illness in randomised clinical trial participants. *Journal of Affective Disorders, 67, (1-3) p123−132.*

Targosz, S., Bebbington, P., Lewis, G., Brugha, T., Jenkins, R., Farrell, M., & Meltzer, H. (2003). Lone mothers, social exclusion and depression. *Psychological Medicine, 33, (4), p715-722*.

Teasdale, J. D. (1996). Clinically relevant theory: integrating clinical insight with cognitive science. In Salkoviskis, P, M. (Eds). *Frontiers of cognitive therapy.* New York: Guilford Press.

Teasdale, J. D. (1999). Multi-level theories of cognition-emotion relations. In Dalgeish, T. & Power, M, J. (Eds). (1999). *Handbook of Cognition and Emotion*. Chichester: Wiley & Sons.

Teasdale, J. D. & Barnard, P. J. (1993). *Affect, cognition and change: Remodelling depressive thought.* Erbaum: Hove.

Thomas, J. & Bentall, R. P. (2002). Hypomanic traits and response styles to depression. *British Journal of Clinical Psychology, 41, p309-313.*

Thomas, J., Knowles, R., Tai, S. & Bentall, R. P. (2007). Response styles to depressed mood in bipolar affective disorder. *Journal of Affective Disorders, 100, (1)p249-252.*

Ventura, J., Liberman, R.P., Green, M. F., Shaner, A. & Mintz, J. (1998). Training and quality assurance of the structured clinical interview for DSM-IV (SCID-I/P). *Psychiatry Research, 79, (2), p163-173.*

.

Weissman, M. M., Bland, R. C., Canino, G. J., Faravelli, C., Greenwald, S., Hwu, H., Joyce, P. R., Karam, E. G., Lee, C., Lellouch, J., Lepine, J. P., Newman, S. C., Rubio-Stipec, M., Wells, J. E., Wickramaratne, P. J., Wittchen, H. U. & Yeh, E. K. (1996). Cross natural epidemiology of major depression and bipolar disorder. *Journal of the American Medical Association, 276, (4), p293-299.*

Wiebe, J. S. & Penley, J. A. (2005). A Psychometric Comparison of the Beck Depression Inventory-II in English and Spanish. *Psychological Assessment, 17 (4), p481-485.*

Williams, J. B. W., Gibbon, M., First, M. B., Spitzer, R. L., Davies, M., Borus, J, Howes, M. J., Kane, J., Pope Jr., H. G., Rounsaville, B., Wittchen, H. U. (1992). The structured clinical interview for DSM-III-R (SCID): II. Multi-site test-retest reliability. *Archives of General Psychiatry, 49, p630-636.*

Winocur, G., Coryell, W., Akiskal, H. S., Endicott, J., Keller, M. & Mueller, T. (1994). Manic-depressive (bipolar) disorder : the course in light of a prospective ten-year follow-up of 131 patients. *Acta Psychiatrica Scandinavica, 89, p 102–110.*

Wittchen, H. U., Muhlig, S. & Pezawas, L. (2003). Natural course and burden of bipolar disorders. *International Journal of Neuropsychopharmacology, 6, p145–154.*

World Health Organisation (WHO). (1992) *The ICD-10 classification of mental and behavioural disorders. Clinical descriptions and diagnostic guidelines.* Geneva: WHO.

World Health Organisation (WHO). (2005). *Mental Health: Facing the challenges, building solutions. Report from the WHO European Ministerial Conference.* WHO.

Wright, K., & Lam, D. (2004). Bipolar affective disorder: Current perspectives on psychological theory and treatment. In (Ed.) M. J. Power. *Mood disorders: A handbook of science and practice.* Chichester: Wiley & Sons.

Zanarini, M. C., Skodol, A. E., Bender, D., Dolan, R., Sanislow, C., Schaefer, E., Morey, L. C., Grilo, C. M., Shea, M. T., McGlashan, T. H. & Gunderson, J. G. (2000). The collaborative longitudinal personality disorders study: Reliability of axis I and II diagnoses**.** *Journal of Personality Disorders, 14, (4), p291-299.*

Zaretsky, A. E., Segal, Z. V., & Gemar, M. (1999). Cognitive therapy for bipolar depression: A pilot study. *Canadian Journal of Psychiatry, 44, p491– 494.*

Appendix 1. DSM-IV Criteria for a major depressed episode.

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

Note: Do not include symptoms that are clearly due to a general medical condition, or mood-incongruent delusions or hallucinations.

(1) depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad or empty) or observation made by others (e.g., appears tearful). Note: In children and adolescents, can be irritable mood.

(2) markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation made by others)

(3) significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. Note: In children, consider failure to make expected weight gains.

(4) insomnia or hypersomnia nearly every day

(5) psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down)

(6) fatigue or loss of energy nearly every day

(7) feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick)

(8) diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others)

(9) recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide

B. The symptoms do not meet criteria for a Mixed Episode.

C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).

E. The symptoms are not better accounted for by Bereavement, Le., after the loss of a loved one, the symptoms persist for longer than 2 months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.

Appendix 2. DSM-V Criteria for a manic episode.

A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood, lasting at least 1 week (or any duration if hospitalisation is necessary).

B. During the period of mood disturbance, three (or more) of the following symptoms have persisted (four if the mood is only irritable) and have been present to a significant degree:

(1) inflated self-esteem or grandiosity

(2) decreased need for sleep (e.g., feels rested after only 3 hours of sleep)

(3) more talkative than usual or pressure to keep talking

(4) flight of ideas or subjective experience that thoughts are racing

(5) distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli)

(6) increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation

(7) excessive involvement in pleasurable activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments)

C. The symptoms do not meet criteria for a Mixed Episode.

D. The mood disturbance is sufficiently severe to cause marked impairment in occupational functioning or in usual social activities or relationships with others, or to necessitate hospitalisation to prevent harm to self or others, or there are psychotic features.

E. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication, or other treatment) or a general medical condition (e.g., hyperthyroidism).

Note: Manic-like episodes that are clearly caused by somatic antidepressant treatment (e.g., medication, electroconvulsive therapy, light therapy) should not count toward a diagnosis of Bipolar I Disorder.

Appendix 3. DSM-IV Criteria for a hypomanic episode.

A. A distinct period of persistently elevated, expansive, or irritable mood, lasting throughout at least 4 days, that is clearly different from the usual nondepressed mood.

B. During the period of mood disturbance, three (or more) of the following symptoms have persisted (four if the mood is only irritable) and have been present to a significant degree:

(1) inflated self-esteem or grandiosity

(2) decreased need for sleep (e.g., feels rested after only 3 hours of sleep)

(3) more talkative than usual or pressure to keep talking

(4) flight of ideas or subjective experience that thoughts are racing

(5) distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli)

(6) increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation

(7) excessive involvement in pleasurable activities that have a high potential for painful consequences (e.g., the person engages in unrestrained buying sprees, sexual indiscretions, or foolish business investments)

C. The episode is associated with an unequivocal change in functioning that is uncharacteristic of the person when not symptomatic.

D. The disturbance in mood and the change in functioning are observable by others.

E. The episode is not severe enough to cause marked impairment in social or occupational functioning, or to necessitate hospitalisation, and there are no psychotic features.

F. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication, or other treatment) or a general medical condition (e.g., hyperthyroidism).

Note: Hypomanic-like episodes that are clearly caused by somatic antidepressant treatment (e.g., medication, electroconvulsive therapy, light therapy) should not count toward a diagnosis of Bipolar II Disorder.

Appendix 4. DSM-IV Criteria for a mixed episode.

A. The criteria are met both for a Manic Episode and for a Major Depressive Episode (except for duration) nearly every day during at least a 1-week period.

B. The mood disturbance is sufficiently severe to cause marked impairment in occupational functioning or in usual social activities or relationships with others, or to necessitate hospitalisation to prevent harm to self or others, or there are psychotic features.

C. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication, or other treatment) or a general medical condition (e.g., hyperthyroidism).

Note: Mixed-like episodes that are clearly caused by somatic antidepressant treatment (e.g., medication, electroconvulsive therapy, light therapy) should not count toward a diagnosis of Bipolar I Disorder.

Appendix 5.1. Letter of invitation: Clinical groups

Dear Participant,

What basic emotions are experienced in bipolar disorder and how are they regulated? A comparison between unipolar and bipolar disorder.

The Clinical Psychology department at the Alloway Centre in Dundee, in conjunction with the Doctoral training course in Clinical Psychology at the University of Edinburgh, are interested in gathering information about how a diagnosis of bipolar disorder or unipolar depression affects people's emotions and the ways in which such individuals cope with these emotions. It is hoped that this information will provide clinicians and researchers with a greater understanding of the nature of these conditions and how they affect individuals, as well as informing them about treatment options. As part of this project, you are invited to tell us about your views and experiences of bipolar or unipolar depression by taking part in an interview and completing some questionnaires. The information you provide will be important and valuable in helping us to gain a better understanding of these conditions and the impact they have on patients.

Please read the enclosed information sheet before deciding whether or not you wish to take part in the study. If you wish to take part, an appointment will be made for you to have a short discussion with myself before completing some questionnaires. This will take no longer than one hour. Participation is entirely voluntary and you are free to withdraw from the study at any time without the need for explanation. Your responses will be treated as confidential and you will remain completely anonymous, although we do ask for some personal details however, those will be separated from your answers on the questionnaires.

Thank you for taking the time to consider this invitation. We value your contribution to this research, the more people who take part, the more meaningful the results will be. If you would like to be involved in this study, I would be grateful if you could sign the consent form and return it to me in the stamped, addressed envelope provided. If you have any questions about this study please feel free to contact me on the details provided at the top of this letter.

Yours Sincerely,

Louise Carolan
Trainee Clinical Psychologist

Appendix 5.2. Letter of invitation: Control group

Dear Participant,

What basic emotions are experienced in bipolar disorder and how are they regulated? A comparison between unipolar and bipolar disorder.

The Clinical Psychology department at the Alloway Centre in Dundee, in conjunction with the Doctoral training course in Clinical Psychology at the University of Edinburgh, are interested in gathering information about how a diagnosis of bipolar disorder or unipolar depression affects people's emotions and the ways in which such individuals cope with these emotions. It is hoped that this information will provide clinicians and researchers with a greater understanding of the nature of these conditions and how they affect individuals, as well as informing them about treatment options. As a healthy individual, you are being invited to tell us about your experiences and emotions by completing some questionnaires. The information you provide will be important and valuable in helping us to compare the responses and to gain a better understanding of these conditions and the impact they have on patients.

Please read the enclosed information sheet before deciding whether or not you wish to take part in the study. If you wish to take part, please sign the consent form and fill out the enclosed questionnaires making sure that you answer all of the questions in order. This will take no longer than fifteen minutes. Participation is entirely voluntary and you are free to withdraw from the study at any time without the need for explanation. Your responses will be treated as confidential and you will remain completely anonymous, although we do ask for some personal details however, those will be separated from your answers on the questionnaires.

Thank you for taking the time to consider this invitation. We value your contribution to this research, the more people who take part, the more meaningful the results will be. If you would like to be involved in this study, I would be grateful if you could sign the consent form and return it with the completed questionnaires to me in the stamped, addressed envelope provided. If you have any questions about this study please feel free to contact me on the details provided at the top of this letter.

Yours Sincerely,

Louise Carolan
Trainee Clinical Psychologist

Appendix 6.1. Information sheet: Bipolar group

What basic emotions are experienced in bipolar disorder and how are they regulated? A comparison between unipolar and bipolar depression.

Participants are being recruited to take part in an investigation comparing the impact that bipolar and unipolar depression have on emotions and the ways in which these emotions are dealt with. Before taking part in the study it is important that you understand why the research is taking place and what is involved. Please take the time to read this information sheet carefully and feel free to discuss it with family, friends carers and/or the researcher. If anything is unclear or you have any questions at all about the study, please feel free to ask the researcher.

What is the purpose of this study?

This study aims to develop a greater understanding about the emotions people with bipolar and unipolar depression commonly experience and how they deal with them. This information is important because relatively little is known about the emotions experienced in bipolar disorder in comparison to other mental illnesses. The information provided by participants will be used to help services and clinicians develop their knowledge in this area and to provide information as to possibilities for treatment.

What will happen if I take part/ what will I have to do?

The researcher will contact you in one week to find out if you would like to participate. If you decide to take part, you will be asked to meet the researcher to discuss the study in more detail, to undertake a short interview and complete some questionnaires in order to determine if you are suitable to take part. If you are, you will be asked to complete some more questionnaires. If not, then you will not be able to participate. Participants meeting the criteria will be assigned to one of three groups (the bipolar disorder group, the unipolar depression group or the control group) and the results will be compared.

What are the possible benefits of taking part?

By taking part, you will be contributing to the development of knowledge and a greater understanding about the nature of bipolar disorder and unipolar depression, and the impact that they have on individuals. Gathering information from patients themselves is important because it provides richer results and allows you to become involved in research and the development of services. Furthermore as our knowledge about bipolar disorder increases,

more information can be sought as to treatment options to ensure that the needs of this population are met.

What are the possible disadvantages and risks of taking part?

It is unlikely that taking part in this study involves any risk to participants because taking part in the study involves talking about the feelings you commonly experience and how you cope with them. However, difficult feelings can be hard to face. If you feel distressed at any point as a result of this study the researcher would recommend that you seek advice and treatment from your GP. If you are currently receiving treatment from a psychiatrist or mental health professional then you would be advised to contact them to discuss your concerns. If you have any questions about this information please feel free to contact the researcher, who would be happy to discuss these with you, on the details provided at the bottom of this form.

Should I take part?

Participation in this study is entirely voluntary. It is your choice to decide whether you wish or do not wish to take part. If you do decide to take part, you will be given this information sheet to keep and be asked to sign a consent form, a signed copy of which you will receive. You are also free to withdraw at any time without giving a reason. Your future planned treatment will not be affected.

What if something goes wrong?

As the study will involve thinking and talking about your bipolar disorder, there is very little that can go wrong. However, the researcher is ethically and legally obliged to tell you that there are no special compensation arrangements. If you are harmed due to someone's negligence, then you may have grounds for legal action but you may have to pay your legal costs. Regardless of this, if you wish to complain about any aspect of the way you have been approached or treated during the course of the study, you can do so with the following details; *Complaints and Care Manager, Complaints and Advice Team, Level 7, Ninewells Hospital, Dundee, DD1 9SY. Freephone: 0800 027 5507. Email: nhstaysidecomplaints@thb.scot.nhs.uk.*

Will my taking part in the study be kept confidential?

Yes all data collected during this study will be kept strictly confidential and will be annonymised. Identifying information on the questionnaires will be removed and data will be held within a secure office in a locked filing cabinet data. Access to the data will be restricted. At times the content of the questionnaires may be shared with the researcher's supervisor however this information will remain anonymous. Other parties may be informed if further information emerges that raises serious concerns about your health and well-

being or the safety of another person. However, in this event the researcher will discuss this with you. Authorised individuals from NHS Tayside may also review the data in order to ensure that the study is being carried out correctly. Otherwise, your name will not be disclosed.

What will happen to the results of the research study?

The results will be included in a Doctoral thesis for fulfilment of the Doctorate in Clinical Psychology. It is also anticipated that the results will be presented at conferences and to relevant staff groups, as well as submission to an academic journal. *However, the results will remain anonymous and all information remains confidential.* The researcher would be happy to give you verbal and written feedback regarding individual response to the treatment.

Who is organising the research?

I am a Trainee Clinical Psychologist at the University of Edinburgh/East of Scotland Doctorate in Clinical Psychology Training Course. I am an experienced clinician who has worked in the field of mental health for eight years. I am interested in the way bipolar disorders affects emotions and the ways in which individuals with bipolar disorder deal with these emotions.

Who has reviewed the study?

This study has been subject to review by a Research Ethics Committee in Scotland. The study will also be reviewed on a regular basis by supervisors with the Clinical and Health Psychology Department at the University of Edinburgh.

Contact for further information:

Please feel free to contact myself at any time should you have any further questions on the details provided on the covering letter.

Thank you for taking time to read this information leaflet.

Appendix 6.2. Information sheet: Unipolar group

What basic emotions are experienced in bipolar disorder and how are they regulated? A comparison between unipolar and bipolar depression.

Participants are being recruited to take part in an investigation comparing the impact that bipolar and unipolar depression have on emotions and the ways in which these emotions are dealt with. Before taking part in the study it is important that you understand why the research is taking place and what is involved. Please take the time to read this information sheet carefully and feel free to discuss it with family, friends carers and/or the researcher. If anything is unclear or you have any questions at all about the study, please feel free to ask the researcher.

What is the purpose of this study?

This study aims to develop a greater understanding about the emotions people with bipolar and unipolar depression commonly experience and how they deal with them. This information is important because relatively little is known about the emotions experienced in bipolar disorder in comparison to other mental illnesses. The information provided by participants will be used to help services and clinicians to develop their knowledge in this area and to provide information as to possibilities for treatment.

What will happen if I take part/ what will I have to do?

The researcher will contact you in one week to find out if you would like to participate. If you decide to take part, you will be asked to meet the researcher to discuss the study in more detail, to undertake a short interview and complete some questionnaires in order to determine if you are suitable to take part. If you are, you will be asked to complete some more questionnaires. If not, then you will not be able to participate. Participants meeting the criteria for the study will be assigned to one of three groups (the bipolar disorder group, the unipolar depression group or the control group) and the results will be compared.

What are the possible benefits of taking part?

By taking part, you will be contributing to the development of knowledge and a greater understanding about the nature of bipolar disorder and unipolar depression, and the impact that they have on individuals. Gathering information from patients themselves is important because it provides richer results and allows you to become involved in research and the development of services. Furthermore as our knowledge about bipolar disorder increases,

more information can be sought as to treatment options to ensure that the needs of this population are met.

What are the possible disadvantages and risks of taking part?

It is unlikely that taking part in this study involves any risk to participants because taking part in the study involves talking about the feelings you commonly experience and how you cope with them. However, difficult feelings can be hard to face. If you feel distressed at any point as a result of this study the researcher would recommend that you seek advice and treatment from your GP. If you are currently receiving treatment from a psychiatrist or mental health professional then you would be advised to contact them to discuss your concerns. If you have any questions about this information please feel free to contact the researcher, who would be happy to discuss these with you, on the details provided at the bottom of this form.

Should I take part?

Participation in this study is entirely voluntary. It is your choice to decide whether you wish or do not wish to take part. If you do decide to take part, you will be given this information sheet to keep and be asked to sign a consent form, a signed copy of which you will receive. You are also free to withdraw at any time without giving a reason. Your future planned treatment will not be affected.

What if something goes wrong?

As the study will involve thinking and talking about your bipolar or unipolar depression, there is very little that can go wrong. However, the researcher is ethically and legally obliged to tell you that there are no special compensation arrangements. If you are harmed due to someone's negligence, then you may have grounds for legal action but you may have to pay your legal costs. Regardless of this, if you wish to complain about any aspect of the way you have been approached or treated during the course of the study, you can do so with the following details; *Complaints and Care Manager, Complaints and Advice Team, Level 7, Ninewells Hospital, Dundee, DD1 9SY. Freephone: 0800 027 5507. Email: nhstaysidecomplaints@thb.scot.nhs.uk.*

Will my taking part in the study be kept confidential?

Yes all data collected during this study will be kept strictly confidential and will be annonymised. Identifying information on the questionnaires will be removed and data will be held within a secure office in a locked filing cabinet data. Access to the data will be restricted. At times the content of the questionnaires may be shared with the researcher's supervisor however this information will remain anonymous. Other parties may be informed if further information emerges that raises serious concerns about your health and well-being or the safety of another person. However, in this event the researcher

will discuss this with you. Authorised individuals from NHS Tayside may also review the data in order to ensure that the study is being carried out correctly. Otherwise, your name will not be disclosed.

What will happen to the results of the research study?

The results will be included in a Doctoral thesis for fulfilment of the Doctorate in Clinical Psychology. It is also anticipated that the results will be presented at conferences and to relevant staff groups, as well as submission to an academic journal. *However, the results will remain anonymous and all information remains confidential.* The researcher would be happy to give you verbal and written feedback regarding individual response to the treatment.

Who is organising the research?

I am a Trainee Clinical Psychologist at the University of Edinburgh/East of Scotland Doctorate in Clinical Psychology Training Course. I am an experienced clinician who has worked in the field of mental health for eight years. I am interested in the way bipolar disorders affects emotions and the ways in which individuals with bipolar disorder deal with these emotions.

Who has reviewed the study?

This study has been subject to review by a Research Ethics Committee in Scotland. The study will also be reviewed on a regular basis by supervisors with the Clinical and Health Psychology Department at the University of Edinburgh.

Contact for further information:

Please feel free to contact myself at any time should you have any further questions on the details provided on the covering letter.

Thank you for taking time to read this information leaflet.

Appendix 6.3. Information sheet: Control group

What basic emotions are experienced in bipolar disorder and how are they regulated? A comparison between unipolar and bipolar depression.

You are being recruited as a healthy individual, to take part in an investigation comparing the impact that bipolar and unipolar depression have on emotions and the ways in which these emotions are dealt with. Before taking part in the study it is important that you understand why the research is taking place and what is involved. Please take the time to read this information sheet carefully and feel free to discuss it with family, friends carers and/or the researcher. If anything is unclear or you have any questions at all about the study, please feel free to ask the researcher.

What is the purpose of this study?

This study aims to develop a greater understanding about the emotions people with bipolar and unipolar depression commonly experience and how they deal with them. It is important to involve healthy individuals in this study so that the results from individuals with bipolar and unipolar depression can be compared. Relatively little is known about the emotions experienced in bipolar disorder in comparison to other mental illnesses or healthy individuals so the information provided by you will be used to help services and clinicians to develop their knowledge in this area and to provide information as to possibilities for treatment.

What will happen if I take part/ what will I have to do?

If you decide to take part, you are asked to sign the consent form and complete the questionnaires enclosed and return them to me in the stamped, addressed envelope provided. *This should take no longer than fifteen minutes.*

What are the possible benefits of taking part?

By taking part, you will be contributing to the development of knowledge and a greater understanding about the nature of bipolar disorder and unipolar depression, and the impact that they have on individuals. Gathering information from healthy individuals is important because it enables results from clinical groups to be compared providing richer results. Furthermore, it enables you to become involved in research and the development of mental health services. As our knowledge about bipolar disorder increases, more information can be sought as to treatment options to ensure that the needs of this population are met.

What are the possible disadvantages and risks of taking part?

It is unlikely that taking part in this study involves any risk to participants because it involves filling out questionnaires about your feelings and how you cope with them. However, if you feel distressed at any point as a result of this study the researcher would recommend that you seek advice and treatment from your GP. If you have any questions about this study or the information provided please feel free to contact the researcher, who would be happy to discuss these with you, on the details provided at the bottom of this form.

Should I take part?

Participation in this study is entirely voluntary. It is your choice to decide whether you wish or do not wish to take part. You are free to withdraw at any time without giving a reason. Your future planned treatment will not be affected.

What if something goes wrong?

As this study involves filling out questionnaires, there is very little that can go wrong. However, the researcher is ethically and legally obliged to tell you that there are no special compensation arrangements. If you are harmed due to someone's negligence, then you may have grounds for legal action but you may have to pay your legal costs. Regardless of this, if you wish to complain about any aspect of the way you have been approached or treated during the course of the study, you can do so with the following details; *Complaints and Care Manager, Complaints and Advice Team, Level 7, Ninewells Hospital, Dundee, DD1 9SY. Freephone: 0800 027 5507. Email: nhstaysidecomplaints@thb.scot.nhs.uk.*

Will my taking part in the study be kept confidential?

Yes. All data collected during this study will be kept strictly confidential and will be anonymised. Identifying information on the questionnaires will be removed and data will be held within a secure office in a locked filing cabinet data. Access to the data will be restricted. At times the content of the questionnaires may be shared with the researcher's supervisor however this information will remain anonymous. Other parties may be informed if further information emerges that raises serious concerns about your health and well-being or the safety of another person. However, in this event the researcher will discuss this with you. Authorised individuals from NHS Tayside may also review the data in order to ensure that the study is being carried out correctly. Otherwise, your name will not be disclosed.

What will happen to the results of the research study?

The results will be included in a Doctoral thesis for fulfilment of the Doctorate in Clinical Psychology. It is also anticipated that the results will be presented

at conferences and to relevant staff groups, as well as submission to an academic journal. *However, the results will remain anonymous and all information remains confidential.* The researcher would be happy to give you verbal and written feedback regarding individual response to the treatment.

Who is organising the research?

I am a Trainee Clinical Psychologist at the University of Edinburgh/East of Scotland Doctorate in Clinical Psychology Training Course. I am an experienced clinician who has worked in the field of mental health for eight years. I am interested in the way bipolar disorders affects emotions and the ways in which individuals with bipolar disorder deal with these emotions.

Who has reviewed the study?

This study has been subject to review by a Research Ethics Committee in Scotland. The study will also be reviewed on a regular basis by supervisors with the Clinical and Health Psychology Department at the University of Edinburgh.

Contact for further information:

Please feel free to contact myself at any time should you have any further questions on the details provided on the covering letter.

Thank you for taking time to read this information leaflet.

Appendix 7. Consent form

What basic emotions are experienced in bipolar disorder and how are they regulated? A comparison between unipolar and bipolar disorder.

Consent Form

Please tick (√) the box.

I have read and understand the information sheet.	☐
I have had the opportunity to consider this information, ask questions about it and these questions were answered satisfactorily.	☐
I feel I now have enough information about the study.	☐
I understand that my participation in the above study is voluntary and that I am free to withdraw at any time without giving any reason and without my medical or legal rights being affected.	☐
I understand that data collected during this study may be looked at by the researcher, her supervisor and authorised individuals from NHS Tayside where it is necessary and I give permission for these individuals to have access to this data.	☐
I agree to take part in the above study.	☐

Participant's signature (printed)	Date	Participant's name
Researcher's signature (printed)	Date	Researcher's name

Appendix 8. Demographics sheet

Age _____ Gender (Please circle) m / f

Marital status (please circle) Single / Married / Divorced / Widowed.

Other (please state) _____

Occupation (if unemployed, what did you do previously?) _____

Number of years in education (including school, HNDs/HNCs, degrees etc) _____

1) Are you currently suffering from a mental health condition (e.g. depression or anxiety)?

Yes ☐ Go to question 1a. **No** ☐ Go to question 2.

1a) If yes, please state what mental health condition(s) you have and how long you have been diagnosed. _____

_____

2) Have you, at any point in the past, suffered from a mental health condition?

Yes ☐ Go to question 2a. **No** ☐ Go to question 3.

2a) If yes, please state what mental health condition(s) you had and how long ago had it. _____

_____

3) Have you ever been hospitalised as a result of a mental health condition?

Yes ☐ Go to question 3a. **No** ☐

3a) Please state how many times and why you were hospitalised _____

Appendix 9. Structured Clinical Interview for DSM-IV (SCID)

Appendix 10.1. Basic Emotions Scale – General Version (BES)

Appendix 10.2. Basic Emotions Scale – Manic Version (BES)

Appendix 10.3. Basic Emotions Scale – Depressed Version (BES)

Appendix 11.1. Regulation of Emotion Questionnaire – Negative (REQ)

Appendix 11.2. Regulation of Emotion Questionnaire – Positive (REQ)

Appendix 12. Beck Depression Inventory (BDI-II)

Appendix 13. State-Trait Anxiety Inventory (STAI)

Appendix 14. The Bech-Rafaelsen Mania Scale (MAS)

Appendix 15.1. Means and standard deviations for the BES data containing outliers before and after these were removed.

BES version and subscale	Group					
	Bipolar		***Unipolar***		***Control***	
	Mean/SD Before	***Mean/SD After***	***Mean/SD Before***	***Mean/SD After***	***Mean/SD Before***	***Mean/SD After***
BES_General Happiness	20.79 SD 3.76	21.40 SD 2.91	N/A	N/A	21.81 SD 4.93	22.85 SD 2.85
BES_General Sadness	11.35 SD 4.92	10.93 SD 4.36	N/A	N/A	N/A	N/A
BES_Depressed Fear	23.42 SD 4.45	23.81 SD 3.92	N/A	N/A	N/A	N/A
BES_Depressed Disgust	27.21 SD 7.47	28.48 SD 5.62	N/A	N/A	N/A	N/A
BES_Depressed Sadness	23.84 SD 4.84	24.77 SD 3.22	N/A	N/A	N/A	N/A
BES_Depressed Happiness	N/A	N/A	7.00 SD 3.87	6.14 SD 2.07	N/A	N/A
BES_Manic Happiness	22.82 SD 6.06	23.77 SD 4.29	N/A	N/A	N/A	N/A
BES_Manic Disgust	10.79 SD 6.55	10.31 SD 5.47	N/A	N/A	N/A	N/A

Appendix 15.2. Means and standard deviations for the REQ negative data containing outliers before and after these were removed.

REQ Negative	Group					
	Bipolar		Unipolar		Control	
	Mean/SD Before	*Mean/SD After*	*Mean/SD Before*	*Mean/SD After*	*Mean/SD Before*	*Mean/SD After*
External dysfunctional	N/A	N/A	2.20 SD 2.56	1.08 SD 0.90	1.33 SD 1.04	1.41 SD 0.90
External functional	N/A	N/A	6.80 SD 4.42	5.38 SD 2.25	N/A	N/A
Internal dysfunctional	N/A	N/A	11.13 SD 4.68	10.50 SD 4.14	N/A	N/A
Internal functional	6.35 SD 3.25	6.51 SD 2.11	N/A	N/A	N/A	N/A

Appendix 15.3. Means and standard deviations for the REQ positive data containing outliers before and after these were removed.

REQ Positive	Group					
	Bipolar		***Unipolar***		***Control***	
	Mean/SD Before	***Mean/SD After***	***Mean/SD Before***	***Mean/SD After***	***Mean/SD Before***	***Mean/SD After***
External dysfunctional	1.19 SD 3.35	1.09 SD 1.27	0.73 SD 1.16	0.50 SD 0.75	0.46 SD 0.83	0.23 SD 0.59
Internal dysfunctional	N/A	N/A	5.00 SD 3.90	4.28 SD 2.86	N/A	N/A
Internal functional	N/A	N/A	10.00 SD 3.54	9.35 SD 2.61	N/A	N/A

Appendix 16. Means and standard deviations for the BES fear data before and after ln transformation.

BES fear subscale	Bipolar (n=34)		Unipolar (n=15)		Control (n=15)	
	Mean/SD Pre	***Mean/SD Post***	***Mean/SD Pre***	***Mean/SD Post***	***Mean/SD Pre***	***Mean/SD Post***
BES General	16.52 SD 4.96	2.75 SD 0.29	15.40 SD 5.53	2.66 SD 0.39	11.60 SD 4.35	2.37 SD 0.42
BES Depressed	23.81* SD 3.92	3.13 * SD 0.18	23.53 SD 5.13	3.13 SD 0.24	N/A	N/A
BES Manic	14.67 SD 7.31	2.57 SD 0.56	N/A	N/A	N/A	N/A

* n=32 in bipolar group BES Depressed due to the removal of an outlier and one participant who experienced mania only

Appendix 17. Mean and standard deviations of anger, happiness, fear, sadness and disgust for the bipolar group in general, manic and depressed states.

BES Emotion Subscale	Bipolar Group n=33					
	General Mean/SD	***Manic Mean/SD***	***Depressed Mean/SD***	***ANOVA (p)***	***Effect Size (n^2)***	***Power***
Anger	15.21 SD 4.17	16.67 SD 6.71	18.66 SD 4.87	0.04 S	0.09 Medium	0.61
Happiness *[1]	20.87 SD 3.90	23.77 SD 4.29	7.12 SD 2.95	<0.001 S	0.84 Large	1.00
Fear *[2] (transformed)	2.75 SD 0.30	2.59 SD 0.56	3.13 SD 0.18	<0.001 S	0.70 Large	1.00
Sadness	11.27 SD 4.98	8.18 SD 4.97	23.84 SD 4.84	<0.001 S	0.89 Large	1.00
Disgust *[2]	15.62 SD 5.27	10.31 SD 5.47	27.21 SD 7.57	<0.001 S	0.69 Large	1.00

*[1] n=31 for happiness due to the removal of 2 outliers
*[2] n=32 for disgust due to the removal of 1 outlier

Appendix 18. Means and standard deviations of anger, happiness, fear, sadness and disgust for bipolar and unipolar groups in depressed states.

BES Depressed version emotion subscale	Bipolar (n=33) Mean/SD	Unipolar (n=15) Mean/SD	ANOVA (p)	Effect Size (n^2)	Power
Anger	18.66 SD 4.87	18.80 SD 6.83	0.93 NS	0.00 Small	0.05
Happiness* [2]	7.12 SD 2.95	6.14 SD 2.07	0.26 NS	0.02 Small	0.196
Fear (transformed)* [3]	3.15 SD 0.18	3.13 SD 0.24	0.87 NS	0.003 Small	0.06
Sadness* [1]	24.77 SD 3.22	24.93 SD 2.93	0.71 NS	0.01 Small	0.05
Disgust* [1]	28.48 SD 5.62	20.40 SD 9.19	0.005 S	0.237 Large	0.95

*[1] n=31 in the bipolar group due to the removal of an outlier
*[2] n=33 in the bipolar group due to the removal of an outlier
*[3] n=32 in the bipolar group due to the removal of an outlier

Appendix 19. Means and standard deviations of anger, happiness, fear, sadness and disgust for the bipolar, unipolar and control group in general states.

BES Emotion Subscale	Bipolar group n=34, Unipolar group n=15, Control group n=15					
	Bipolar Mean/SD	***Unipolar Mean/SD***	***Control Mean/SD***	***ANOVA (p)***	***Effect Size (n^2)***	***Power***
Anger	15.11 SD 4.14	12.06 SD 4.38	12.06 SD 3.48	0.02 S	0.11 Medium	0.61
Happiness *[1]	21.40 SD 2.91	19.93 SD 4.90	22.87 SD 2.85	0.08 NS	0.08 Small	0.48
Fear *[2] (transformed)	2.76 SD 0.29	2.66 SD 0.39	2.37 SD 0.42	0.003 S	0.16 Large	0.30
Sadness	10.93 SD 4.36	10.53 SD 4.45	10.66 SD 6.34	0.96 NS	0.001 Small	0.05
Disgust *[2]	15.88 SD 5.31	11.53 SD 6.73	10.60 SD 4.61	0.004 S	0.16 Large	0.87

*[1] n=32 in the bipolar group and 14 in the control group due to the removal of outliers
*[2] n=33 in the bipolar group due to the removal of outliers

Appendix 20. Means and standard deviations for the frequency with which external and internal, dysfunctional and functional coping strategies are used for regulating negative emotion by the bipolar, unipolar and control groups.

Coping strategy for negative emotion	Bipolar group n=34, Unipolar group n=15, Control group n=15					
	Bipolar Mean/SD	***Unipolar Mean/SD***	***Control Mean/SD***	***ANOVA (p)***	***Effect Size (n^2)***	***Power***
Internal dysfunctional *[1]	11.02 SD 4.24	10.50 SD 4.14	2.86 SD 1.72	<0.001 S	0.46 Large	1.00
Internal functional *[2]	6.51 SD 2.11	7.26 SD 5.03	8.40 SD 3.52	0.18 NS	0.05 Small	0.30
External dysfunctional *[3]	4.91 SD 4.62	1.08 SD 0.90	1.14 SD 0.90	<0.001 NS	0.20 Small	0.92
External functional *[4]	7.70 SD 4.34	5.38 SD 2.25	9.80 SD 5.15	0.02 S	0.11 Large	0.66

*[1] n=14 in the unipolar group due to the removal of an outlier
*2 n=29 in the bipolar group due to the removal of 5 outliers
*3 n=12 in the unipolar and control group due to the removal of 3 outliers in these groups
*4 n=13 in the unipolar group due to the removal of 2 outliers in this group

Appendix 21. Means and standard deviations for the frequency with which external and internal, dysfunctional and functional coping strategies are used for regulating positive emotion by the bipolar, unipolar and control groups.

Coping strategy for positive emotion	Bipolar group n=34, Unipolar group n=15, Control group n=15					
	Bipolar Mean/SD	*Unipolar Mean/SD*	*Control Mean/SD*	*ANOVA (p)*	*Effect Size (n^2)*	*Power*
Internal dysfunctional *[1]	4.82 SD 2.93	4.28 SD 2.86	3.20 SD 2.56	0.19 NS	0.05 Small	1.34
Internal functional *[2]	9.64 SD 3.44	9.35 SD 2.61	7.73 SD 4.57	0.22 NS	0.04 Small	0.31
External dysfunctional *[3]	1.09 SD 1.27	0.50 SD 0.75	0.23 SD 0.59	0.03 S	0.11 Large	0.65
External functional *[4]	12.94 SD 4.36	12.86 SD 3.75	10.00 SD 5.11	0.09 NS	0.07 Small	0.48

*1 n=14 in the unipolar group due to the removal of an outlier
*2 n=31 in the bipolar group, n=14 in the unipolar group and n=13 in the control group due to the removal of outliers

Appendix 22. Letter confirming ethical approval for the study.