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## PROFESSIONAL DOCTORATES

**Social cognition and the manic defence: attributions, selective attention and self-schema in bipolar affective disorder.**

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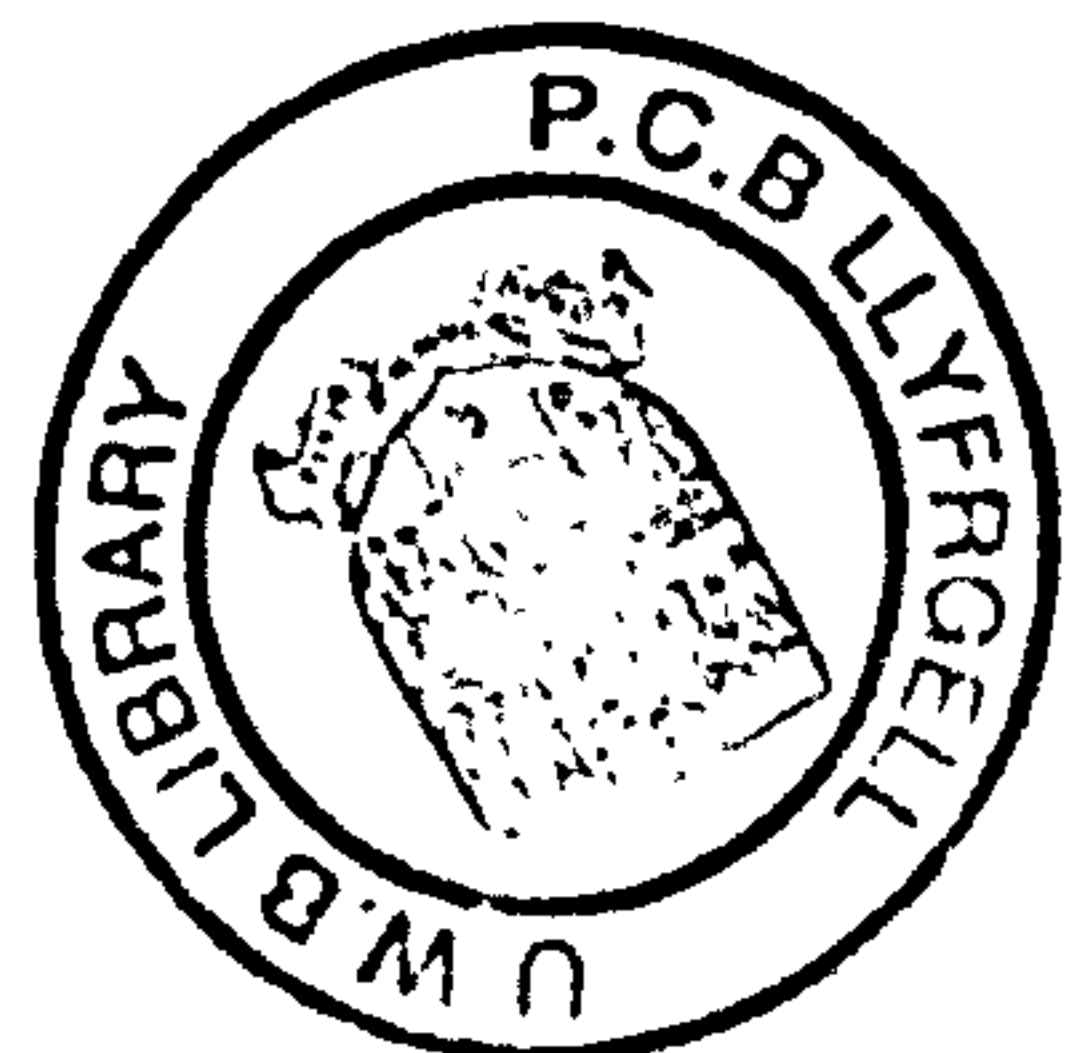
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**Social Cognition and the Manic Defence:  
Attributions, Selective Attention and Self-Schema  
in Bipolar Affective Disorder.**

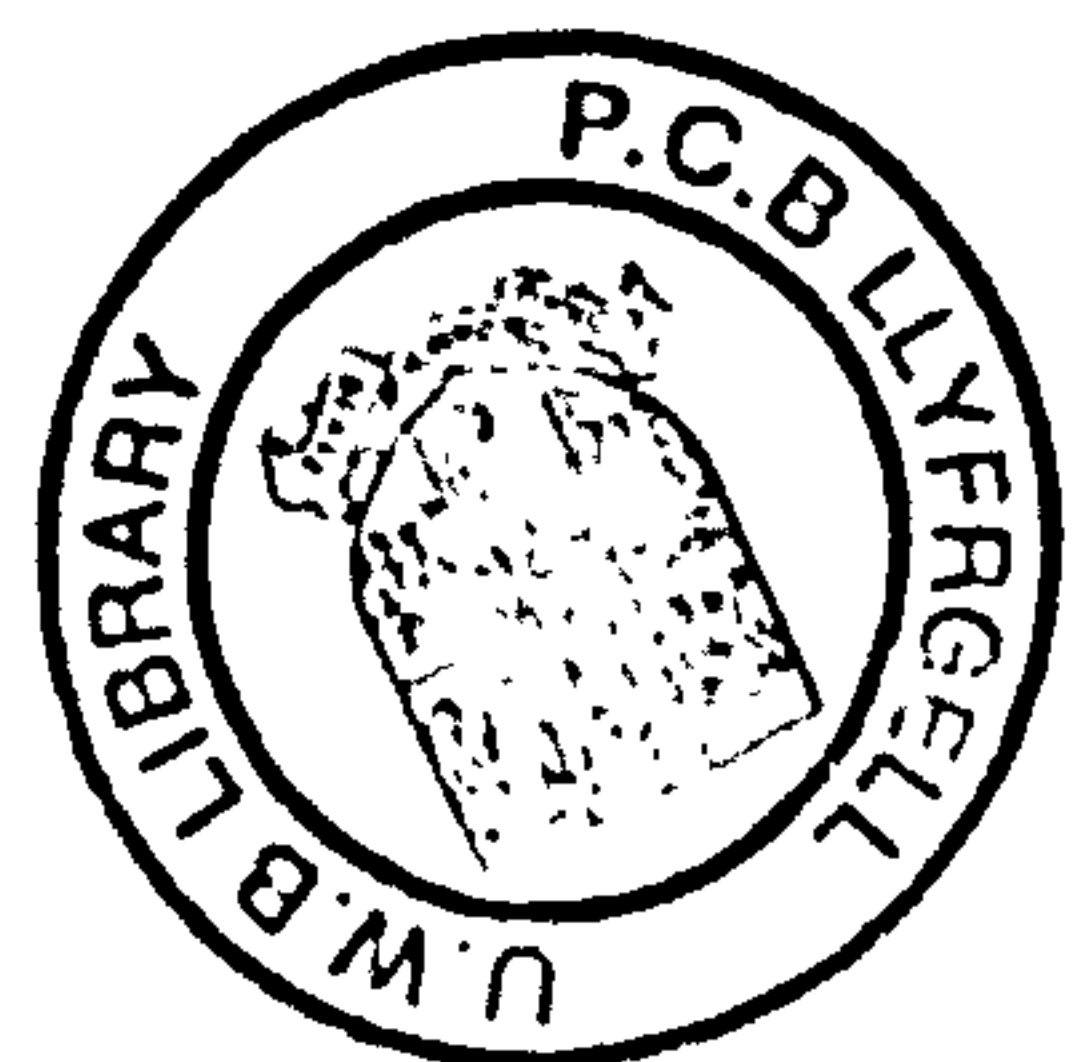
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**March 2000.**

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**SOCIAL COGNITION  
AND  
THE MANIC DEFENCE.**



## **Acknowledgements.**

Writing a thesis, I have discovered, is like building a dry stone wall. Having found stones of the desired shape or size, the challenge is to puzzle them together to resist assailing winds. In the absence of mortar, I am indebted to the following individuals and institutions:

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### Summary.

Psychological studies in bipolar affective disorder and analogue conditions suggest that mania may be the product of an abnormal defence against depression. In this study, currently manic bipolar individuals, currently depressed bipolar individuals, and normal controls were assessed using explicit and implicit measures of attributional style, an emotional Stroop test with euphoria-related and depression-related words and a recall measure of the self-schema. Manic individuals showed a normal self-serving bias on a version of the explicit attributional style questionnaire, attributing positive events more than negative events to self, in contrast to bipolar-depressed individuals who attributed negative events more than positive events to self. However, on an implicit test of attributional style, both manic and bipolar-depressed individuals attributed negative events more than positive events to self. Both bipolar-manic and bipolar-depressed individuals demonstrated slowed colour naming for depression-related but not euphoria-related words on an emotional Stroop test. Manic individuals, like normal controls, endorsed primarily positive words as true to self on a self referent questionnaire, but like bipolar-depressed individuals, recalled primarily negative words in a surprise recall test afterwards. Findings from the implicit tests therefore indicate a common form of psychological organisation in manic and depressed individuals, whereas the contrasts between the scores on the implicit and explicit measures are in accord with the hypothesis of a manic-defence. Future avenues for research and implications for treatment are discussed.

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## Preface.

**“Few activities engage our lives so profoundly as the defence and enhancement of the self”.**

**Morris Rosenberg, (1979, p. 260).**

The following thesis is organised into eleven chapters of which the first six are introductory. These set the scene for a study, which attempts (by way of defence-penetrating measures), to empirically test the psychoanalytical hypothesis that bipolar mania is an abnormal defence against, or reaction to depression or threats to self-representation (e.g. Freud, 1921; Rado, 1928). Such accounts suggest that the same psychic complexes drive both the manic and depressed episodes evidenced in bipolar disorder. These overwhelm the individual during depression but are treated with indifference during mania (Abraham, 1911 [1927]). Zigler and Glick in 1988, and Neale in the same year, proposed contemporary reformulations of this notion. Zigler and Glick argue that mania, like paranoia, is a “camouflaged depression”; a cognitive defence mechanism used by depressed individuals to deny inadequacies and unhappiness, and hence escape the psychic pain of dysphoric mood. Similarly, Neale believes that unstable self-esteem, coupled with unrealistic standards for success, can predispose an individual to bipolar disorder. Thus grandiose ideas, which serve to banish distressing cognitions from the consciousness, are precipitated by any intensification of low self-regard, and in turn lead to mood

elevation or mania. In a recent elaboration of this functional view of delusions, Bentall, Kinderman, and Kancy (1994) implicate self-concept discrepancies, suggesting that persecutory delusions are the product of attributional processes attempting to maintain a positive explicit self-concept. Such a “delusions-as-defence” model of persecutory delusions closely parallels the “manic-defence” model of grandiose ideation, examined in the present research.

The remainder of this preface is devoted to a brief outline of the structure of the following thesis, giving details of chapter aims and general content.

The first chapter reviews the current knowledge of bipolar affective disorder, its clinical manifestations, diagnostic classification, and epidemiology. The personal and socio-economic costs of this disorder are discussed, including estimates of self-harm and suicide risk relative to other psychopathologies. Treatment and research considerations are touched upon briefly. Attention is then drawn to the pattern and course of symptomatic episodes. Cycle length and putative precipitants of relapse are discussed in relation to the above theory of a manic-defence.

Chapter two then reviews various differences between symptomatic episodes in recurring unipolar depression and bipolar disorder, and challenges the prevailing conception of the unipolar-bipolar dichotomy. A spectrum or continuum model of affective disorders is proposed as an alternative to the current categorical classificatory system. Also emphasised is the spectrum of manic



states, which unlike the well-established depressive spectrum is often overlooked.

Chapter three addresses the notion of mania as a defence against depression or impoverished self-esteem, in more detail. Early psychoanalytic writings on this subject are discussed, their influence assessed, and modern revisions of these accounts are presented.

Chapter four reviews the ostensible diagnostic imprecision between bipolar disorder and other psychotic conditions, and a schizophrenic-affective continuum conception is outlined with reference to the manic defence. Two parallel models of defence, the delusions-as-defence and the manic-defence, are compared. The relative benefits of psychological research into individual symptom clusters (as opposed to syndrome orientated research) is briefly considered and the existence of unipolar mania and its potential challenge to the manic-defence argument is explored.

Chapter five reviews the limited number of bipolar studies that have employed psychological measures. It presents an explanation of attribution theory and considers investigations of overt and covert explanatory style and self-schema in the fields of unipolar depression, bipolar disorder, and persecutory delusions. This chapter also reviews the evidence for the putative defensive function of both mania and paranoia and the delusions-as-defence model is elaborated. The use of contingency judgement tasks to explore implicit attributional functioning in paranoid individuals is explained and the most commonly used overt test of attributional

style is introduced with reference to a bipolar population. Chapter five concludes with some broad research hypotheses.

Chapter six, the last of the introduction, is a short chapter. It explains the rationale of the present study in more detail, to include specific hypotheses.

Chapters seven and eight constitute the method and results section of this thesis. Chapter seven comprises the methodology of the current investigation, with details of participant selection, psychometric measures and procedure. Certain ethical and professional issues, pertaining to test procedures are also highlighted. Chapter eight reports the results and provides some preliminary interpretations of the data.

The final three chapters constitute the discussion. The first of these, chapter nine attempts a more detailed interpretation of the results obtained by bipolar participants in the depressed and manic episodes of the disorder. Certain limitations of the present investigation in terms of the questions it leaves unanswered are also noted. Parallels between bipolar mania and paranoia are revisited, as are the similarities between the delusions-as-defence and manic-defence notions. A qualification of the defence model of bipolar disorder is considered within the context of other prevailing models of delusions.

Chapter ten attempts to address certain methodological limitations of the present study.

Chapter eleven is the final chapter, and is concerned with the clinical application of the present results and those of related

studies. It opens with an account of maladaptive strategies putatively evoked to preserve self-representation in the face bipolar disorder. It then reviews the role of cognitive behaviour therapy and its relationship to both pharmacotherapy and a new form of adjunctive individual psychotherapy for those with bipolar disorder. Various obstacles to bipolar treatment are discussed and tentative modifications to the psychological management of bipolar patients are suggested. Some general conclusions are drawn and indications for future research are derived.

## INTRODUCTION.

### Chapter One.

#### Bipolar Affective Disorder.

Bipolar affective disorder was previously referred to as manic-depressive illness and is characterised by repeated episodes of depression and mania. It afflicts individuals from a wide diversity, if not all cultures and has been with us for thousands of years. Bipolar disorder can be recognised in the scriptures of the Old Testament and has fascinated medical observers ever since. In their comprehensive and compassionate text devoted to this condition, Frederick Goodwin and Kay Jamison describe bipolar disorder as the magnification (sometimes to grotesque proportions) of the normal human experiences of sadness and fatigue, joy and exuberance, sensuality and sexuality, irritability and rage, energy and creativity (Goodwin & Jamison, 1990).

Although many clinical accounts emphasise differences across the two apparently separate “poles” or mood states of bipolar disorder, these differences are perhaps artificially enhanced by the prevailing classificatory systems (e.g. the American Psychiatric Association's current diagnostic and statistical manual, DSM-IV, APA, 1994a). These attempt to describe discrete diagnostic entities or subtypes within larger entities. Such a categorical approach tends to neglect the multiple forms and degrees of severity of bipolar disorder, often missing subtle or sub-syndromal affective states. Goodwin and Jamison describe the characteristic “oscillation into,



out of and within the various forms and states of the disease”. They argue that the manic and depressive states do have a polar quality but suggest that the “overlapping, transitional, and fluctuating aspects are enormously important in describing and understanding the illness as a whole”, (Goodwin & Jamison, 1990, p. 17). They thus champion a spectrum model of bipolar and other affective disorders, as a more meaningful alternative to the prevailing classificatory system. This debate is discussed in detail in chapter two, along with a parallel discussion of the relative benefits of a psychosis-affective spectrum of psychopathology.

### Diagnostic Classification.

Bipolar disorder is a long-term, episodic and chronic illness with a variable course and poor prognosis. It is classified as a disorder of mood within DSM-IV (APA, 1994a), and by this definition, individuals with Bipolar I Disorder will have had at least one episode of mania. Some have had previous major depressive episodes, and most will have subsequent episodes that can be either manic or depressive. In addition, hypomanic and mixed episodes can occur, as well as significant sub-clinical mood lability between episodes (APA, 1994b). Individuals with Bipolar II Disorder will have had recurrent major depressive episodes but will not have met manic episode criteria. Diagnostic criteria for mood episodes included in bipolar diagnoses are listed next.



### Major Depressive Episodes.

A major depressive episode is characterised by symptoms of:

- 1) depressed mood most of the day, nearly every day;
- 2) markedly diminished interest in all or most activities, nearly every day;
- 3) significant unintentional increase or decrease in weight;
- 4) sleep disturbances (oversleeping or insomnia);
- 5) psychomotor changes (agitation or retardation);
- 6) low energy level or fatigue;
- 7) feelings of guilt or extreme self-criticism;
- 8) inability to concentrate or make decisions;
- 9) recurrent thoughts of death or suicidal ideation.

At least five of these, which must include either depressed mood or loss of interest or pleasure, must be present for a minimum of two weeks. Such symptoms need be of sufficient severity to cause marked clinical distress, or impairment in occupational, social or other significant areas of functioning.

### Manic Episodes.

By contrast, in a manic episode, mood is required to be abnormally and persistently elevated, expansive or irritable for a

period of at least a week, during which, the following associated symptoms are manifest:

- 1) inflated self-esteem or grandiosity;
- 2) decreased need for sleep;
- 3) increased talkativeness, pressure of speech;
- 4) flights of ideas, thoughts racing;
- 5) distractibility;
- 6) increased goal-directed activity;
- 7) excessive involvement in pleasurable activities that might have a high potential for painful consequences.

A minimum of three of these symptoms are required (four if mood is only irritable) in addition to mood disturbance for at least a week. Such symptoms must be of sufficient severity to cause marked impairment of occupational functioning, usual social activities, and interpersonal relationships. It may require hospitalisation and psychotic features could be present.

### Mixed Episodes.

A mixed bipolar episode is one in which symptom criteria for both manic episode and major depressive episode (minus duration criterion) are met nearly every day for at least a week. Once again the symptoms must be sufficiently marked to cause impairment in all important areas of functioning and hospitalisation may be required.

### Hypomanic Episodes.

A hypomanic episode has the same symptoms as a manic episode with the exception of delusions or hallucinations. Psychotic features are absent. Mood disturbance is required for a minimum of four days only, and must be 'different' from the individual's usual un-depressed mood, rather than 'abnormal'. This suggests a less severe disturbance of function than in mania. Disruption in occupational functioning is not marked and hospitalisation is not required (DSM-IV; APA, 1994a).

### Prevalence of Bipolar Disorder.

Epidemiological studies of bipolar illness are confounded by methodological problems, which result in complex pragmatic and interpretative issues, as discussed below. Recent studies, however, consistently suggest that bipolar affective disorder is a relatively common disorder that affects men and women equally, although estimates of true prevalence may be obscured by diagnostic imprecision.

Goodwin and Jamison (1990) appraise that the lifetime risk for bipolar disorder is less than 1 per cent, in the industrialised nations. Lam, Jones, Hayward, and Bright (1999) suggest a somewhat higher figure of around 1 – 1.5% of the adult population. In accord with this, the APA cites lifetime prevalence of Bipolar I Disorder in community samples as between 0.4% and 1.6%. Prevalence of the rarer Bipolar II Disorder is estimated at

approximately 0.5% (DSM-IV; APA 1994a). Compared to prevalence rates for unipolar depression, these figures appear relatively low (lifetime risk for Major Depressive Disorder is estimated at between 5% and 25%: APA, 1994a). However it has been argued that, as mania and, especially, hypomania are not always recognised, and as they are essential for diagnoses of Bipolar I and Bipolar II Disorders, respectively, there is a tendency to under-diagnose bipolar disorders (Rice, et al., 1986; Angst, 1998). Various authors have therefore questioned the commonly accepted 5 to 1 ratio of unipolar to bipolar disorder on clinical grounds suggesting instead that the relationship may be closer to 1 to 1 (e.g. Bowden, 1993).

Just as bipolar-depression can be mistaken for its unipolar counterpart, similarly, bipolar-mania has often been overlooked in favour of diagnoses of schizoaffective disorder or schizophrenia. Schizoaffective disorder, the term used to describe patients with both affective and psychotic features, has long had an uncertain relationship with bipolar disorder, giving rise to much diagnostic imprecision (Stakowski, Keck, Sax, McElroy & Hawkins, 1999). In like manner, Carlson and Goodwin (1973) observed that psychotic symptoms during a manic phase could be indistinguishable from those of acute schizophrenia. For the uninitiated, schizophrenia is defined as a disorder lasting for at least 6 months (if untreated) and including at least one month of active phase symptoms (i.e. two or more of the following, delusions,



hallucinations, disorganised speech, chaotic or catatonic behaviour and negative symptoms [the loss or diminution of normal functions], APA, 1994). Clearly this semblance can lead to other instances of misdiagnosis and under-reporting of bipolar disorder (e.g. Gonzalez- Pinto, et al., 1998). The relationship between bipolar disorder and schizophrenia (and other psychotic disorders) is discussed more fully in chapter four. Thus the traditional view of bipolar disorder as substantially less common than unipolar depression could be in need of revision because of problems of heterogeneity and symptom sharing.

Compatible with this, various authors report considerably increased rates of bipolarity when using longitudinal experimental designs or those with enhanced diagnostic procedures. Angst (1978) found that the ratio of bipolar to unipolar patients was equal on a 16-year follow-up, with at least 10% of unipolars being reclassified as bipolar when hypomania was subsequently established. More recently, in a 15-year follow up of 82 Dutch schizophrenic and other nonaffective functional psychotic patients, Wiersma, Nienhuis, Sloof, and Giel (1998), found that one in seven went on to develop affective symptoms (typically within 6 years of diagnosis) and five patients were reclassified as bipolar disordered.

Associated to this, Egeland, (1983) refers to typical Bipolar I Disorder as merely the tip of the bipolarity illness iceberg. His longitudinal Amish study suggests that the incidence of cyclothymia may be greater than that of Bipolar I Disorder

(Egeland & Hostetter, 1983). Cyclothymic patients are those whose episodes of moderate depression and hypomania are not severe enough to warrant hospitalisation. As their family history and pharmacological response fits the bipolar pattern, Egeland argued that this group should be included as part of the bipolar spectrum (Egeland, 1983), which fits with the suggestion that cyclothymic disorder is often the precursor to the later development of full-blown bipolar disorder (Goodwin & Jamison, 1990; Akiskal, 1997). It thus appears that bipolar disorder could be more common than is currently thought, but diagnostic imprecision and ambiguity in epidemiological studies seriously confound efforts to quantify the extent of bipolar affective disorder in the general population.

#### Limited Research into Bipolar Disorder.

Depending on definition and accuracy of diagnosis, as just discussed, bipolar disorder afflicts many and has repercussions for still more. Although it is one of the most serious forms of mental disorder, comparatively little research has been conducted into the role of psychological processes in this condition. This is despite the considerable advances that have been made in the understanding of unipolar depression (e.g. Gotlib & Hammen, 1992; Williams, et. al., 1988; Abramson, Alloy, & Metalsky, 1995), and recent efforts to elucidate the role of psychological processes in nonaffective psychoses or schizophrenic symptoms (e.g. David & Cutting, 1994, Garety & Freeman, 1999). As a natural paradigm for explorations of state and trait differences, the study of bipolar

disorder could yet develop as a plentiful source of data and theory for researchers interested in the psychological mechanisms of psychopathology.

### Social and Occupational Cost of Bipolar Disorder.

**“It is estimated that an adult developing bipolar disorder in his/her mid-20s effectively loses 9 years of life, 12 years of normal health and 14 years of work activity.”**

**(Jan Scott, 1995, p. 581).**

### Personal Impairment.

The paucity of bipolar research is all the more surprising given the high personal and economic costs of bipolar symptoms (Coryell, et al., 1998). DSM-IV describes the various antisocial behaviours associated with severe manic episodes (APA, 1994a). These include; substance abuse, debt, spouse abuse, child abuse and other violent behaviour, truancy, gambling and, sexually disinhibited behaviours. Or as Goffman put it when describing the family life of bipolar patients, “the issue is not that the family finds home life made unpleasant by the patient...the issue is that meaningful existence is threatened” (Goffman, 1969, p. 374).

Common to most individuals with bipolar disorder are fears and worries about the inherent unpredictability of the course of the disorder, terror of its recurrence and fear of inheritance of the disorder by offspring. Perhaps because of the intervening periods of



normality, many find it difficult to accept that theirs is a serious and chronic illness requiring lifelong prophylactic medication for its control (Goodwin & Jamison, 1990). Conversely, hypomania sufferers emphasise their sense of well being and report difficulty acknowledging that their ebullient and exalted (albeit irritable) mood is pathological. Other sufferers emphasise some positive aspects of even a full-blown manic episode. See Goodwin and Jamison (1990, chapter 14) or Jamison (1993) for reviews of the ample evidence for a superior level of creativity and leadership in those affected by this condition, (a topic which is returned to in the discussion, chapter 11). However, for many of those in the process of recovering from an episode of mania, there exists extreme chagrin and shame for bizarre behaviours undertaken during psychosis. They also report a lingering uncertainty as to the veracity of present mood, coupled with doubt about the reliability of current decisions. Ruminations about manic behaviour are an inevitable part of the post-psychotic adjustment phase for many sufferers. As with its unipolar counterpart, the pain and despair experienced during recurrent depressive or mixed manic-depressive episodes leave a destructive legacy for sufferers and their families, as patients can become hopeless, indecisive, withdrawn and suicidal (Lam et al., 1999). In the aftermath of failed suicide attempts, bipolar patients typically experience a resurgence of acute shame and humiliation, along with a dread of relapse. Bipolar patients in remission or only mildly symptomatic as measured by clinician



ratings, nonetheless have significant impairments to functioning and well-being (Leidy, Palmer, Murray, Robb, & Revicki, 1998).

### Social and Economic Impairment.

Such clinical accounts are consistent with research evidence, indicating that a large proportion of bipolar individuals experience severe, pervasive and enduring work and social impairments. For example, Coryell and co-workers' longitudinal study found that bipolar respondents were only one-half as likely as their closely matched comparison respondents to have ever been married. Those that had were highly susceptible to terminal strain on the marital relationship; the bipolar depression probands (like unipolar depressives) were twice as likely as their comparison groups to have been divorced or separated (Coryell, et al., 1993). They also reported high levels of impairment on measures of occupational status and annual income. Despite a modest educational advantage over comparison subjects with similar occupational status at intake, the bipolar participants demonstrated significantly lower incomes at follow-up. Similarly, subsequent comparisons of mean educational levels reflected the disappearance of their initial academic advantage, as bipolar individuals were significantly less likely to have improved their educational status in the six years to follow-up. Unfortunately these psychosocial impairments endured for years, and persisted even amongst those individuals who experienced a sustained remission of clinical symptoms. It is therefore clear that

bipolar disorder has profoundly adverse and far-reaching consequences, for many, even in the inter-episodic abatement of psychopathological manifestations. Quantification of the aggregate costs of lost productivity, domestic upheaval and overall suffering caused by this disorder should therefore be viewed as having major public health significance.

### Suicide Risk Amongst Bipolar Sufferers.

Not surprisingly, the cumulative impact of the varied bizarre and frightening bipolar symptoms can be so painful that suicide seems the only means of escape for those afflicted. For example, the degree of personal and marital devastation caused by Virginia Woolf's manic-depressive illness and its influence on her final decision to throw herself into the River Ouse, is poignantly reflected in her suicide letter to her husband;

“Dearest, I feel certain I am going mad again. I feel we can't go through another of those terrible times. And I shan't recover this time. I begin to hear voices, and I can't concentrate. So I am doing what seems the best thing to do....I don't think two people could have been happier until this terrible disease came. I can't fight any longer.”

(Bell, 1972 [1996], p. 226).

Virginia Woolf had endured her “terrible illness” for many years. She was in her eighties and in a prodromal stage of mania when she

ended her life. However, there now exists some equivocal evidence that the risk of suicide tends to be higher in the early years of this disorder. It has been suggested that episodes of suicide are rare in manic phases and occur more typically in depressive episodes in the wake of mania. Consistent with this Dilsaver, Chen, Swann, Shoaib, Dilsaver and Krajewski, (1994) studied 129 persons with bipolar disorder in a university teaching hospital and reported suicidality rates of nearly 80 % in their bipolar-depressed patients, whereas mania was characterised by low rates (2.3%) of suicidality. Goodwin and Jamison (1990) point out that suicide risk is also especially high in mixed manic-depressive episodes, an observation that is consistent with the Dilsaver et. al.'s (1997) study described above. However, a more recent study (Strakowski, McElroy, Keck & West, 1997) suggests that it is the severity of concurrent depressive symptoms in mania, rather than the presence of a depressive syndrome per se (i.e. mixed state), that is associated with suicidality in bipolar disorder. Perhaps inconsistent with this, but certainly compatible with Mrs. Woolf's obvious insight shortly before her death, is the suggestion by Ghaemi (1997) that suicidal proclivities may increase with insight.

Lam et. al., (1999) propose that suicide and attempted suicide risk is disproportionately high in bipolar disorder compared to other psychiatric conditions. Goodwin and Jamison (1990) estimate that between 20 % and 56% of bipolar individuals attempt suicide and, of those who remain untreated one in every four or five



actually does commit suicide. These rates are at least equal to those reported in unipolar depression and schizophrenia. The APA cites completed suicide in 10% to 15% of Bipolar I Disorder and Bipolar II Disorder cases combined, compared to up to 15% in Major Depressive Disorder, and 10% in schizophrenia (DSM-IV, 1994a).

Thus it is clear that depressed bipolar patients have a higher rate of significant suicide attempts contrasted to their unipolar equivalents. Other evidence suggests that lethality intensifies with recurrence (Goodwin & Jamison, 1990). Studies of attempted suicide in bipolar patients show that over 50% have attempted suicide at least once, with women appearing far more at risk than men (Goodwin & Jamison, 1990). Johnston and Hunt's (1979) study of suicide attempts specified severity, and classified 90% of bipolar attempts as serious enough to warrant hospitalisation. More recently, in 1996, Chen and Dilsaver reported a lifetime rate of suicide attempts in bipolar disorder of almost 30%, which compares to 16% in unipolar depression and 4% in "any other axis I disorder". Thus the earlier estimates of alarmingly high risk for suicidal behaviour were confirmed.

Whilst not inconsistent with the manic-defence theory of bipolar mania (Zigler & Glick, 1988), since suicide behaviour usually denotes unbearably low self-esteem (Neale, 1988), these findings do suggest that the escape into mania is sometimes ineffective, hence the recourse to the ultimate escape of suicide. Alternatively, the relatively low rates of suicide behaviour in the

manic phase could be seen as giving credence to the notion of the manic defence as a protective, if sometimes tardy, mechanism. However, Freud (1917/50) himself acknowledged that this subconscious attempt to overcome inner turmoil is rarely accomplished in full and is often transitory (detailed in chapter 3 and subsequently revisited in chapter 9).

### Treatment Response and Compliance.

As can be seen from the above, untreated bipolar affective patients are at enormous risk of suicide and serious self-harm. The most reliable method of preventing suicide in these patients is to treat the underlying illness effectively (Goodwin & Jamison, 1990). However, it has been variously estimated that, less than a third (Goodwin & Jamison, 1990), to less than half (Kessler, Rubniow, & Holmes, 1997) of those afflicted are in receipt of treatment. This is despite the advent of relatively effective pharmacologic treatments over the last thirty years (e.g. antimania, antidepressant, and antipsychotic drugs). Low rates of compliance appear to confound the beneficial effect of modern medication; meta-analysis of the lithium compliance literature suggesting only 60% of patients are likely to be compliant (Basco & Rush, 1995). In addition to this, Prien and Potter (1990) estimate that lithium is ineffective in up to 40% of bipolar patients, which highlights the limitations of available pharmacological treatments even for those who do adhere. Patchy availability of mental health services and difficulty

accessing alternative or adjunctive treatments has also been widely reported (Lam et al., 1999). Thus the new forms of psychotherapy tailored specifically for this population such as cognitive behaviour therapy (CBT; Scott, 1995 & 1996a; Drury, Birchwood, Cochrane, & MacMillan, 1996; Sensky, et al., 2000), or the more recent Interpersonal and Social Rhythm Therapy (IPSRT; Frank, et al., 1997; Frank, Swartz, Mallinger, Thase, Weaver, & Kupfer, 1999) are not easily obtainable. The stigma of psychotic illness can also exacerbate the problem of patient denial, and poor patient insight is also of relevance to treatment non-compliance (Ghaemi, 1997).

Further research to investigate the psychological mechanisms at play might contribute to the development of effective alternatives to pharmacologic treatment, (thus smoothing the traditionally uneasy alliance between psychotherapy and psychosis), and promote better drug compliance. For example, improved understanding of how the links between relapses and environmental stresses relate to individual sufferer characteristics, could make the psychosocial components of maintenance programmes more focused and deliberate. CBT and IPSRT are discussed further in chapter 11 of the discussion.

## The Pattern and Course of Symptomatic Episodes.

### Age of Onset.

Although usually thought of as an adult disorder, in their charmingly titled paper, Weller, Weller and Dogin (1998), report the case of a 24 year old male with bipolar disorder whose illness first manifest itself with disabling intensity when he was three years old. Despite a significant family history of affective disorder, he experienced recurrent symptomatic episodes at ages five, six and eight and a half years but remained inadequately diagnosed or treated. This case is not wholly exceptional as other authors report confirmed or suspected bipolar diagnoses in prepubertal children (e.g. Carlson, 1998; Nottelmann & Jensen, 1998).

The occurrence of bipolar disorder in childhood could be viewed as necessitating a qualification to the mania as defence theory; as one might question the capacity of a child to hold both good and poor self-esteem simultaneously. Psychodynamic theorists, however, have long espoused the notion of infantile mechanisms of defence. For example, Melanie Klein comments “quite little children pass through anxiety-situations (and react to them with defence-mechanisms), the content of which is comparable to that of the psychoses of adults” (1934, p. 282), suggesting that adult defences are often evolved in the puerile stage of development. Compatible with this, other instances of cognitive defending, such as repression or dissociation consequent to



childhood sexual abuse have been largely accepted (See BPS, 1995 for review). The simplest construction would be that mania is motivational in some but not all incidences, as such an explanation is consistent with the relative infrequency of this disorder in young children.

Examination of data from multiple epidemiological studies has resulted in an estimated bipolar disorder mean age of onset of 28.1 years. On reanalysis, to overcome cohort effects, peak onset frequencies in the 15 to 19, and the 20 to 24 age ranges were indicated (Goodwin & Jamison, 1990, chapter 6). Thus bipolar affective disorder is a condition which usually first strikes in early adulthood but which can also occur in adolescence or before.

### Course of Bipolar Disorder.

The course of bipolar disorder is complex and highly variable. Since the ancient Greeks first recognised mania and melancholia as linked psychiatric conditions, the notion of bipolar disorder as distinguished by the paradoxical manifestation of apparently opposite affective states continues to prevail. Whereas, it is true that an individual with bipolar disorder will present, at times, with episodes of mania and, at other times, with episodes of depression, the true picture is not one of a biphasic or simple temporal cycle.



Present estimates suggest that, on average, Bipolar I patients will suffer 8 – 12 major depressive episodes and 4 – 8 manic episodes during their lifetimes if clinical criteria instead of hospitalisation rates are employed as episodic indicators (Lam, et al., 1999). Rather than simply moving between depressed and manic episodes, however, individuals with bipolar disorder typically experience abrupt shifts between various emotional and psychological states. As alluded to earlier, DSM-IV and other diagnostic manuals recognise four distinct mood episodes (major depressive episodes, manic episodes, mixed manic-depressive episodes, and the less disabling hypomanic episodes), which can be included in a diagnosis of this disorder. There are also periods of remission or sub-syndromal mood lability (APA, 1994a). Individual bipolar patients vary considerably both in average duration of episode and length of time between episodes. This variation has been found to reflect various personality and environmental factors, and hence challenges the notion of a simple cyclical pattern of bipolar episodes, as discussed below.

### Duration of Episodes (Cycle Length).

The time between one episode and the next is highly variable but there is general agreement that cycle-length decreases with number of cycles (or as the individual ages). It has been estimated that early in the course of the disorder, cycle length can be as long as 40 - 60 months, but that this decreases markedly. Following the

third episode, cycle length is reduced to 10 – 20 months. As the disorder progresses to the sixth episode, the cycle length can be as little as 10 months, at which point this decrease subsides and cycle length becomes more constant (Lam, et al., 1999).

Shorter cycle lengths primarily reflect variation in the symptom-free episodes since the pattern and duration of manic or depressive episodes are believed to be relatively stable within the individual. However, approximately 20% of bipolar patients experience at least four affective episodes a year from the onset. These “rapid-cycling” patients are predominantly female and begin their illness with a depressive episode. Others are in the later stages of their illness (Goodwin & Jamison, 1990; Koszewska, 1996). However, this could reflect the acceleration of the natural course of the disorder by certain treatments. Wehr and Goodwin (1987), suggest that MAOIs and tricyclic antidepressants can worsen the course of the disorder by triggering manic episodes in some bipolar patients. Similarly, Goodwin and Jamison (1990) note that cycle length can be decreased by certain medications perhaps linked to the female susceptibility to the antithyroid effects of lithium. Other socio-environmental precipitants of symptomatic episodes of this disorder are discussed in the following paragraphs.

### Socio-Environmental Precipitants of Episodes.

Recurrences of mood episodes in bipolar illness have been associated with many different factors. Although there is much

research data in support of a biological basis for this condition, there is increasing evidence that psychosocial phenomena, such as life events and interpersonal communication factors, are associated with the onset and recurrence of both depressed and manic episodes (Goodwin & Jamison, 1990; Lam, et al., 1999, Alloy, Reilly-Harrington, Fresco, Whitehouse & Zechmeister, 1999). Compatible with this, Wehr and associates (Wehr, Sack, & Rosenthal, 1987; Wehr, 1989) emphasised the importance of sleep and life-style disruption as precipitants of symptomatic episodes in bipolar patients. They hypothesised that many of the psychological, interpersonal, environmental, and pharmacological factors that have been related to the onset of a manic phase do so through their ability to disrupt sleep.

Early episodes especially, seem to be precipitated by life events. Surprisingly it was the renowned German psychiatrist, Emil Kraepelin, who was one of the first to make this observation. In a late revision of his classic textbook, he commented that psychosocial stresses sometimes triggered individual episodes (Kraepelin, 1913 [1979]). Consistent with this, Mathew, Chandrasekaran and Sivakumar (1994) studied 46 manic patients and found that traumatic life events were present in the 6 months prior to onset of initial manic episode and that the associated levels of stress also escalated during this period. In a like manner, Beddington et al. (1993) investigated whether there was any evidence for an increased number of life events prior to the onset of



psychosis. They compared the life event histories of 31 manic, 52 schizophrenic and 14 psychotic depressed participants. They found that all three psychotic groups had experienced a significant excess of life events compared to local non-psychotic psychiatric controls and this pattern was emphasised in the three months prior to onset of psychosis (Beddington, Wilkins & Jones, 1993). These findings are also consistent with Neale's postulate that episodes of mania have a motivational function. Specifically, manic phenomena, such as grandiose delusions, reduce or provide relief from states of psychological discomfort resulting from the intensification of low self-esteem triggered by stressful life events (Winters & Neale, 1983, Neale, 1988). However, psychological antecedents appear to be less important in later symptomatic episodes, as can be seen below, although available data on this are largely retrospective, (Goodwin & Jamison, 1990).

### Relationship between Precipitants and Later Episodes.

The precise determinants of cycle length or relapse into the next episode remain only incompletely understood but it has been suggested that the increasing frequency of episodes could reflect an increasing vulnerability to emotional disturbance (Goodwin and Jamison, 1990, Reilly-Harrington, Alloy, Fresco & Whitehouse, 1999). Thus even minor or unremarked environmental stresses could trigger relapse. Consequently, although the link between certain extra-organismic factors and symptom recurrence seems to



disappear as the disorder progresses, this link might be camouflaged by increasing sensitivity to such events and reflected in increased frequency of recurrent episodes.

Consistent with this, it has been suggested that the quality of relationships with key relatives could predict the course of manic depression. Various authors have investigated ratings of “expressed emotion” in the families of bipolar patients. A high expressed emotion family denotes critical, rejecting or over-involved attitudes or behaviours of relatives. The presence of high levels of expressed emotion has already been implicated in the relapse of psychotic and depressive disorders, and has also been found to induce rapid cycling in bipolar disorder (Miklowitz, Goldstein, Nuechterlein, Snyder, & Mintz, 1988; Priebe, Wildgrube, & Muller-Oerlinhausen, 1989). Similarly, familial “negative affective style”, which connotes a critical, demanding or intrusive style of communication towards the patient, was discovered to be associated with more frequent relapse in bipolar disorder (Miklowitz et. al., 1988). Furthermore, ratings of affective style (based on frequency of critical, supportive, guilt-inducing or neutral-intrusive statements) in a family discussion of conflict issues, was found to predict patients' social adjustment at a 9 month (Miklowitz et al., 1988) and a 12 month follow-up (O'Connell, Mayo, & Flato, 1991).

The findings described above suggest that bipolar illness does not have a simple temporal pattern, nor can bipolar episodes

always be predicted on the basis of external precipitants. Cycles of symptomatic episodes and periods of remission appear to be influenced by an interaction of environmental or inter-personal events and intra-individual variables, as yet not fully comprehended. However, these results remain largely compatible with Neale's (1988) manic defence ideas. Although Neale accepted that some manic episodes are the product of the environmentally precipitated activation of low self-esteem, he suggested that the majority of manic episodes occur in the absence of major stresses. Neale elaborated that bipolar mood fluctuation and inter-episode adjustment negates the predisposing factor of a simple or continuous deficit in self-esteem. Instead, he inferred a more complex susceptibility resulting from fundamental deficits in cognitive processes. Thus inconsequential external triggers precede some manic episodes. Other episodes of mania are preceded, not by external stimuli at all, but by a cognitively produced intensification of low self-regard. The manic episode is thus the means of temporarily reducing the negative affect associated with unfavourable real-ideal self-comparisons (Neale, 1988).

Perhaps less consistent with the manic-defence theory, are the results from a more recent longitudinal study. Gottschalk and co-workers examined the temporal pattern of bipolar mood in detail and demonstrated that bipolar mood fluctuations are not patterned in a biphasic cyclical manner. They enlisted the help of seven rapid-cycling bipolar participants, who were required to monitor their

mood on a daily basis for up to two and a half years. Participants recorded their average moods over the previous 24 hour period using a short analogue scale, with anchor points “best I have ever felt” and “worst I have ever felt”, and the midpoint of “normal”. Deviation from normal mood was well correlated with objective ratings of global psychopathology. Their results suggest that mood changes in rapid cycling bipolar individuals are not truly cyclical for extended periods. No recognisable pattern of precipitation was observed but mood changes over time could be characterised as a low dimensional chaotic process (Gottschalk, Bauer, & Whybrow, 1995).

Some of Gottschalk et al.’s participants noted a continuum of affective mood states and others, the co-occurrence of depressed and elated mood. These observations could be thought of as in accord with the view of mania as the product of a defence against underlying depression (Zigler & Glick, 1988; Neale, 1988). However, the absence of any recognisable relationship between depressed and elated moods does not fit so well, perhaps indicating that the defence exists, but as suggested by Freud (1917 [1950]) it is transitory or not entirely effective. Certainly the process is likely to be multifaceted, interactional and highly complex, a notion which will be returned to in the discussion. However, Gottschalk’s challenge to the notion of episodic cyclicity in bipolar disorder could be criticised, as it remains unclear whether rapid-cycling bipolars represent a discontinuity from typical bipolar disorders. Rapid

cyclers (episodes every 3 months) and ultra-rapid cyclers (episodes every 48 hours) would perhaps be better understood on a spectrum of cyclicity (Goodwin & Jamison, 1990). An affective disorder spectrum would also be consistent with the observation that, although much longer overall, cycle length in unipolar depression has also been found to vary considerably (Stancer Persad, Wagener, & Jorna, 1987). The following chapter outlines various other areas of bipolar-unipolar dissimilitude and considers the merits of an affective spectrum as an alternative to the accepted categorical system of classification.



## Chapter Two.

### The Bipolar-Unipolar Dichotomy.

The distinction between bipolar affective disorder and recurring unipolar depression is now one of the most broadly recognised sub-divisions of affective disorders. It rests on the proposition that depressions with manic episodes and depressions without manic episodes should be viewed as discrete disorders. As such they have presumed differences in their likely cause, course, and treatment.

However, the accepted criterion that distinguishes bipolar and unipolar illness is evolving and has changed considerably over the years. In his classic studies, Leonhard, (1957) used both the terms “bipolar” and “unipolar” to describe patients with a phasic course of recurring endogenous episodes. Some clinicians (especially in America) now use the term unipolar as synonymous with nonbipolar. It thus covers the heterogeneous population of all depressed patients without a history of mania. Interestingly, the unipolar-bipolar distinction was not formally incorporated into the World Health Organisation’s International Classification of Diseases until the most recent revision (WHO, ICD-10, 1992). Earlier editions, up to ICD-9 (WHO, 1977), advise that “circular” manic-depressive psychosis is equivalent to bipolar illness, and manic-depressive illness (depressed type), represents unipolar depression. As already outlined, the APA classificatory system

adopted this separation somewhat earlier in their 1980 revision (DSM-III).

The distinction between unipolar major depression and bipolar disorder is not without considerable controversy however (Gotlib & Hammen, 1992), and may have gone further than the evidence warrants (Goodwin & Jamison, 1990). Clearly the features of the depressive episode may be very similar for some individuals with unipolar or bipolar depression. Indeed, differential diagnosis to distinguish unipolar depression from the atypical Bipolar II Disorder is especially difficult if the hypomanic experiences that define the bipolar component are brief or infrequent (Gotlib & Hammen, 1992).

Problems of heterogeneity, discussed in relation to unipolar studies above, have also been encountered in bipolar research. Many bipolar studies do not specify inclusion criteria adequately (Dunner, Gershon, & Goodwin, 1976) and others might reflect the problems diagnosing mania or hypomania outlined above. Prior to the mid 1970s, research literature did not specify the number of bipolar patients with or without a history of mania. Over the last ten years however, numerous studies have examined the bipolar-unipolar dichotomy, covering family history, personality factors, natural course, clinical symptoms, biological measures, and response to pharmacological treatments. Their findings, many of which have already been alluded to, are summarised below (see Goodwin & Jamison, 1990, p.63, for details). Of the following reported differences, not all have been replicated uniformly.

## Unipolar-Bipolar Depression Differences.

### Natural Course.

Bipolar disorder patients were found to have a younger and narrower range of onset, a higher number of symptomatic episodes but shorter cycles compared to unipolars. The greater impact of environmental precipitants, already alluded to, in bipolar individuals is not reported in unipolar depression.

### Epidemiology.

The lifetime risk for unipolar depression is believed to be considerably greater than for bipolar disorder, although this remains contentious as already discussed. Substance abuse and suicide are reported as higher in bipolar individuals. The sex ratio is approximately equal in bipolar disorder but females predominate in unipolar depression. The proportion of time wherein the patient meets criteria for a major affective illness is greater in unipolar disorder but psychosis is believed to occur more often in bipolar disorder. Anxiety and agitation are reported to be higher in unipolar depression than bipolar depression.

### Family History and Genetics.

The genetic contribution appears to be greater in bipolar disorder, with a higher monozygotic twin concordance rate than in unipolar disorder.

### Personality and Interpersonal Factors.

Bipolars are reported as having higher divorce rates but more normal personality profiles than unipolar individuals. They are also reported to be more stimuli seeking, to have less impulse control,



but more social desirability. Bipolar individuals are also reported as rating lower on measures of introversion and inter-episodic depression than unipolars (although this does not fit with the data on enduring vocational impairment). Being single has been reported as a risk factor for unipolar depression but not for bipolar disorder.

### Biological and Physiological Factors.

Bipolar individuals are reported to demonstrate less (peripheral) noradrenergic function and lower serotonergic function than unipolar individuals. They are reported to be less sensitive to pain than their unipolar counterparts. They demonstrate more hemispheric dysfunction and lower regional metabolism. Psychomotor retardation and sleep disturbance is more frequent in bipolar than unipolar depressives, and bipolar sleep duration is longer. The two groups are reported as demonstrating different seasonal patterns (i.e. bipolars tend towards depressed episodes in autumn or winter and to mania in spring and summer, whereas unipolars are believed to become depressed in spring and autumn).

### Pharmacological Response.

Bipolar individuals are thought to demonstrate a somewhat lesser antidepressant response to tricyclics than unipolars but have certainly been found to have a greater antidepressant response to lithium than unipolars. Tricyclic and MAOI antidepressants are reported as eliciting a manic or hypomanic response in bipolars more frequently than in unipolars. The prophylactic response to lithium has been demonstrated to be the same in the two groups when bipolar and unipolar cycle length is comparable. Prophylactic



response to tricyclics is poor in bipolar individuals but good in unipolar individuals.

The above account of reported unipolar-bipolar differences is compelling despite the heterogeneity of the groups. Nonetheless, bipolar and recurrent unipolar depressions seem to be very alike in some notable aspects.

### Polarity Spectrum.

Clearly the relationship between unipolar and bipolar affective illnesses is of both clinical and theoretical interest. As alluded to above, data relating to family histories and drug responses (Goodwin & Jamison, 1990, chapter 3) suggest that a considerable proportion of unipolar individuals are closely related to bipolar individuals and could occupy an intermediate position on a spectrum of polarity. In one of the few recent studies to consider bipolar personality dimensions, Rosenfarb, Becker, Khan, and Mintz, (1998) report high levels of self-criticism in both unipolar and bipolar women, and note that this trait was not dependent on depressed mood. Over and above the common neglect of hypomanic episodes in mistakenly diagnosed unipolars (referred to previously), many unipolar patients exhibit “bipolar features” (Goodwin & Jamison, 1990). Specifically, unipolar patients with early onset, frequent symptomatic episodes and bipolar family histories have been found to share pharmacological response characteristics with typical bipolar patients (Cassano, et al. 1988).

Like a proportion of bipolar patients, this unipolar group is at risk of developing hypomania on antidepressants. They also demonstrate equivalent rates of prophylaxis (e.g. Davis, 1976) and antidepressant response on lithium (Goodwin, Murphy, & Bunney, 1969). Similarly, in a more methodologically rigorous trial, Schou (1979) reported that certain early onset and short cycling unipolar individuals exhibited a better antidepressant response to lithium than to tricyclics and that their prophylactic response to lithium was equal to that of bipolar individuals.

Also of interest on the polarity spectrum, is the observation that unipolar-bipolar differences diminish as the unipolar group encompasses the more seriously ill (Stancer, et.al., 1987). Of course this might reflect a homogeneity problem as there is likely to be some overlap between bipolar and the increased severity unipolar forms. Compatible with this, Klein, Taylor, Harding, and Dickstein (1988) observed that unipolar patients with co-existing dysthymia (the subsyndromal but chronic form of unipolar depression) have more bipolar relatives than those without the double diagnosis and typically will have experienced hypomanic episodes at follow-up.

As the above findings suggest, the spectrum or continuum concept of affective disorders is closely related to cyclicity, and integrates various aspects of bipolar illness also helping to explain the continuity between the unipolar and bipolar forms of affective disorder. The continuum model also accommodates the less disabling forms of hypomania or cyclothymia that merge into personality.

### Spectrum of Depressive States.

The debate as to whether depressive states should be divided into categories or arrayed across a continuum has proceeded, unresolved for decades (see Kendell, 1976, for review). Goodwin and Jamison (1990) argue that a spectrum approach to depressive disorders would apply well to the depressive episodes which occur in bipolar disorder. For example, it would better accommodate instances where patients presently described as having only “mild” or “reactive” (and hence sub-syndromal depressed symptoms) go on to develop either major affective disorders or bipolar disorders. A spectrum or continuum concept was implicit in Kraepelin’s classic descriptions of bipolar disorder. For example:

“Manic-depressive insanity (includes) certain slight and slightest colourings of mood, some of them periodic, some of them continuously morbid, which on the one hand are to be regarded as the rudiment of more severe disorders, on the other hand pass without sharp boundary into the domain of personal predisposition. I have become..... convinced that all of the above-mentioned states only represent manifestations of a single morbid process”.  
(Kraepelin, 1921, p. 1).

More recently, Depue and Klein, (1988) used a self-report measure to look at intensity, duration and frequency of mood lability in normal, cyclothymic and Bipolar II Disorder populations. Their results demonstrated that reports from one group merged imperceptibly into the next: a finding that lends verisimilitude to



argument that an affective spectrum applies equally to bipolar and unipolar disorders.

### Spectrum of Manic States.

As already stated, the notion of a manic spectrum can be traced back as far as Kraepelin's (1921) conjecture that people with manic and cyclothymic temperaments belong to a manic-depressive group which embraces a continuum from normal temperament to full blown psychosis. More recently to champion the manic spectrum model, is Angst whose 1998 study provides further evidence for a broad spectrum of manic – hypomanic syndromes. Such disorders he asserts are more highly prevalent than previously thought, and occur in a variety of complex forms in association, either simultaneously or subsequent to, depression. This is consistent with the earlier postulate by Goodwin and Jamison (1990) that manic episodes are complex, often comprising elements of depressive manifestations. Phenomenological studies have suggested that manic episodes appear to progress through three stages. In the first euphoria, elation and grandiosity are usually most evident. Pressured, tangential speech is noted and the individual is hyperactive but irritable if demands are not met. Patients describe this stage as "going high". This first stage is revisited later in the manic episode, in the wake of the cumulative psychotic stage, when the individual is "coming down" but presents as still over-talkative, seductive and depressed. The second stage is mainly characterised



by anger and irritability, but increased depression and dysphoria, suspicion and hopelessness are noted. The third and most severe stage is mainly characterised by panic and delirium. Psychotic symptoms, especially thought disorder, are common in this stage (Carlson & Goodwin, 1973). However, it has been noted that such florid psychotic disorganisation tends to be under-reported by both patients and relatives (Orvaschel, Thompson, Belanger, Prusoff, & Kidd, 1982).

The above range of experience relates to hospitalised and hence more severely impaired bipolar patients. The notion that mania reflects a spectrum of ever increasing joy has also been challenged in the less severe manifestation of mania (i.e. Bipolar II Disorder). Here too, rather than hypomanic or cyclothymic states representing the antithesis of depressed states, they can be marked by desultory, incoherent and aimless thought, hasty and shallow judgements, and restlessness. Similarly, the relatively pleasant emotions typically associated with hypomania are replaced by qualitatively different, and less pleasant moods, as one passes into mania (Klerman, 1981).

Thus it can be seen that overall mood is depressed as often as it is euphoric during the manic phase (Goodwin and Jamison, 1990). For example, Cassidy and co-workers (Cassidy, Murry, Forrest & Carroll, 1998) conducted a large-scale study of signs and symptoms in 316 patients meeting DSM-III-R criteria for bipolar manic or mixed states. Mania, they discovered was characterised

most frequently by motor activation, accelerated thought processes, pressured speech and decreased sleep. However, they also found that although euphoria and grandiosity was present in a large proportion of their manic respondents and more common than in mixed bipolar participants, irritability, dysphoric mood, anxiety and mood lability were also prominent in the entire cohort. Further, the occurrence of paranoia did not differ significantly between groups. In accord with this, Kotin and Goodwin (1972) had earlier reported regularly observing concurrent depression during acute episodes of mania in hospitalised bipolar patients. Over the course of a longitudinal study, they further observed that depression scores are sometimes higher in manic episodes than they are in depressive episodes. However, it is not clear whether such “mixed” or “dysphoric” manic episodes represent a state distinct from simple mania, a more severe form of mania, ultra-rapid cycling between manic and depressed mood states, or a stage in the evolution of manic episodes (McElroy, Keck, Pope, Hudson, Faedda, & Swan, 1992; Koszewska, 1996). Nonetheless, the view of bipolar disorder as manifest in a spectrum of different affective states might be seen as giving some credence to the psychoanalytic hypotheses that mania is a defence against depression (c.f.; Zigler & Glick, 1988). Similarly, Kotin and Goodwin’s (1972) evidence of concurrent depression in a proportion of manic individuals is in accord with Neale’s account (Winters & Neale, 1983, Neale; 1988) and those of the earlier psychodynamic theorists, that mania is a defence against depression. However, this observation could once again, impinge

upon such a theory to imply that the manic defence is a poor, unstable or sometimes inadequate one (e.g., Freud, 1921; Freeman, et al., 1998; Garety & Freeman, 1999).

Clearer evidence consistent with the hypothesis of a manic-defence is available from a limited number of studies that have used psychological measures. These will be discussed later, in chapter four.

### Manic Episodes in Bipolar Disorder.

The various differences between unipolar and bipolar illness have been discussed above. Nonetheless it is probably safe to say that the depressive features, which appear in the depressive episodes of bipolar disorder are not entirely dissimilar to those which occur in unipolar illness, and which have been subject to such intensive psychological investigation. Much less, then, is known about the psychological characteristics of episodes of bipolar-mania.

As previously alluded to, mania is usually regarded as a state that exists at the opposite end to depression on a spectrum of affect. This is reflected by the use of the term “bipolar” to describe individuals who suffer from both types of episode. Such a conception of mania, however, has not been universally accepted. The idea that mania might be an end stage of melancholia can be traced at least as far back as Aretaeus of Cappadocia in the 2nd

century AD (Goodwin & Jamison, 1990) a view that was to prevail for centuries to come.

The following chapter considers the hypothesis that bipolar mania might be the outcome of an abnormal defence against underlying depression; a “psychoanalytical” notion that has clear roots in the wisdom of ancient Greece.



### **Chapter Three.**

#### **Mania as a Defence against Depression.**

**“I’m not happy, I’m high. Have you ever been high? Its horrible” (the words of a detained bipolar-manic patient; overheard talking to his nursing escort, 27/9/99).**

#### **Early Psychodynamic Writings.**

**The precise nature of the relationship between the two manifestations of bipolar disorder is still poorly understood. Leonhard (1957) suggested a close alliance between the psychological processes involved in depression and those implicated in mania. Early psychoanalytic writers have argued that the different phases of manic-depressive illnesses were linked together through some shared underlying mechanism. They believed that mania could thus be understood as an extreme defence against underlying and unbearable negative affect, or as a reaction to an unbearable depression. This fundamental psychoanalytic concept has been stated in different ways by different authors.**

#### **Abraham.**

**Karl Abraham (1911[1927]), was one of the earliest writers to formulate psychoanalytic principles in manic-depression. In the first psychodynamic essay in this field, he suggested that mania and depression were dominated by the same complexes which were**

overwhelming during depression but which individuals were able to treat with indifference during episodes of mania. The basic tenet of his approach being that the same processes equally underlie mania and depression, and that both phases of the disorder are different reactions to the same enduring difficulty. Abraham further described the abnormal character development and inability to maintain satisfying relationships so often seen in untreated bipolar individuals. These features coupled with the ongoing sense of impending loss of love-objects (targets of affection), combine to produce a “rageful” stance against these objects and their inability to gratify narcissistic demands.

### Dooley.

Dooley, (1921), summarised quite eloquently;

“The behaviour found in the manic attack, in which the patient throws himself with almost equal vim into every possible avenue of expression, is in itself a defence reaction. By thus taking the offensive he keeps himself safe from the approach of the painful thought or feeling which is usually a realisation of some failure or degradation, or fundamental inferiority of his own. When he is depressed his defence is no longer possible and he is weighed down by the pain of the acknowledged defect.” (p. 167).

Freud.

On the whole, Freud agreed with Abraham and suggested that bipolar interpersonal behaviour demonstrates the narcissistic tendency to be simultaneously over-fixated on love-objects and too quick to withdraw [their] object from them, in the form of cathexis (Freud, 1917[1950]). In Freudian terms, the development of bipolar disorder could be seen as a reaction to the loss [and] introjection of the ambivalent feelings toward the lost object (Freud, 1921). That is, in lay terms, hostility or anger towards the significant other (frustrating object) is redirected inwards (introjected to the ego). This results in unbearable inner turmoil (the anger enlists on the side of the superego and attacks the ego). The excessive or delusional self-reproach is aimed at manipulating the significant other (an attempt to attack the introjected self-object).

Freud also suggested a tentative reason for the appearance of a manic episode:

“The ego must have surmounted the loss of the object... whereupon ... the whole amount of anticathexis which the painful suffering of the melancholia drew from the ego and “bound” becomes available”. (Freud, 1917[1950], p. 176).

Thus, the ego attains what it is striving for in the form of perceived narcissistic supplies of complete approval, acceptance, and love. The ego vanquishes the superego, and releases energy previously constrained by the depressive struggle. This process is manifest in the increased self-esteem, grandiosity, and apparent imperviousness to risk, typical of mania. However, the fears and concerns of the

superego are rarely or not entirely overcome and are wholly or partially defended with denial, reaction formation, and over-compensation.

As previously suggested, this qualification, of the manic-defence as only partially effective or transient, perhaps relates to and answers some of the critiques of contemporary reformulations of the manic-defence.

### Rado.

Elaborating on Freud's account, Sandor Rado (1928) proposed the important addition of an intra-individual predisposition to bipolar affective disorder. He argued that an intense narcissism renders such individuals vulnerable to events that seriously threaten self-esteem, and he writes:

“Those predisposed ... are wholly reliant and dependent on other people to maintain their self-esteem; they have not attained the level of independence where self-esteem has its foundations in the subject's own achievements and critical judgement. They have a sense of security and comfort only when they feel themselves loved, esteemed, supported and encouraged. Even when they display an approximately normal activity in the gratification of their instincts and succeed in realising their aims and ideals, their self-esteem largely depends on whether they do or do not meet with approbation and recognition. They are like those children who, when their early narcissism is shattered, recover their self-respect only in complete dependence on their love-objects”. (Rado, 1928, p. 422).



Self-esteem is thus considered unstable and based exclusively on an obsessive need for external approval. Consistent with previous psychoanalytic theories of bipolar disorder, Rado goes on to claim that the lost object was introjected twice. Once when the good object is introjected into the superego, and then again when the bad object is introjected into the ego. The psychic function of melancholia is to demolish the bad object, and thus the ego can recover the love of the good object. In psychoanalytic theory, the advent of a manic episode heralds the accomplishment of this task.

#### Klein and Kohut.

Also important to this review are the works of Melanie Klein and Heinz Kohut. Klein is recognised as principally responsible for developing the “object-relations theory” from Freud’s earlier ideas. See chapter seven of Segal (1964 [1973]) for a review of Klein on “manic defences” or Cheshire and Thomae (1987) for a more general critique of her theories. In summary however, her somewhat impenetrable 1934 paper places the emphasis on “denial” and “omnipotence” and makes explicit the notion of a manic defence, which was merely implied by Freud (although the term “manic defence” actually originated with Fairburn in 1941). Also explicit in Klein’s work and those of her adherents is the concept of delusions as a defence; the defensive role of paranoid delusions as relating to the projection of repressed hostility or aggression becoming a standard psychoanalytic notion.

In line with the earlier theories of Klein, Kohut (1977) is noted for the restoration of idea of the self in relation to very early infantile omnipotence and self-esteem associated with the quality of the mother – infant bonding. Based primarily on Freud's (1914) notion of "primary narcissism", Kohut's self-psychology contrasts two poles of primitive self. One pertains to the ideal self which incorporates a sense of completeness. The other portrays ambition, the focus of which is the restoration of the aforementioned completeness. People (psychological objects) provide a "mirror" to reflect this ideal in various forms of transference. Thus, feelings of grandiosity are seen as reaction-formation defences. Similarly, in Kohut's account, difficulties in the experience of self-regard or self-esteem can be traced to interpersonal conflicts and defence processes established at the beginning of the human interaction between mother and child. Kohut's well-established concept of "self-object" describes the internalisation of the qualities of others which heighten the grandiosity (completeness) of the primitive self.

### The Influence of Such Psychoanalytic Writers.

Various other early psychoanalytical theorists discussed both the aetiology of mania and the underlying personality structure of bipolar patients (e.g. Fenichel, 1945; Jacobson, 1953). Most concern the projection of an unfilled need, desire or wish, and have acknowledged an underlying psychic process that connects mania and depression whilst serving to reduce, or provide relief from,

states of psychological discomfort. These psychoanalytic formulations could be viewed as both reminiscent of the more ancient observations that mania grew out of melancholia, and as anticipatory of the contemporary continuum or spectrum models of this disorder.

Such ideas have long been fairly influential in North America, (as reflected in the empirically based American DSM-IV's reference to psychosis as "a loss of ego-boundaries"; APA, 1994, p. 273). However, until fairly recently, the British and German psychoanalytical and psychosocial traditions continued to develop in relative isolation from the mainstream of psychiatry which largely retained its medical disease model (Goodwin & Jamison, 1990). Psychoanalytical perspectives of psychopathological phenomena (including those of bipolar disorder) also suffered from the recurrent problems of interpreting open-ended clinical phenomena. They are often viewed as retrospective, interpretative, and speculative. Comparison groups are lacking, and there are few methods by which to empirically test underlying theories. However many clinicians, of both psychodynamic and cognitive-behavioural persuasion value the psychodynamic contribution to the understanding of this and other psychiatric conditions and acknowledge its influence on their research and practice.

### Contemporary Reformulation of Psychodynamic Accounts.

Neale's modern reformulation of earlier psychodynamic hypotheses attempted both to rekindle interest in such ideas and muster empirical evidence in support of them (Winters & Neale, 1983 & 1985; Neale, 1988). In his 1988 paper, John Neale proposes a motivational theory of the development of bipolar disorder and in particular the grandiose delusions and ideas typical of mania and predictive of a diagnosis of bipolar disorder. Carlson and Goodwin (1973) reported that 75 per cent of their bipolar patients had delusions during a manic episode. Even where full-blown delusions do not occur, grandiose ideation is likely; for example, 90 per cent of bipolar patients showed grandiosity in an early high-risk project by Strauss (1969).

### Unstable Self-Esteem as a Predisposition for Mania.

It has already been noted that, like Rado, Neale (1988) argues that unstable self-esteem, is an important predisposing factor in the aetiology of bipolar disorder. His full account revolves around two principal themes. Firstly, he advocates a specific motivational force which gives rise to manic phenomena, and in turn serves to reduce, or provide relief from, states of psychological discomfort. Secondly, he suggests that these delusional beliefs result from a fundamental deficit in cognitive processing.



Hence, as mentioned earlier, Neale suggests that vulnerable individuals do not merely suffer a simple, or more importantly, a constant deficit in self-esteem as evidenced by inter-episodic adjustment. Rather, theirs is a complex predisposition, arising from an abnormal need to achieve in specific life spheres married to characteristic and unrealistic standards for success, thus setting the scene for unfavourable comparisons between the individual's actuality and ideal self. Thus the person's self-esteem becomes vulnerable to any life experience that might threaten or suggest failure, however apparently inconsequential. Whilst some manic episodes will be triggered when conspicuous external precipitants activate poor self-esteem, on other occasions, internal stimuli act as precipitants. Indeed, Neale (1988) suggests that in most instances mania occurs as the result of a cognitively produced intensification of low self-regard, in the absence of stressful life events. Hence, for example, thoughts of anticipated failures or recollections of previous disappointments give rise to unbearably low self-regard. Because of the emotional salience of internalised impossible standards, these individuals are unable to access the more normal defence mechanisms of inflating their own worth or deflating their faults and weaknesses.

It should be mentioned though, that this account is closely derivative of William James' (1902) classic account of the "material" and the "social self" as discussed with some erudition by

Cheshire and Thomae (1987, pp. 25 - 29). Just as the founding father of modern psychology pre-empted Neale's motivational account of mania with his equation of "pretensions versus achievements"(James, 1902), he also anticipated Freud's (1923), theory of disturbances of self-concept with his 1890 writings on the consciousness of Self (James, 1890).

To return to Neale however, who enjoins the evidence from Depue and Munroe, (1978) in support of his argument. They suggest that the link between preceding trauma and bipolar episodes is weaker than it is for unipolar episodes, and that it is less evident for manic than for depressive episodes in bipolar disorder. These findings are largely consistent with more recent studies. Neale (1988) also hypothesised that a predisposition to grandiose delusions arises as a result of regular use of pleasant fantasies to cope with routine stress. However, whilst an effective distraction technique for many, for those individuals with a marked real-ideal incongruence, fantasy can come to predominate the mundane constraints of reality. As self-esteem is frequently threatened so recourse to fantasy becomes more frequent, and detailed fantasy constructions develop. With time and practice, the boundaries between reality and fantasy are diminished. This process is exacerbated as the individual develops increasingly detailed fantasies often with complex sensory associations. Such sentient stimuli may subsequently trigger delusional thinking. Elicitation of fantasies then becomes increasingly easy and necessary, to the point

where it is almost automatic. Thus “fantasies that occur frequently and become increasingly realistic set the stage for the development of grandiosity and delusion” (Neale, 1988, p.146). Alternatively, Freudians would perhaps see this defence as a “regression” to the attitude or disposition of “infantile omnipotence”, which is seen as a normal developmental stage of infancy.

Given that various aspects of mania themselves bring disagreeable repercussions, the likely success of Neale’s model could be questioned if the defence mechanism itself causes psychological distress. Grandiose delusions, associated with behavioural excesses often resulting in loss of friendship, unemployment or debt, do not immediately occur as ideal candidates for keeping distressing cognitions and life-events out of consciousness. However such adverse consequences tend to be rather long-term. In the short term, the public announcement of some grandiose assertion, for example, rarely meets with an overtly hostile reaction. Typically the grandiose person is humoured by relatives or professionals, or ignored by strangers.

Translating Neale’s theory into the language of modern cognitive psychology, mania can be regarded as the extreme product of self-regulatory processes, which might have the function of preventing negatively self-referent cognitions from entering into the consciousness, a mechanism perhaps much akin to the self-regulatory process which becomes particularly evident in the normal individual during life-threatening situations (Taylor, 1983).



## Two Models of Defence.

Clearly related to Neale's original "manic-defence" model (as applied to delusional bipolar and schizophrenic individuals), is the "delusions-as-defence" model, as applied by Bentall and colleagues (e.g. Kinderman, Kaney, Morley & Bentall, 1992, Bentall, Kinderman, Kaney, 1994; Kinderman, 1994; Kinderman & Bentall, 1997) to individuals with persecutory delusions (summarised by Bentall & Kinderman, 1998). As already established the delusions-as-defence conception goes back a long way in the psychoanalytic literature; it was certainly implied in Freud's early writings and made explicit by Klein (1934). At its simplest, the delusions-as-defence premise is directly parallel to the manic-defence line for grandiose delusions in bipolar disorder. That is, persecutory delusions reflect a tendency to avoid blaming oneself for bad events in the environment, a proclivity, which serves to prevent underlying low self-esteem from reaching the consciousness. (Bentall, 1974). The parity of the two models reflects their shared roots in psychodynamic theory and is consistent with the more recent influence of Zigler and Glick's (1988) suggestion that paranoia and mania have shared common cognitive mechanisms to defend against depression and feelings of inadequacy. It is likely therefore, that the recent criticisms of the delusions-as-defence hypothesis (Freeman et. al., 1998; Garety & Freeman, 1999) will also apply to the manic-defence hypothesis. These commentaries will be alluded



to later (in chapter 5, and again in the discussion), before which it is appropriate to consider the relative semblance of bipolar disorder and schizophrenia or other psychotic conditions.

## Chapter Four.

### Bipolar Disorder and Other Psychotic Disorders

Compounding the dilemmas caused by attempts to keep bipolar and unipolar major affective disorders discrete, are the commonly encountered problems of overlapping boundaries between bipolar disorder and schizophrenia or schizoaffective illnesses. Of particular pertinence here are the distinguishing characteristics of hallucinations and delusions found in psychoses. This conundrum has been charmingly elucidated, as follows:

“We have before us a fruit called psychosis, and we don’t know whether it’s a citrus that will divide itself into separable sections or an apple that we must divide along arbitrary lines”. (Belmaker & van Praag, 1980, p. 258).

### Bipolar Disorder and Schizophrenia.

Thought disorder is not limited to schizophrenia-type conditions, and strict application of the Bleulerian concept of thought disorder as pathognomonic of schizophrenia can result in misdiagnoses of bipolar disorder. Thought and perception are profoundly changed in bipolar disorder and although there are few quantitative differences in formal thought disorder between bipolar mania and schizophrenia, qualitative differences do exist (Goodwin & Jamison, 1990). Manic patients exhibit more pressured speech,

flight of ideas, grandiosity, and over-inclusive thinking (Ragin & Oltmans, 1987). Thought disorder can continue long after acute episodes have subsided in bipolar-mania, and speech patterns tend to be characterised by greater totality and complexity of speech, and more fluctuation between discourses, than in schizophrenia (e.g. Ragin & Oltmans, 1987). Delusions are common during both bipolar-depression and bipolar-mixed episodes, and especially frequent during bipolar-mania. Depressive delusions tend to be characterised by themes of poverty, guilt, persecution, or somatic concerns, whereas manic delusions are generally expansive, religious, and communal. Manic delusions are generally less fixed than schizophrenic delusions (Kendler, Glazer, & Morgenstern, 1983). Approximately half of bipolar patients report a history of delusions compared to only one fifth reporting hallucinations (Goodwin & Jamison, 1990). However, although less common than delusions, hallucinations do occur in both bipolar-manic and bipolar-depressive episodes but are characterised by their fleeting and often ecstatic or religious themes. Bipolar hallucinations are qualitatively more akin to the hallucinations of organic psychosis than schizophrenic psychosis (Carlson & Goodwin, 1973).

Clearly there are various qualitative differences between bipolar and schizophrenic psychosis, but it appears that these are quite subtle and as such are not immediately identified in all instances (Carlson & Goodwin, 1973). Incorrect clinical diagnoses in the absence of a reliable informant or other source of accurate personal and family history apply equally to the bipolar-

schizophrenia symptom similitude as to the bipolar-unipolar overlap, discussed earlier. This is consistent with the results of the Iowa 500 study (Tsuang, Winokur, & Crowe, 1980) which noted that the diagnosis of mania was made more often in relatives of bipolar probands if hospital charts were used instead of personal interviews for diagnostic purposes. In fact sole reliance on clinical interview resulted in an under-estimation of bipolar disorder by up to one third, suggesting that bipolar patients may deny psychotic symptoms perhaps because of poor insight or because they are otherwise unable to report them.

This suggests that information regarding the previous course of the individual's disorder from an outside source is essential for accurate diagnosis and a reliable estimate of the incidence of the disorder. Examination of the comparative courses of these disorders could also provide retrospective clues to diagnoses. The prognosis for bipolar disorder, although chronic, is believed to be better than for schizophrenia or schizoaffective disorder, with many bipolar individuals experiencing complete interepisode and functional recovery (APA, 1994), as opposed to higher levels of persistent psychosis in schizophrenia and schizoaffective disorder (Stakowski, Keck, Sax, McElroy and Hawkins, 1998). Details of a patient's premorbid background can also contribute to the diagnostic dilemma. For example, seasons of birth tendency in different psychiatric groups have also been well documented. Castrogiovanni, Iapichino, Pacchierotti and Pieraccini, (1998) reviewed the literature of this intriguing field and conclude that



persons who go on to develop schizophrenia are more likely to have been born in winter and spring, whereas those with subsequent bipolar disorder, like unipolar depressives, are more likely to have birthdays in the first quarter of the year.

### Bipolar Disorder and Schizoaffective Disorder.

Regardless of clear diagnostic indicators available from an extensive examination of the course of the individual's illness, Goodwin and Jamison (1990) maintain that one of the most commonly encountered problems in making a differential diagnosis in bipolar illness involves its symptom sharing with schizophrenia and the lesser-known schizoaffective illness. Schizoaffective illness was a term first coined by Kasanin some 60 years ago (Kasanin, 1933), and although not synonymous with "schizophreniform disorder" (a quick schizophrenia!), it has largely replaced that diagnosis. Schizoaffective disorder is a disturbance during which there is either a major depressive or manic syndrome, concurrent with symptoms that meet criteria for schizophrenia (delusions, hallucinations, incoherent or loosened associations, catatonic behaviour, flat or grossly inappropriate affect) for at least two weeks (DSM-III-R, APA, 1987). In earlier editions of DSM however, schizoaffective disorder was included as a subtype of schizophrenia, but subsequent research (e.g. Taylor, 1984) acknowledged Schneiderian first rank symptoms (thought broadcasting, thought insertion, experiences of influence, delusional

perceptions and incomplete auditory hallucinations) in patients whose family history, symptoms and disorder course and outcome would otherwise put them on the manic spectrum. Strakowski et. al.'s (1998) study found similar rates of "so called" first-rank schizophrenic symptoms and mood-incongruent psychosis in 27 bipolar and 27 schizoaffective patients, and thus challenged the historical differentiation of the two disorders on the basis of positive symptoms alone. However, Strakowski and colleagues did detect differences at 12-month follow-up, by which time the schizoaffective group were significantly less likely to achieve syndromic recovery, although neither group attained high rates of symptomatic or functional recovery

### An Affective-Psychotic Disorder Continuum.

Compatible with reports of gross similarities between bipolar and other psychoses, various authors (reviewed in Goodwin & Jamison, 1990) have highlighted methodological difficulties not dissimilar to those previously outlined in the unipolar-bipolar dichotomy deliberation. Contention also persists concerning differential diagnosis of bipolar disorder in relation to personality disorders, schizophreniform disorder, brief reactive psychosis, cycloid psychosis (another hybrid schizophrenia-affective overlap), drug-induced psychosis, organic brain disorders and the epilepsies. Such controversies of diagnosis and treatment have implications for current practices in the clinical and research diagnosis of the

disorder, particularly in perhaps obscuring a correct epidemiological appreciation of the prevalence of bipolar disorder (Ghaemi, 1997). Further debate of these issues lies beyond the scope of the present thesis, but a cogent review can be found in Goodwin and Jamison (1990, chapter 7).

The existence of schizoaffective admixtures however, are compatible both with a two-entity model of psychotic illness, which assumes discontinuity of symptoms, and a continuum model (not unlike the unipolar-bipolar paradigm outlined above). In the latter, affective and schizophrenic symptoms represent different dimensions of vulnerabilities interacting to produce a spectrum of states from pure schizophrenia to pure bipolar disorder. As Toomey, Faraone, Simpson and Tsuang (1998) point out, similar symptom factors in schizophrenia and mood disorders suggest a continuity in the major affective and psychotic disorders that appears to reflect an underlying dimension of a psychotic process. Such a schizophrenia-affective continuum is both compatible with Neale's (1988) notion of motivational interconnectedness between affective and psychotic manifestations of bipolar disorder, and Zigler and Glick's (1988) model of bipolar mania and schizophrenic paranoia sharing a common mechanism to escape psychic discomfort. A spectrum conception of psychological disorders might contribute to the elucidation of the difficulties posed the manic-defence argument; namely the defence's failure completely to protect the individual from impoverished self-esteem during mania or at all during depression.



### Phenomenological Investigations.

Alternatively, various authors have chosen to transcend the difficulties of heterogeneity in psychiatric groups by focusing their enquiries on individual symptom clusters, rather than the syndromes, which remain in dispute. Indeed such phenomenologically focused research, as recommended by Persons in 1986 and subsequently by Bentall, Jackson and Pilgrim in 1988, amongst others, has already proved a profitable research strategy. In particular, a number of such studies have been undertaken in the field of persecutory delusions (e.g. Bentall & Kaney, 1989; Bentall & Thompson, 1990), and have provided valuable insights into the cognitive structures associated with this symptom cluster, which in turn, led to the formulation of the delusions-as-defence model described above. This argument aside, nosological and classificatory debate is of both theoretical and clinical interest. Improved understanding of regularly confused psychiatric conditions, whether through specific symptom focused research or more traditional syndrome focused exploration, will inevitably lead to more precise diagnosis and hence more effective treatments for those afflicted whose main concern will surely be that of symptom relief rather than nosological resolution.

The advantages of studying psychological phenomena rather than psychiatric diagnoses are aptly illustrated in the following paragraphs. These pertain to unipolar-mania and its purported



existence as a discrete psychiatric syndrome, rather than a symptomatic manifestation of another condition (most likely bipolar disorder). The existence of unipolar mania as a unitary entity is discussed with regard to its implications for the manic-defence hypothesis.

### Unipolar mania – A Challenge to the Notion of a Manic Defence.

It could be argued that the psychoanalytic notion of mania functioning as a defence against depression is threatened (or at least in need of considerable qualification) if mania can be demonstrated to exist in the absence of depression. If unipolar mania does exist as a separate and unitary entity, then an alternative or parallel aetiological mechanism to that outlined above must be sought.

Leonhard (1957) observed that mania nearly always occurs in the context of a history of depression, so that unipolar mania is rare. It is usually assumed that unipolar manic patients either are vulnerable to depressive episodes yet to occur in their futures as they have not been followed long enough (Goodwin & Jamison, 1990) or have unreported episodes of depression in their pasts (Davenport, Adland, Gold & Goodwin, 1979). That individuals could experience unremarked or unrecorded episodes of depression seems less implausible when considered in the context of the relative impact of manic versus depressive symptoms. It is generally reported that individuals with bipolar disorder and their healthy spouses cope more readily with depression than with mania

(Targum, Dibble, Davenport, & Gershon, 1979; Scott, 1995). The management challenges or interpersonal strain posed by an episode of depression could be perceived as the less memorable but welcome return to normality after the unpredictable and bizarre behavioural manifestations of mania. Similarly, where depression precedes mania, it could be viewed as the comparatively unremarkable and peaceful precursor to the subsequent storm. For example, Lam et al. (1999), describe how spouses feel more tense and anxious during manic phases, finding it hard to accommodate their partner's increased sex drive or manic sense of humour. They report that there is more disloyalty or infidelity, and less give and take in their partner's actions. Further, they suspect that their partner's manic behaviours arise out of maliciousness rather than as a result of their illness. This is largely consistent with the notion that depressed episodes have less impact and thus might be overlooked because of their juxtaposition to more florid manic episodes. Conversely, but not inconsistent with Leonhard's view (that mania is inevitably tied to depression) is the observation by Davenport et al. (1979) that family members can often remember depressive episodes even when manic patients can not. For some unipolar manic individuals, the diagnosis of depression is missed because it is lacking in any conspicuous mood change. Rather the depression is manifest in the negative symptoms of increased sleep, reduced energy, and slowed thinking (Goodwin & Jamison, 1990).

Abrams and co-workers (Abrams & Taylor, 1974; Abrams, Taylor, Hayman, & Krishna, 1979) had previously identified

methodological problems, which may have resulted in false unipolar mania diagnoses. They investigated the manner in which a history of depression was ascertained at admission to hospital. It transpired that clinicians often have to rely on patients' own (often unreliable) account of their current and past affliction, at a time when they are still severely ill. Also consistent, is the acknowledged under-reporting and poor clinical recognition of the symptoms of depression even in unipolar depression (e.g. APA, 1994, Gotlib & Hammen, 1992).

Clearly, the aetiological account of mania as a defence against underlying bipolar depression might be weakened by the existence of mania in the absence of depression. However, there are various arguments to suggest that unipolar mania is very rare. If mania does occur in the absence of depression, other than as an artifact of inadequate diagnostic procedures, it could be considered as a variant of bipolar affective disorder. This is compatible with the absence of "unipolar mania" as a diagnostic category in DSM-IV (APA, 1994) or ICD-10 (WHO, 1992), and the observation that the many authors who employ the term "pure mania" (i.e. Dilsaver, et. al., 1994; Cassidy et al., 1998) are actually referring to a bipolar-manic episode, which is devoid of obvious concomitant depressive symptoms, to distinguish it from a bipolar-mixed episode. The existence of true unipolar mania therefore remains questionable, and there is little compelling evidence that it represents a valid separate entity (Goodwin & Jamison, 1990,



Alloy, Reilly-Harrington, Fresco, Whitehouse, & Zechmeister, 1999).

Alternatively, and somewhat more radically, it could be suggested that rather than threaten the case for a manic-defence, the existence of unipolar mania is in accord with the very efficient functioning of such a strategy. Various authors (e.g., Freeman, et al., 1998; Garety & Freeman, 1999) derogate the “delusion-as-defence” mechanism as inadequate, pointing out that many deluded individuals are unable to maintain their self-esteem to normal levels, as should be the case if the defensive function of delusions is efficient. Perhaps unipolar manics, if indeed they exist, are more successful in their manic defending and hence avoid completely the distressing experience of depressive episodes. Of course the presence of concurrent symptoms of depression or low self-esteem, in unipolar mania could belie this claim to absolute efficacy, however there is a dearth of convincing evidence for unipolar mania (Goodwin & Jamison, 1990, Alloy et al., 1999) and less still for concomitant depression in this putative disorder.

In summary, unipolar mania appears to be exceedingly rare (although see Abrams & Taylor, 1974, for an alternative view). As elucidated earlier, it is clear that certain present endeavours to comprehend manic phenomena derive from, or perhaps react against, early psychoanalytical writings. The conceptualisation of mania as a defence against unpleasant psychological states, such as unacceptable id impulses or psychic pain related to object loss and its associated depression, has been successfully translated into the



language of modern cognitive psychology. Research findings perhaps with a more direct bearing on the conception of a manic-defence are reviewed in the following chapter.

## **Chapter Five.**

### **Psychological Studies of Bipolar Disorder.**

Notwithstanding clear biological roots, bipolar disorder, as expressed to the world and experienced by sufferers and their families, is exquisitely psychological. However, as previously stated, there exists relatively little research as to the psychological mechanisms that underlie this disorder. The following chapter reviews the modest number of studies, which have employed psychological measures in the investigation of bipolar disorder and its related conditions.

#### **Overt Tests of Self-Concept.**

##### **Self-Construct Measures.**

Variation in overt self-esteem between the manic and depressed bipolar manifestations is consistent with a theory of a manic-defence. Thus transparent measures, wherein the respondents are aware of the nature of the tests, generally reveal evidence of high self-esteem and high defensiveness during the manic phase and the opposite during the depressive episodes. For example, Owen and Nurcombe (1970) report a single case study involving a 14 year-old bipolar girl. They used the semantic differential technique to elicit and assess her constructs for self and

others. Her self-construct was much more negative when she was in a depressed state than when in a manic state. It changed from being “tasty”, “good”, “pleasant”, “harmless”, and “kind” in the manic phase, to being “distasteful”, “somewhat less clean”, “less good” and “very harmful” in the depressive phase. Her constructs for others, however, did not radically differ between the two types of affective episode.

In a similar vein, Ashworth, Blackburn and McPherson (1982) devised a cross-sectional study of six psychiatric groups, including depressed and manic participants, to assess state-related changes in self-esteem. They employed repertory grids with ten role titles (e.g., “self-as I am now”, “father”, “mother”, “spouse”, “friend”, “boss”, “ideal self”, etc) as elements, and thirteen constructs which included “like myself now” – “unlike myself now”. From these, various cognitive measures, including “cognitive simplicity”, “integration of self and others” and, “self-esteem” were derived. Depressed and manic patients were found to occupy extremes on all the measures, and differed significantly on most. Depressed respondents tended to produce grids of relative cognitive simplicity, and large perceived distance between self and others. The manic respondents, on the other hand, inclined to relatively confused grids, with a small self-other distance.

Of more direct relevance to this study, is the finding that bipolar mania was characterised by high self-esteem whereas bipolar depression was characterised by low self-esteem. At follow

up, self-esteem had returned to normal levels after both depressed and manic episodes.

### Measures of Depressotypic Cognitions.

The findings from investigations into depressotypic cognitive processes using transparent tests are perhaps more ambiguous. Although there exists ample evidence of variation in habitual cognitive patterns between different mood states (e.g. Weary-Bradley, 1978; Abramson, Seligman & Teasdale, 1978; Teasdale & Dent, 1987; Brewin, 1988; Williams, 1992; Bentall, 1994; Abramson et al., 1995), some recent studies report a level of stability of aberrant cognitive processes both between euphoric and depressed episodes in bipolar spectrum disorder and across diagnostic conditions.

Hollon, Kendall and Lumry's (1986) cross-sectional study involved bipolar, unipolar and substance abuse patients (all currently depressed) and non-depressed substance abuse, obsessive-compulsive and schizophrenic patients as nonaffective controls. Remitted unipolar and bipolar patients, along with medical and non-patient controls were also tested. Using two relatively overt measures; the Automatic Thoughts Questionnaire (ATQ; Hollon & Kendall, 1980) and the Dysfunctional Attitudes Scale (DAS; Weissman & Beck, 1978), Hollon and colleagues found that bipolar outpatients endorsed dysfunctional attitudes and negative automatic



thoughts to the same degree as unipolar depressives both when in the depressed and remitted states. As a study of specificity of depressotypic cognition in clinical depression, currently manic bipolar patients were not included in this investigation.

Bipolar-manic patients and individuals experiencing a euphoric mood disorder did appear, however, in recent examinations of the interaction between cognitive style and stressful life events in predicting depressive and manic symptomatology. Reilly-Harrington and colleagues devised two longitudinal investigations (Alloy, et al., 1999; Reilly-Harrington, et al., 1999), both of which put to use transparent tests of cognitive function (the DAS) and self-construct (the Self Perception Questionnaire; SPQ, Greenberg & Alloy, 1989). Their findings suggest a high level of stability of dysfunctional attitude across gross changes in mood. Individuals with Bipolar I, Bipolar II, and cyclothymia diagnoses in both depressive and manic or hypomanic periods demonstrated attitudes and self-perceptions as dysfunctional as those of the depressed unipolar cohort. Clearly, these studies (like that of Hollon et al.) did not produce unequivocal evidence of variation in bipolar patients' self construct or cognitive styles between manic, depressed and remitted states as one might have anticipated from the reports of fluctuating self-esteem related above. However, although Alloy et al. (1999) describe similarly negative cognitive patterns in respondents with dysthymia (subsyndromal depression) and cyclothymia (subsyndromal bipolar

disorder) they also note that these two cohorts displayed more dysfunctional attitudes than did respondents with hypomania (subsyndromal mania). This is largely in line with the notion that bipolar participants, if cognisant of the nature of the task, often exhibit positive self-schema and less depressotypic cognitions when manic but impoverished self precept and a more depressotypic cognitive function when depressed. Thus, in general, these findings are compatible with the psychodynamic formulation that cyclothymic and bipolar individuals' hypomanic or manic episodes are a counter-reaction to underlying depressive tendencies. Such data also implies an essential resemblance between unipolar and bipolar depressive disorders, giving further credence to the continuum concept of affective disorders.

The Reilly-Harrington team also employed the ASQ (the classic overt test of attributional style) in both investigations discussed above. The attributional data will be discussed later in this chapter in the section devoted to explanatory style. However, before that, studies using explicit tests, this time to reveal evidence of defensiveness in bipolar individuals are reviewed.

### Measures of Social Desirability.

Social desirability (a tendency to give false but socially acceptable responses) which is usually regarded as evidence of defensiveness, has been shown to be high in bipolar patients who

are either ill or in remission. Donnelly and Murphy (1973) administered a variety of psychological instruments to 30 bipolar and 29 unipolar patients hospitalised for depression. The bipolar group were found have more sociably desirable response sets compared to the unipolar group. Ratings of social desirability were also found to covary (in a negative direction) with depression. Donnelly and Murphy thus inferred a defensive relationship between the bipolar's conscious endorsement of socially desirable responses and their inaccurate self-report of depression or overall psychopathology. Similarly, Winters and Neale (1985), looked at equal sized groups ( $N = 16$ ) of remitted unipolar, remitted bipolar and non-psychiatric participants. They reported that the remitted bipolars scored higher than both other groups on tests of self-deception (unwillingness to admit undesirable and uncomfortable feelings and attitudes). These results were linked with the abnormally high self-report of social-desirability, an indicator of defensiveness, also identified in the bipolar group. Such findings of high social desirability, and hence defensiveness, in bipolar individuals are largely consistent with a manic-defence theory. Clearer evidence for this proposition can be drawn from studies, which have used less explicit tests and found indications of depression or low self-esteem during the manic phase of the condition.



### Covert Tests of Self-Concept.

On opaque tests (where respondents are not aware of the purpose of the procedure), manic participants usually produce profiles, which closely resemble those of depressed participants. The theory of a manic-defence proposes that the individual is motivated to prevent unpleasant thoughts and feelings from reaching consciousness. On this view, a person's delusional attributions are a dysfunctional mechanism to reduce discrepancies between real and ideal self-concept and hence maintain a positive perception of the self. Evidence of a discrepancy between implicit and explicit self-representation in manic individuals would thus provide strong support for the manic-defence hypothesis. Attempts to demonstrate such a disparity would need to access the impoverished self-esteem hidden beneath the putative defence and must therefore involve somewhat clandestine means, in the shape of defence-penetrating measures. Such non-obvious tests would require the subtle capability to negate the psychological mechanisms, about which respondents appear to be unaware and may wish to remain ignorant, but which they are unconsciously (or consciously) evoking to maintain an apparently hearty equilibrium of self-esteem. One such test, the Emotional Stroop Task, is thought to measure preconscious or automatic cognitive processes (Garety & Freeman, 1999) and has been described as unaffected by psychological defences (Giles & Cairns, 1989). Frequently employed to measure covert attentional bias in unipolar depressed



patients, this test (described in more detail later), signals the valence of words by slowed colour-naming of emotionally salient as opposed to neutral words (Brewin, 1988). Since there is good evidence for the Stroop effect even when participants are not consciously aware of the material they have been shown (Williams, Mathews & MacLeod, 1996), the emotional Stroop task can be regarded as a valid measure of an individual's covert concerns (Garety & Freeman, 1999).

Using such a test, Bentall and Thompson (1990) were able to provide modest evidence of covert poor self-concept in a bipolar analogue study. They found evidence of a Stroop effect in students selected on the basis of high scores on Eckblad and Chapman's (1986) Hypomanic Personality Scale. Hypomanic students demonstrated interference with colour-naming for depression-related words (e.g. "misery" or "defeat") rather than euphoria related words (e.g. "care-free" or "amazing"). This selective bias of information processing was taken to reflect an underlying emotional salience of depressive rather than manic themes. This result has been replicated by French, Richards and Scholfield (1996) who controlled for the known effects of anxiety on Stroop performance. They support the explanation by Bentall and Thompson (1990), that a psychological defence might be (at least partially) effective in terms of preventing awareness of depressive tendencies within the hypomanic individual. This data further implies that these pervasive tendencies remain at a non-conscious level and have the potential to

evade the defence, as occurred during Stroop performance. However, as analogue studies, the generalisability of these Stroop findings are limited. Nonetheless they are broadly consistent with emotional Stroop findings for depressed patients (Gotlib & Hammen, 1992), and suggest that depression-related words are of more emotional valence than are euphoria-related words to hypomanic individuals.

A far more impressive demonstration of depression-like responding on opaque or non-obvious measures was reported by Winters and Neale (1985). But first, in order to fully understand this demonstration it is necessary to know something of “attribution theory”, and its application to psychopathology.

### Social Attribution Theory.

Social attribution theory, that is, the study of lay causal explanations, considers the attributions made by individuals to explain their own and other people’s behaviour (Hewstone, 1989). This perspective, has already been applied to the examination of cognitive biases in patients with paranoia (see Bentall & Kinderman, 1998; Garety & Freeman, 1999, for critical reviews) and has been found to be of particular relevance since many of their abnormal beliefs (e.g. persecutory delusions) are primarily concerned with the patient’s relative positions in the social universe (Bentall, 1994). Given that the delusions of grandeur, which typify

manic episodes, are also so concerned, attribution theory is likely to prove of similar relevance to the understanding of bipolar disorder. It has also been pointed out that causal attributions or explanations are essential to everyday life, and utterances containing or implying the word “because” occur in every hundred or so words of speech. Thus many delusional beliefs amount to abnormal attributions; that is, dysfunctional attempts to explain every day events (Bentall & Kinderman, 1998). Attributional theory, however, has been most widely applied to accounts of unipolar depression and there follows a review of the attributional research in this area, with reference too, to the field of persecutory delusions.

### Attributional Biases in Unipolar Depression.

In their reformulation of the “learned helplessness” model of depression, Abramson et al., (1978) postulate that individuals with a certain habitual style of explaining events in their lives are particularly vulnerable to developing depression. People with this attributional style have learned both that previous events were uncontrollable, and to expect future outcomes to be equally beyond their control. The model predicted that when confronted with negative life events, depressed people tend to make “internal”, “stable” and “global” attributions about negative outcomes. For example, an unemployed person who is currently depressed would typically;



- 1) fail to blame the poor job market and attribute their inability to find work entirely to some characteristic within themselves (internality),
- 2) believe that they will remain unemployed indefinitely (stability), and
- 3) believe that this area of failure will affect other aspects of their life (globality).

Abramson et al. (1978) also predicted that those prone to depression would, to a lesser extent, attribute positive outcomes to external, specific and unstable causes. These patterns of attributions were hypothesised to have a causal role in the aetiology of depression. Thus according to the attributional reformulation of the learned helplessness model, depressive episodes are precipitated by negative events. These trigger the characteristic expectations that future negative events will also be beyond control and will be attributable to internal, global and stable factors. There are various criticisms of this model, which will be referred to later.

### Attributional Studies of Individuals with Unipolar Depression.

One of the most commonly used measures of hypothetical attributions is the Attributional Style Questionnaire (ASQ; Peterson, Semmel, von Baeyer, Abramson, Metalsky, & Seligman, 1982), developed in part to test Abramson et al.'s (1978) hypotheses. This measure briefly describes a positive or negative outcome involving the participant, who is then required to record the one major cause for this event and to rate the (self-generated)



cause in terms of the three dimensions; internality, stability and globalness. Studies using the ASQ have consistently shown a modest association between this kind of attributional (or explanatory) style, and depressed mood (Brewin, 1985, Sweeney, Anderson & Bailey, 1986). However, subsequent revisions of the ASQ have focused more on the globalness and stability dimensions, than the internality–externality dimension (Abramson, Metalsky & Alloy (1989), and other authors have noted that the pessimistic attributional style characteristic of depressed people is possibly due to low self-esteem rather than depression (Tennen & Herzemberger, 1987; Tennen, Herzemberger & Nelson, 1987). Moreover, research with depressed adults has not consistently supported the contention that attributional style is causal and predates the depression (Brewin, 1988; Robins & Hayes, 1995). It is now also clear that this style reflects even-handedness of depressed patients who do not show the “self-serving bias” demonstrated by normal participants (Musson & Alloy, 1988). To put this in context, a self-serving bias is the systematic tendency to accept greater personal responsibility for positive events than for negative ones, as described by Zuckerman (1979) in relation to the normal inclination to ascribe successful performances to personal skill or effort rather than circumstantial or external causations. Hewstone (1989) suggested that there were, in fact, two different but compatible biases. One he called the “self-enhancing” bias whereby successful experiences are attributed to internal causes. The other, he referred to as the “self-protecting” bias, being the tendency to ascribe failure to external

rather than personal causes (i.e. blame the tool, the job, or workmate). Thus a degree of self-serving bias (however inaccurate) is psychologically healthy as it is thought to be a normal mechanism for regulating self-esteem and coping with threats. This view is consistent with the findings of Weary-Bradley (1978) who suggested that self-protecting attributions about failure are mediated by, and serve to reduce, high levels of negative affect. Thus, Alloy and Abramson (1979) concluded that depressed patients who were unable to maintain self-esteem using a self-serving bias, were “sadder but wiser” and other authors refer to the absence of a self serving bias as “depressive realism”(c.f. Ackermann & DeRubeis, 1991). See Brewin, (1985) and Buchanan and Seligman, (1995) for more comprehensive reviews of explanatory style and depression. In summary; previous ASQ studies have shown that whereas normal subjects show a self-serving bias in their habitual explanations of events, depressed people show a self-depreciating bias (Abramson, Seligman, & Teasdale, 1978; Robins & Hayes, 1995) with apparently excessive insight into life and an over-emphasis of real or perceived problems and obstacles.

### Attributional Studies of Individuals with Bipolar Depression.

As alluded to earlier, a limited number of investigators have already examined overt attributions in bipolar and analogue cohorts, unfortunately with somewhat conflicting results. Tracy, Bauwens, Martin, Pardoën, and Mendlewicz (1992) administered the ASQ to

23 remitted recurring unipolar depressives and 26 remitted bipolar patients, all with normothymic range depression scores. They found specific cognitive biases in the unipolars, who attributed negative events to primarily stable causes. No such attributional bias was found for the remitted bipolars who more resembled the healthy controls. This result reflects a clear difference in the attributional style of unipolar and bipolar patients on a transparent test, since bipolar patients alone were able to employ protective attributional biases. As such it this study is in line with the proposition under consideration, although it proffers only limited support given that both patient groups were tested when in remission.

However, Alloy et al. (1999; one of the Reilly-Harrington studies referred to earlier) used the ASQ to examine cognitive style in symptomatic, albeit subsyndromal bipolar conditions. Their cross-sectional study found that their 13 cyclothymic (subsyndromal bipolars) and 8 dysthymic participants (subsyndromal depressives) both had more “depressogenic” attributional styles for negative events than did their 10 hypomanic or 12 healthy participants. That the hypomanics demonstrated a self-protective explanatory bias similar to non-depressed controls on an overt attributional style test is consistent with the performance of the remitted bipolar participants reported by Tracey et al. (1992). Less consistent, however is the potentially counter-intuitive demonstration (according to the manic-defence theory) of a pessimistic attributional response by the cyclothymics (who



display both hypomanic and depressive states) on the same overt measure. Unfortunately although this study specified that their clinical groups were actively symptomatic at time of testing, they did not indicate whether their cyclothymic participants were undergoing euthymic or dysthymic episodes. Of course, if the latter were the case, then this finding fits the manic-defence line quite neatly, and has clear implications for the state or trait debate.

More clearly concordant with proposition under investigation, are the attributional performances of clinically depressed undergraduates in the second study referred to earlier, once more using the ASQ. In this, Reilly-Harrington et al., (1999), noted equal levels of “depressotypic” attributional style in unipolar and bipolar depressions and cyclothymia, an observation compatible with their previous findings (Alloy et al., 1999). Further, it was also apparent that the attributional style and self-referent processing of bipolar participants varied over time according to whether or not they were in a depressed episode, a finding which seems to support the manic-defence theory. Unfortunately, Reilly-Harrington et al. do not specify whether the non-depressed bipolar respondents were manic or remitted at second testing. Nor is it wholly clear in what direction these (unremarked) shifts in explanatory style and self-precept occur. Interpretation of the Alloy/Reilly-Harrington team data requires some caution in the context of the proposal currently under contention. Since their primary objective was an exploration into



possible predictors of affective symptomatology their attributional data does not lend itself to simple comparison with other attribution studies. As the authors themselves point out (Reilly-Harrington et al., 1999), the experimental participants of both studies, were essentially an analogue cohort (given that one criteria for Bipolar I manic episode is severe disruption of everyday activities sufficient to warrant hospitalisation). All psychiatric groups were drawn from a young student sample, who despite apparently meeting diagnostic criteria for bipolar, unipolar or subsyndromal conditions, had invariably received no previous or current treatment for their mood disorders.

Taken together, the ASQ data from bipolar studies to date, are largely consistent with earlier findings of bipolar defensiveness (Donnelly & Murphy, 1973; Winters & Neale, 1985) and with the contention that, at some level, bipolar-manic individuals experience an impoverished affect which is largely incongruous with their behavioural presentation. However, these results fail to provide strong evidence for a manic defence, since they do not provide an overt – covert attributional style comparison and subjects were not re-tested when symptomatic. That some remitted bipolar and hypomanics patients demonstrated an enhanced self-serving bias, is also in accord with the proposition that bipolar individuals have a mechanism by which to hide latent feelings of low self-esteem when the nature of the measure employed is explicit. Though the fact that depressogenic attributional styles as dysfunctional as those

observed in the unipolar control participants, could be readily elicited through non-covert means, does perhaps obviate the need for defence penetrating tests to access a camouflaged or underlying affective state.

Interestingly this self-serving attributional bias, demonstrated as absent in depressed people but believed common in the general population, has been found to be exaggerated in paranoid participants, as detailed below.

### Attributional Studies of Individuals with Paranoia.

Similarities between bipolar and paranoid individuals have already been suggested. The parallels between delusions of grandiosity which typify episodes of bipolar mania (but also occur in paranoia) and delusions of persecution which are more characteristic of paranoia, but also occur in mixed and manic bipolar states, are obvious. There is also an inherent grandiosity in the certainty that you are sufficiently consequential to be persecuted! In contrast to the depression literature, it is the internality dimension which is of chief importance in the research on persecutory delusions. This reflects the proposition of Bentall and his associates (Bentall, Kaney, & Dewey, 1991; Bentall, Kinderman & Kaney, 1994; Kinderman & Bentall 1996) that people with paranoia construct persecutory delusions in an attempt to maintain positive self-regard or integrated self-concept, and

avoid conscious acknowledgement of discrepancies between how they perceive themselves and how they would like to be. Clearly this “persecutory delusions-as-defence” model shares much with the “bipolar manic-defence” model presently under consideration, and has the same derivation; (i.e. Zigler and Glick’s 1988 notion of paranoia and mania as camouflaged forms of depression). Bentall argues that externalising causal attributions (persecutory delusions) are evoked in response to adverse events, which would otherwise increase the accessibility of underlying negative self-representations. In an early ASQ study, Kaney and Bentall (1989) found that people with persecutory delusions made excessively internal attributions for positive events and abnormally external attributions for negative events in comparison with depressed or normal controls. This finding was substantially replicated by Candido and Romney (1990). Other ASQ studies have provided further evidence that deluded individuals make abnormally external attributions for negative events (e.g. Lyon, Bentall & Kaney, 1994; Fear, Sharp & Healy, 1996; Sharp, Fear & Healy, 1997). The two studies which investigated the specificity of attributional style produced only mixed results but suggested that the externalising bias for negative events may be specific to both persecutory and grandiose delusions (Fear et al., 1996; Sharp et al., 1997). However, as Garety and Freeman (1999) remark, the evidence for an internalising bias for good events in paranoid patients is less strong and has not been well replicated in all studies. In order to understand Garety and Freeman’s criticisms of Bentall et. al.’s



theory, it is first necessary to elucidate the delusions-as-defence model a little further.

### Elaboration of the Delusions-as-Defence Model.

In what amounts to a more specific version of Zigler and Glick's (1988) account, Bentall and colleagues have elaborated their delusions-as-defence ideas, with reference to Higgins' (1987) self-discrepancies theory. They argue that paranoid patients have latent negative self-representations not dissimilar to the more explicitly negative schemata of depressed patients. These can become primed by threatening events, leading to discrepancies between the self-representation and the self-ideal, at which point external attributions for the threat event are elicited. These attributions are self-protecting as they diminish the patient's consciousness of discrepancies between the self and self-ideals. However, this mechanism results in the activation of schemata that represent threats, or persecution, from others. (Bentall, Kinderman, & Kaney, 1994; Kinderman & Bentall, 1997).

This actual-own, ideal-own discrepancy, whilst not identical to the complex construct of low self-esteem, is conceptually similar, and has also been challenged. Garety, Freeman and colleagues (1999) suggest that there is insufficient evidence to accept a defence model (based on attributional style and self-discrepancies) for all deluded patients. Instead, they champion a multi-factorial approach which



implicates a reasoning or data gathering bias, which implicates deficits in attributional style, perceptual processing, and meta-representation or theory of mind (Freeman, et al., 1998). Just as the “delusions-as-defence” model of paranoia and the “manic-defence” model of bipolar disorder have common theoretical underpinnings (attributional biases and self-schema discrepancies); so the criticisms leveled against one might apply to the other. However, review of the available evidence in the paranoia debate suggests considerable overlap across the different approaches, which are themselves far from contradictory. It has thus been suggested that the different processes purported by the different models might co-occur or interact (Garety & Freeman, 1999). Further, different processes (e.g. a defence process as opposed to a reasoning deficit) might apply to different delusional subgroups, to different sorts of delusions (persecutory or grandiose), or to “different” psychopathologies (the grandiose delusions of mania as opposed to those evident in paranoia). To return to the exploration of attributional deficits, the phenomenon accepted as implicated in the aetiology of delusions, albeit though variously weighted by opposing camps, it may be useful to consider a different means by which researchers have tapped into the realm of persistent prejudices of explanatory style.

### Contingency Judgement Measures.

Evidence of an externalising attribution bias has not been limited to use of the Attributional Style Questionnaire. Kancy and Bentall (1992) employed a contingency judgement task in which they asked paranoid subjects to estimate their degree of control over winning or losing points on a pre-programmed computer game. Interestingly, all respondents mistakenly believed they had some control over the outcome (i.e. could win on merit), and showed some degree of attributional change with regards to their apparent success or failure, the normal controls demonstrated a robust self-serving bias, claiming more control over win conditions. This was vastly exaggerated in the deluded subjects whose estimates of control were excessively high when winning points and excessively low when losing points. The depressed subjects, however, had a more realistic view as to their level of control for both win and lose situations (a replication of a finding by Alloy & Abramson, 1979). Results from this and other attributional studies could help explain the relationship between grandiose and persecutory delusions and their co-occurrence in the same individual. That is, taking credit for positive outcomes and blaming other people (or circumstances) for negative outcomes could be thought of as two compatible cognitive traits driven by the same underlying psychopathology. This proposition would fit nicely with Hewstone's ideas, since delusions of grandeur could be viewed as associated with a self-enhancing

bias and delusions of persecution could be associated with a self-protecting bias (Kaney & Bentall, 1989).

### An Opaque Test of Attributional Style.

The attributional data above is in accord with the hypothesis that persecutory delusions reflect self-enhancing biases, which in turn reflects a sustained defence against low self-esteem. This explanation is consistent with Zigler and Glick's (1988) ideas about the defensive function of paranoia and mania. The history of attempts to test for the presence of defensive processes has not been encouraging and only limited experimental investigation undertaken. Indeed Zigler and Glick's theoretical paper was based solely on clinical observations. One way to test this might be to show that, despite self-reports of high self-esteem, manic patients do experience extreme feelings of low self-esteem. This could be done by accessing the social attributions made about certain events using a "defence penetrating" test, which is able to ensure that participants remain unaware of what is being tested as any direct approach would automatically trigger their defensive attributions.

One such opaque test of attributional style has already been invented. The Pragmatic Inference Task (PIT; Winters & Neale, 1985) was developed from the ASQ (described above) and presented as a non-specific test of memory. PIT participants are requested to listen to self-referent stories with success and failure



outcomes and are then required to complete a series of multiple choice questions. One question from each story required them to make attributional inferences by apparently “remembering” either an internal or external cause for the outcomes described. Winters and Neale (1988) used the PIT in an attempt to provide evidence of hidden low self-esteem in individuals with a history of mania, and thus test the traditional psychodynamic account of the defensive function of grandiose delusions. Consistent with the findings of the studies already described, their remitted manic respondents scored high on explicit measures of self-esteem and social-desirability. However, like the depressed control participants, they suggested internal causes for negative outcomes and external causes for positive outcomes. Thus, although not immediately apparent, their manic patients possessed an attributional style similar to that of depressives. As this was not wholly consistent with their high self-esteem (as reflected on the self-rated measures), the Pragmatic Inference Task was considered to be defence penetrating. These findings could be taken as initial experimental support of the hypothesis that bipolar mania, and associated attributional style, may serve to reduce feelings of low self-esteem in some manic patients.

However, as Garety and Freeman (1999), rightly point out, it is somewhat of a leap to directly equate a depressive attributional style with a cognitive schema of low self-esteem. This criticism being in line with the observation by Tennen and colleagues



(Tennen & Herzemberger, 1987; Tennen, Herzemberger & Nelson, 1987) that poor self-esteem rather than depression is associated with the deficits of explanatory style frequently reported in depressed individuals. Unfortunately, Winters and Neale did not compare remitted manic performances on the PIT to those on a traditional measure of attributional style. Nor were remitted manics re-tested when their symptoms returned. Nevertheless, the discrepancy in reports of remitted bipolars' attributional style as measured using transparent (Tracy, et al., 1992) and opaque tests (Winters & Neale, 1985), both lends support to, and is suggestive of a more elegant research design better to test the hypothesis under consideration. More recently the present author has used the PIT with paranoid participants who were also found to perform like depressives on this measure (Lyon, et al., 1994). This finding is consistent with the hypothesis that both paranoia and mania are manifestations of defences against low-self esteem and depression (Zigler & Glick, 1988).

### Self-Referent Encoding Tasks.

Although attentional bias has been most widely demonstrated using emotional Stroop tests, motivational factors associated with information processing have also been demonstrated by other means. For example, depressed patients display enhanced recall of negative-affect laden material (Williams, et. al., 1988). This phenomena has been employed in studies of the role of self-

schemata in depression, and in particular vulnerability to depression (as suggested by Beck's cognitive model; Beck, 1967). Building on the association between information processing and affect, the schema concept contributes the idea of enduring structures by which information is attended to, interpreted, stored and retrieved. Self-referent encoding tasks require participants to endorse positive and negative trait words as descriptive or non-descriptive of the self. They are then required to recall as many of the trait words as possible in a surprise recall test. A number of studies, using these techniques, have shown that depressed patients, when required to recall trait words previously presented as part of a personality test, tend to recall more negative trait words. The obverse being true for normal controls (Dent & Teasdale, 1988; Hammen, Marks, Mayall, & de Mayo, 1985; Williams Healy, Teasdale, White & Paykel, 1990). This finding is usually interpreted in terms of the self-referent encoding effect. That is the general tendency, shown by normal subjects, for superior recall of words which have been encoded in relation to self-schema (Rogers, Kuiper & Kirker, 1977), although the exact mechanism responsible for this effect has been the subject of some controversy (Higgins, 1987; Klein & Lofthouse, 1988; Rudolph, 1993).

Tests of self-schema responding have also been applied to individuals with persecutory delusions. Bentall and Kaney, (1996) found that paranoid patients, like normal controls but unlike depressed subjects, endorsed as true of themselves positive rather

than negative traits. However, on the incidental recall of trait adjectives presented during the encoding phase of the task, the deluded subjects' recall of items did not reflect their pattern of endorsement but was similar to that of the depressed control group. This "endorsement-recall" discrepancy was taken as offering equivocal support for the defensive model of delusions. It also suggests that the Self-Referent Incidental Recall Task (SRIRT) employed by Bentall and Kaney may be acting as an indirect or opaque measure capable of tapping underlying abnormalities of self-representation. Such a test might usefully be applied to bipolar patients, a group also known to be highly defensive. Indeed the suggestion to use an incidental recall measure to access latent self-schema bias was initially made by Mark Williams some eleven years ago (Healy & Williams, 1989).

### **Broad Hypotheses.**

The findings outlined in chapter four clearly indicate the value of studying psychological factors in bipolar disorder and suggest the following broad hypotheses.

- 1) Low self-esteem is an important aspect of the psychopathology in both bipolar mania and bipolar depression.
- 2) In mania the low self-esteem is hidden by protective attributional biases.
- 3) Opaque or defence penetrating tests should therefore measure an underlying bipolar trait irrespective of symptomatology.

The following chapter shall outline these hypotheses in detail and thus present the rationale for the present research.



## Chapter Six.

### Rationale for the Present Study.

The present study was designed further to test predictions derived from the hypothesis of a manic-defence. The ideal research design for studies of bipolar disorder would be comparison of manic, depressed, and normal (remitted) states, using subjects as their own controls. However, one implication of the cycle length literature, reviewed above, is that longitudinal observations would be impracticable within the time constraints of this study. A cross-sectional design was thus employed to investigate the psychological processes underlying the symptoms of bipolar disorder in both the manic and depressive episodes of the disorder. It was predicted that cognitive biases known to be present in unipolar depression are present in both the manic and the depressive bipolar phases, but that these will only be detectable by “defence penetrating” measures in the manic phase.

Measures that had already been shown to be abnormal in unipolar depressed patients were administered to actively manic bipolar participants, currently depressed bipolar participants, and normal controls. Three of these measures, an emotional Stroop test, a traditional test of attributional style and the Pragmatic Inference Task, had previously been administered to either hypomanic students or remitted bipolar patients as detailed above.

### Emotional Stroop Task (EST) Prediction.

On an emotional Stroop test, (accepted as a covert measure of attentional bias, Garety & Freeman, 1999), it was predicted that both the currently manic and currently depressed bipolar participants would demonstrate slowed colour-naming for depression-related words but not for elation-related words. Such a finding would indicate the emotional salience of depression words to both groups, and reflect a level of covert low self-esteem in the manic group. Previous studies have reported this effect in analogue populations (Bentall, & Thompson, 1990; French, et al., 1996).

### Attributional Style Questionnaire (ASQ p.f.) Prediction.

Subjects also completed a traditional questionnaire measure of attributional style similar to the Attributional Style Questionnaire (ASQ) developed by Peterson et al. (1982). Given existing evidence that manic patients are abnormally defensive (Donnelly & Murphy, 1973; Winters & Neale, 1985), it was predicted that currently manic bipolar patients would respond like normal subjects and demonstrate a self-serving bias (attribute positive events to internal causes and negative events to external causes) on this explicit measure. Such a result would be consistent with the previous finding by Tracy et al. (1992) for remitted bipolar depressives. Depressed bipolars were predicted to demonstrate the self-depreciating bias (attributing negative events to causes that are internal, global, and stable) as already demonstrated in unipolar depressives (Abramson, et. al., 1978). Such a result is consistent

with the idea that in mania, low self-esteem is concealed by protective attributional biases.

### Pragmatic Inference Task (PIT) Prediction.

On the opaque PIT however, it was anticipated that both the currently manic and the currently depressed bipolar participants would attribute negative events to internal causes, as previously found for remitted manic patients by Winters and Neale (1985) and for persecutory patients by Lyon et al., (1994). This result would suggest that the PIT is an effective means of penetrating the manic defence.

### ASQ (p.f) – PIT Discrepancy Prediction.

Comparisons of performance on a parallel version of the ASQ and PIT by the same manic participant could be utilised to detect an overt-covert self-esteem discrepancy (the original ASQ cannot be used concurrently with the PIT for methodological reasons outlined later). Such a design was first devised by the present author in a study to investigate similar abnormalities of social reasoning in patients with persecutory delusions (Lyon et. al., 1994). In that study, deluded patients showed a reverse of internality, attributing negative outcomes to external causes on the transparent (parallel form) ASQ but, on the more opaque PIT, were found to attribute negative outcomes to internal causes, thus



showing a cognitive style resembling that of the depressed group. In the present research therefore, it was predicted that the currently manic patients would undergo a radical shift in attributional style from a normal optimistic bias on the overt ASQ, to a self-denigratory bias similar to that demonstrated by the depressed bipolar group on the PIT. No such shift in attributional style was predicted for the bipolar-depressed or normal control group.

### Self-Referent Incidental Recall Test (SRIRT) Prediction.

In addition to the above measures, respondents completed a self-referent encoding task similar to that employed by Kaney and Bentall (1996) and previous depression authors. It was predicted that, like the normal controls, the manic subjects would endorse more success trait words as true of self, reflecting their apparently positive view of themselves. In contrast, bipolar-depressed subjects were expected to endorse more failure and fewer success words than normal subjects, as observed by previous authors for unipolar depressives (Dent & Teasdale, 1988; Hammen et. al., 1985; Williams et. al., 1990). It was also predicted that, when given the subsequent incidental recall task, the manic subjects would perform in a manner similar to the bipolar-depressed subjects and show evidence of an underlying negative self-schema. That is, both bipolar groups recalling more failure than success trait adjectives. This “endorsement-recall” discrepancy (seen previously in persecutory deluded subjects) was anticipated in the bipolar-manic



group alone. The normal subjects were expected to both endorse and recall far more positive than negative words.

Having explained in detail the rationale for the present study, the following chapter will provide precise methodological and procedural detail.

## METHOD AND RESULTS SECTION.

### Chapter Seven.

#### Method.

##### Participants.

Given the impracticalities of longitudinal observations, the present study employed a cross-sectional group comparison design. The following three groups were recruited: bipolar-manic, bipolar-depressed and healthy controls, each comprising 15 participants. Groups were approximately matched for age and preferred language (English or Welsh), and for IQ as estimated by the restandardised National Adult Reading Test (NART; Nelson, 1992). The NART is a brief measure of verbal intelligence based on the correct pronunciation of irregularly spelled English words (e.g. “chord”, “gouge”, “campanile”), which correlates well with other measures of intelligence (Crawford, Parker, Stewart, Beeson, & DeLacey, 1989). A one-way analysis of variance (ANOVA) confirmed that the groups were comparable for both age, ( $F [2, 42] = 1.18, p = 0.32$ ), and estimated IQ, ( $F [2, 42] = 0.44, p = 0.65$ ). Similarly, no significant between-group differences were found for gender ( $\chi^2 = 0.19, d. f. = 2, p = 0.9$ ) or language ( $\chi^2 = 1.32, d. f. = 2, p = 0.52$ ).

##### Inclusion and Exclusion Criteria.

Clinical participants were recruited with the co-operation of mental health colleagues (see appendix 3; recruitment flyer), and

lithium clinic records. Diagnostic assignment of the clinical groups was determined by the author. This was subsequent to consultation with medical staff and careful review of clinical notes to evaluate the past course of the illness, and ascertain the likelihood of an appropriate bipolar diagnosis. Potential participants were excluded (from clinical or control groups) if aged below 18 years or above 75 years, or if there was evidence of substance abuse or alcohol dependence within the previous six months. Individuals were also excluded from the psychiatric groups if there was any indication of organic brain syndrome, mental retardation, or physical impairment that would interfere with completing the measures. Patients were included in the bipolar-manic or bipolar-depressed groups only if they met full DSM-IV criteria (as outlined in the introduction and specified below). Thus currently Bipolar II or cyclothymic patients were excluded from the experimental or psychiatric control groups. Final selection of the clinical groups followed the administration of the appropriate sections of the Diagnostic Interview Schedule (DIS, version III, Helzer et al, 1985, see appendix 4) to verify diagnostic eligibility.

### Bipolar-Manic Group.

Of the 28 patients approached as potential bipolar-manic participants, six were not recruited as they failed to meet the above inclusion criteria (according to their DIS responses) and five refused to consent to testing or were otherwise non-compliant.

As it had been anticipated that many of the bipolar-manic participants might be difficult to test, this was not attempted when their symptoms were at their peak. As already noted, Carlson and Goodwin (1973) have suggested that manic episodes have three relatively distinct phases through which patients pass as their symptoms wax and wane. The bipolar-manic participants were therefore monitored until they returned to the first of these stages (characterised by lability of affect, euphoria, grandiosity, overconfidence, increased speech, and psychomotor activity) after the zenith of an episode, whereupon testing was carried out. However, a further two proved to be “untestable” and the author was unable to re-recruit them as their symptoms subsided.

The experimental group therefore consisted of ten female and five male patients who met DSM-IV (APA, 1994a) criteria for bipolar disorder-manic episode. Twelve of the bipolar-manic group were inpatients on acute psychiatric wards. The remaining three were outpatients, initially approached whilst in hospital but recently discharged and tested at their local day hospital or community mental health team (CMHT) base. Eight of the hospitalised patients were compulsorily detained under a section of the Mental Health Act 1987 (see appendix 1 for details). The mean age of the manic group was 47.87 years ( $SD = 18.06$  years) and their mean estimated IQ was 107.53 ( $SD = 10.41$ ). Eight of this group spoke English as their first language and the remainder spoke Welsh. No control was exercised over psychopharmacotherapy prescription for any participant. Thirteen of the bipolar-manic group were in receipt of



mood stabilizing medication (carbamazapine or lithium carbonate) and five, including the two who were not in receipt of mood stabilising drugs, were prescribed antipsychotic medication. Further details of participants' diagnoses, prescribed medications, marital and employment status can be found in appendices one and two.

### Bipolar-Depressed Group.

Fifteen bipolar-depressed patients were approached and all 15 were recruited. Thus, the psychiatric control group consisted of nine female and six male patients who met DSM-IV criteria for bipolar disorder-depressive episode. These participants included six inpatients interviewed on an acute psychiatric ward and nine outpatients. Of those not in hospital, four were tested at their homes, one at a supported bed unit, three at a day hospital, and the other at the author's CMHT offices (at their convenience). Of the six hospitalised participants, two were detained under MHA section. The bipolar-depressed group had a mean age of 44.47 years (SD = 13.16 years) and an estimated mean IQ of 103.93 (SD = 11.42). Eleven of the bipolar-depressed participants preferred to speak English and four preferred Welsh. Fourteen were currently in receipt of mood stabilising medication, four were in receipt of antipsychotic medication, and eight (including the patient not in receipt of mood stabilising medication) were prescribed antidepressant medication.

### Non-Psychiatric Control Group.

Finally, the normal control group consisted of ten female and five male participants who had been recruited via informal contacts with no present or previous history of psychiatric disturbance requiring treatment. Their mean age was 46.47 years ( $SD = 14.47$  years) and their estimated mean IQ was 106.00 ( $SD = 10.41$ ). Of the normal controls, nine spoke English as their preferred language and the remainder spoke Welsh. Healthy controls were tested either in their homes or at their places place of work (again at their convenience).

### Measures.

In addition to the NART, six measures were administered to subjects across the three groups. Two questionnaires, the Rosenberg Self-Esteem Questionnaire (RSEQ; Rosenberg, 1965) and the Beck Depression Inventory (BDI; Beck, Ward, Mendleson, Mock, & Erbaugh, 1961), were employed to determine current psychological functioning. Four dependent measures consisting of two attribution questionnaires and two measures of information processing of self-referent material were also administered. The attribution questionnaires administered were the Attributional Style Questionnaire (parallel form, ASQpf; Lyon, et. al., 1994) and the Pragmatic Inference Task (PIT; Winters & Neale, 1988). The information processing tasks employed were an Emotional Stroop

Task (EST) and a Self-Referent Incidental Recall Task (SRIRT; Dent & Teasdale, 1988). Copies of all measures employed with, where appropriate, additional administration or scoring details, are included as appendices (5 to 11).

### The Rosenberg Self-Esteem Questionnaire (RSEQ).

The RSEQ (Rosenberg, 1965) was given to all participants as a uni-dimensional index of overtly reported global self-esteem. It is a self-report measure consisting of ten statements, which the subject is asked to rate on four-point scales as follows, “strongly agree”, “agree”, “disagree”, and “strongly disagree”. Five of the statements are negative (e.g. “I certainly feel useless at times”) and five positive (e.g. “I am able to do most things as well as most other people”), in random order to prevent an acquiescence set. Positive statements are scored; “strongly agree” (2), “agree” (1), and negative items are scored “disagree” (1), and “strongly disagree” (2). Other response combinations (e.g. strong/disagreement with positive items or strong/agreement with negative items) are scored zero. Thus, a high RSEQ score indicates high self-reported self-esteem and vice-versa.

The RSEQ has demonstrated adequate psychometric properties. Specifically Dick and Shepherd (1994) report that self-esteem as measured by the RSEQ correlates significantly with levels of psychological distress as measured by the General health Questionnaire (GHQ-28, Goldberg & Hillier, 1979).



### The Beck Depression Inventory (BDI).

The BDI (Beck, Ward, Mendleson, Mock, & Erbaugh, 1961) is a 21-item multiple-choice scale that has been widely used to measure overt depressive symptomatology in previous studies. Each item describes a specific symptom of depression. Subsequent to various revisions of the original inventory, all BDI items consist of four self-evaluative statements assigned a score from zero to three. These are rank ordered and weighted to reflect increasing severity of symptom. The test is self-administered and participants are required to indicate the statement corresponding to the symptom level that best describes their experiences over the previous week. Thus high BDI scores indicate high self-reported depressive symptomatology and vice versa. In previously published research, this scale has been shown to have good internal reliability and to be valid for use with both clinical (Williams, Barlow, & Agras, 1972) and non-clinical (Blumberry, Oliver & McClure, 1978) populations. The present investigation was designed before the advent of ready access to the revised Beck Depression Inventory (BDI-II), the psychometric properties of which have yet to be clearly established (Beck & Beamesderfer, 1974; Beck, Steer, & Garbin, 1988).



### The Parallel Form Attributional Style Questionnaire (ASQpf).

The ASQpf was used to access the participant's overt expression of attributional style. It explicitly requires subjects to state causes for hypothetical successes and failures and thus allows self-presentation biases. The ASQpf is a parallel form of Peterson et. al.'s original Attributional Style Questionnaire (ASQ, 1982), designed by Lyon, Kaney and Bentall (1994) specifically for use alongside the Pragmatic Inference Task (PIT, see below). The original ASQ could not be employed concurrently, as many of the PIT items were derived directly from the ASQ, and thus would be too similar. Like the original ASQ, the ASQpf is a 12-item self-report inventory, which explicitly requires participants to generate possible causes to positive and negative, hypothetical events involving themselves. The six positive items include "You pass somebody who smiles at you" and "Going on a journey to a strange place, you get there very quickly". The six negative items include "Your steady romantic relationship ends" and "You experience a major personal injury". After generating causes for each event, participants are required to self-rate their causal statements on three, 7 point scales for internality versus externality, stability versus instability, and globality versus specificity. As already outlined, the internality dimension indicates to what extent events are attributed to self or to other people or circumstances. The stability sub-scale pertains to the degree to which events are likely

to be present in the future. Finally, the globality domain relates to the level to which causes are likely to influence a wide range of events in addition to those specifically mentioned in the questionnaire. Of particular interest to this study is the ASQpf internality – externality sub-scale, from which a self-serving bias score can be calculated by subtracting internality scores for negative events from internality scores for positive events, as described by Kaney and Bentall (1989).

Although the internal reliabilities for the original ASQ sub-scales, have been found to be poor, its very extensive use in research on depression (Robins & Hayes, 1994) and paranoia (Bentall, Kinderman, & Kaney, 1994) reflects the fact that it taps an important psychological domain, and there are few alternative scales available (Rehm, 1988; Reivich, 1995; Tennen & Herzemberger, 1985). Unfortunately, this study was designed prior to the development of the Internal, Personal and Situational Attributions Questionnaire (IPSAQ), which has superior psychometric properties to the ASQ or ASQpf (Kinderman & Bentall, 1996b, 1997).

Lyon et. al., (1994) reported that the sub-scales of the ASQpf correlated moderately with those of the ASQ in a mixed group of 13 muscular dystrophy, 15 depressed and 15 chronic fatigue patients, plus 8 normal subjects (Pearson correlations varying between 0.21 and 0.64). Of the six sub-scales (internality, stability, and globality for positive and negative events), only the ASQ/ASQpf correlation for positive stability failed to reach

statistical significance ( $r = 0.022$ ,  $p < 0.75$ ). When self-serving bias scores were calculated for internality, a highly significant correlation ( $r = 0.568$ ,  $p < 0.001$ ) was observed between the original and parallel form ASQ.

### The Pragmatic Inference Task (PIT).

The PIT (Winters & Neale, 1985) was used as a covert measure of implicit attributional style. It is presented as a memory test to avoid conscious response biasing. The task consists of 12 short hypothetical vignettes, all of which are self-referent and which were directly derived from the original ASQ. Six of the vignettes describe positive events including, for example, “You recently receive a number of compliments”. The remaining six vignettes describe negative events, including one which begins “You have been looking unsuccessfully for a job as a factory worker”. Positive and negative vignettes are randomly ordered. Each story contains the implication of both an internal and an external locus of causality. For example, in the item whose theme is that of inability to secure employment, a bad personal work record (internal cause) and the poor local job market (external cause) are both alluded to in the vignette.

Each story is followed by four questions presented as multiple-choice items. Two of the questions require memory for stated fact (e.g. “How long do you go for without finding work?”)



and one question requires the participant to make a non-attributional inference (e.g. “What kind of job interests you?” with no answer provided in the story). The target, or attributional inference question, requires the participant to recall what they perceive to be the main contributing factor in the described outcome (e.g. “Why do you have trouble finding work?”), by selecting one of the two causes (internal or external) provided in the vignette. Only target questions are scored, to provide response frequencies in four categories: internal and external attributions for negative events, and internal and external attributions for positive events. A PIT self-serving bias score can thus be calculated by subtracting the number of internal attributions selected for negative events from the number of internal attributions selected for positive events.

In the present study an Anglicised version of the PIT, which had previously been employed with paranoid patients (Lyon et. al., 1994), was used to ease comprehension by British subjects (e.g. “Thanksgiving” was changed to “New Year” and “Kurt Vonnegut” to “John Fowles”). The vignettes were recorded on to audiotape for presentation to subjects, immediately after which the author read aloud the corresponding questions, and noted the participant’s verbal responses. Lyon et. al. (1994) reported a comparison between ASQ scores and scores on this version of the PIT for 15 depressed, 13 muscular dystrophy, 15 chronic fatigue, and 8 healthy participants. A highly significant correlation ( $r = 0.50$ ,  $p <$



0.001) was found between PIT self-serving bias scores and the ASQ(pf) self-serving bias scores.

### The Emotional Stroop Test (EST).

The emotional Stroop task employed in this study was similar to that previously used by Bentall and Thompson (1990) to measure selective information processing for emotional words in hypomanic students. However, in order to increase word comparability (using the Francis & Kucera, 1982, word norms) and control for emotionality, a largely new bipolar Stroop test, using different words, was devised. Thus in the new bipolar Stroop, neutral, depression-related and euphoria-related words were equivalent in terms of word length, frequency of occurrence in the English language, and number of genres in which the words occur (i.e. academic articles versus publicity material).

### Development of a New Bipolar Emotional Stroop Test.

In accord with previous researchers, six cards were used as stimulus material, each showing 50 words printed in Times New Roman 23-point font, in one of five colours (pink, blue, black, green, red), with five words to a line. Five words were used for each card, so that each line contained one of each of the words in random order. Each colour appeared on each line once, again in

random order. Two cards contained negative emotion (depression-related) words (including “loss” and “agony”) and two cards contained positive emotion (euphoria-related) words (including “happy” and “glorious”). Two cards contained neutral words (including “metaphor”, and “tendency”), and there was a practice card with 20 random words (including “apple” and “Neil”), also printed in the five ink colours. The complete colour Stroop test can be seen as appendix number ten. A one-way ANOVA found no significant differences between the six cards (excluding the test card) for word length ( $F [5, 24] = 0.215, p = 0.953$ ), frequency of word occurrence ( $F [5, 24] = 0.002, p = 1.000$ ), and genre occurrence ( $F [5, 24] = 0.204, p = 0.917$ ).

As already alluded to, all words had previously been rated in terms of “pleasantness” and “emotionality”. The final 30 words were selected from 180 (word norm compatible) candidate words, which were pilot tested by a group of 14 normal subjects. These raters were required to grade each word on a ten-point scale for emotionality (anchor points: 0 = “totally unemotional or no emotional impact”, and 10 = “extremely emotional or strong emotional impact”). They were then asked to rate each word on a similar scale for pleasantness (with anchors: 0 = “extremely unpleasant” and 10 = “extremely pleasant”). Words were excluded if there was poor agreement about their ratings, and the final selection was made so that the positive and negative emotion words were matched for emotionality. This precaution was taken to

determine whether any abnormal Stroop responses reflected general emotionality of items rather than their specific content. Statistical analysis of the final word groups demonstrated significant differences in “pleasantness” ratings between the three groups of words ( $F [5, 24] = 151.13, p < 0.00001$ ). The final negative emotion words were rated as significantly less pleasant than the neutral words, which in turn were less pleasant than the positive-emotion words. Post hoc Tukey’s honestly significant difference (HSD) tests indicated significant differences between each card and all others from different word groups ( $p < 0.01$ , for each comparison) but no significant differences between each card and the other in its word group ( $p > 0.05$ ). However, depression-related and euphoria-related words were selected on the basis of comparable emotionality. As expected, a one-way ANOVA demonstrated a significant main effect between all 6 cards ( $F [5, 24] = 124.66, p < 0.0001$ ), since this reflected the difference in emotionality between neutral and both kinds of emotion words. However, Tukey’s (HSD) comparisons demonstrated significant differences between the 2 neutral cards and the four emotion word cards ( $p < 0.01$ ), but no significant differences ( $p > 0.05$ ) between the emotionality of the four emotion word cards. That is, the euphoria-related and depression-related words were matched for emotionality.

Participants were all given the test card as practice.

Thereafter the Stroop cards were presented in quasi-random order



with the constraint that one card from each condition (depressed-related, euphoria-related or neutral) was presented prior to the presentation of a second card from any other condition. This allowed an estimation of order effects (first presentation versus second presentation) in the analysis. The time taken by participants to colour name each card was measured with a stopwatch.

### The Self-Referent Incidental Recall Task (SRIRT).

The SRIRT employed in the present study was identical to that used to explore the role of self-schema in depression (Dent & Teasdale, 1988; Hammen et. al., 1985; Williams et. al., 1990), and paranoia (Bentall & Kaney, 1996). The test was presented as a personality test wherein participants completed a 30 item Self-Statement questionnaire to indicate which trait items described them. Items consisted of the mixed list of trait adjectives derived from those previously employed (i.e. Dent & Teasdale, 1988; Williams et. al., 1990; Bentall & Kaney, 1996). Twelve were positive and related to the concept of success (including “successful”, “capable”, and “important”) and 12 were negative and related to the concept of failure (including “deficient”, “stupid”, and “unloved”). Success and failure items were presented in random order. As in previous studies, six neutral words (including “choosy”, “cautious”, and “ordinary”) were included to control for primacy and recency effects, three preceding the main list and three subsequent to it. A “yes” and “no” box followed each trait



word, and the participants were asked to tick the “yes” box if the trait adjective described them or tick the “no” box if it did not . Positive or negative explicit self-schemas were indicated by the number of success or failure endorsements in this first part of the test. The questionnaire was removed on completion and participants were required immediately to recall aloud as many of the trait words as possible. When they appeared to give up, participants were prompted: “See if you can remember any more”. The numbers of positive and negative words recalled were recorded by the author, and taken as an indicator of their more implicit self-referent schemata.

### Procedure.

All participants were given a verbal rationale for the study and measures used, without revealing the opaque nature of the defence-penetrating tests. They were then required to give written consent by signing a bilingual consent form (see appendices 12 & 13) before being included in the study. They were informed that they would gain no direct benefit from participation and that they were free to withdraw at any time. Diagnostic criteria were then confirmed (in the case of clinical participants) with the Diagnostic Interview Schedule. Testing procedure and order of presentation was identical across the three groups. As the PIT and parallel form ASQ are essentially different versions of the same test, it was important that subjects were exposed to the covert measure prior to

the overt measure. Tests were thus presented in the following order: the BDI, the RSEQ, the PIT, the ASQpf, the EST, the NART, and finally the SRIRT. Subjects were encouraged to pause for a break after the ASQpf. The test battery was completed within between 60 and 120 minutes after which participants were given the opportunity to discuss any aspect of the testing experience. A full debriefing was then available.

### Ethical and Professional Issues Relating to Procedure, Use of Covert Measures.

Clearly the covert nature of three of the tests employed has some ethical implications. However, both the procedure and tests complied with the British Psychological Society's Code of Conduct (BPS, 1996). All measures used (or those very similar) have been previously administered to healthy, depressed and paranoid (perhaps the most sensitive group) participants to no ill effect. The participants' mood, dignity, or safety was at no time at risk and was not adversely affected. Participants were fully debriefed, and given opportunities to give feedback about their experience of being a participant or to withdraw without explanation at any point.

### Poor Compliance from Bipolar-Manic Participants.

Regardless of these assurances (or perhaps because of them), several of the bipolar-manics did exactly that; withdrew consent or were non-compliant. Some abandoned testing, failed to complete the test battery or completed but took their papers. The request for

written consent, and particularly the sight of the Welsh consent form seemed especially provocative, often eliciting delusional ideas or concerns about involuntary detention. Also, a minority of bipolar-manic patients demonstrated sufficiently inappropriate behaviour to cause the author to terminate proceedings prematurely (although, apart from the two already mentioned, these were re-enlisted a few days later as their manic symptoms waned). Of those who did complete testing, most found the process interesting (this includes participants from all three groups). They also valued the opportunity to talk on a one-to-one basis with a mental health professional. Some were surprisingly insightful but none requested a direct explanation regarding the less transparent tests. As a chartered clinical psychologist the author was competent to respond appropriately should any evidence of psychological distress have arisen during the testing.

### Participants' Preferred Language.

Another, obvious professional issue concerns the author's monolingualism in what aspires to be a bilingual health service. As a result of this a substantial minority of the respondents were denied the opportunity to participate in this study through the medium of Welsh, their preferred language. Clearly, however, all of the measures administered have been validated, and are available, only in English, and from a methodological point of view, the groups were controlled for language.

**Ethical Approval.**

Ethical approval was granted from the then Gwynedd and Clwyd Health Authorities, and from the Psychology Department in University College North Wales, Bangor.



## Chapter Eight.

### Results.

#### Beck Depression and Rosenberg Self-Esteem Results.

The Beck Depression Inventory (BDI) and Rosenberg Self-Esteem Questionnaire (RSEQ) mean scores for the three groups are given in Table 1 (p. 133).

#### The BDI.

The significant difference between the groups for the BDI,  $F[2, 42] = 112.11, p < 0.0001$ , was accounted for by the higher scores of the bipolar-depressed patients compared to the two other groups, as is more clearly illustrated in Figure 1 (p. 132). Only the bipolar-depressed group mean score came within the range of “clinical depression” and Post hoc Tukey’s H.S.D. tests confirm significant differences in BDI mean scores ( $p < 0.01$ ) for comparisons between the depressed and other groups.



**Figure 1. Mean Scores on the Beck Depression Inventory (BDI) and Rosenberg Self-Esteem Questionnaire (RSEQ) for Normal, Bipolar-Depressed and Bipolar-Manic Groups.**

Table 1: Mean Scores and Standard Deviations of Bipolar-Manic, Bipolar-Depressed, and Normal Participants on the BDI and the RSEQ.

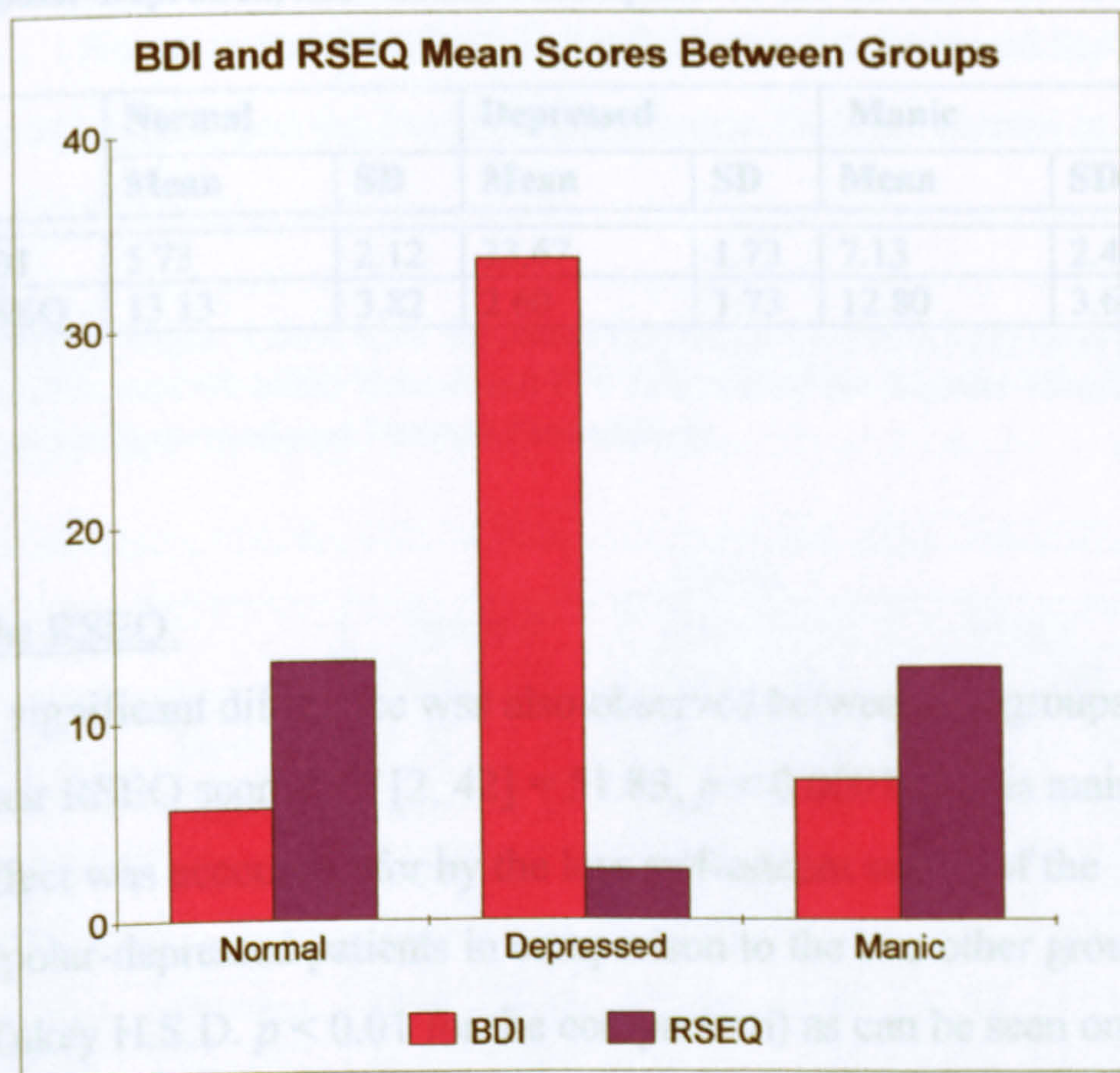


Figure 1, above.



**Table 1: Mean Scores and Standard Deviations of Bipolar-Manic, Bipolar-Depressed, and Normal Participants on the BDI and the RSEQ.**

	Normal		Depressed		Manic	
	Mean	SD	Mean	SD	Mean	SD
BDI	5.73	2.12	33.67	1.73	7.13	2.42
RSEQ	13.13	3.82	2.60	1.73	12.80	3.69

The RSEQ.

A significant difference was also observed between the groups for their RSEQ scores, ( $F [2, 42] = 51.83, p < 0.0001$ ). This main effect was accounted for by the low self-esteem scores of the bipolar-depressed patients in comparison to the two other groups (Tukey H.S.D.  $p < 0.01$  for the comparison) as can be seen on Figure 1, above.

Attributional Style Questionnaire Results.

Group mean data for the parallel form Attributional Style Questionnaire and the Pragmatic Inference Task are shown in Table 2., below.

**Table 2: Mean Scores (and Standard Deviations) for ASQ(pf) Internality, Stability and Globality Scales and PIT Internality for Bipolar-Manic, Bipolar-Depressed and Normal Participants.**

		Group					
		Normal		Depressed		Manic	
		Mean	SD	Mean	SD	Mean	SD
ASQ(pf) Internality	Positive	28.27	3.15	23.13	4.87	31.93	1.26
	Negative	19.07	5.35	30.73	5.30	17.80	5.70
ASQ(pf) Stability	Positive	29.87	5.11	22.20	2.81	31.20	4.25
	Negative	19.93	7.31	26.73	5.23	18.00	5.31
ASQ(pf) Globality	Positive	27.93	4.38	21.20	4.90	27.80	1.27
	Negative	18.00	8.05	25.53	6.12	15.67	4.53
PIT Internality	Positive	4.00	0.76	1.20	0.86	2.27	2.00
	Negative	1.93	1.10	4.20	0.94	3.87	1.36

ASQ Internality.

Figure 2 (p. 136) shows the mean internality scores for all three groups on the ASQpf (and also on the PIT). Inspection of these data suggests that both the manic patients and the normal participants showed a self-serving bias on the ASQ(pf), whereas the bipolar-depressed patients demonstrated a self-denigrating bias

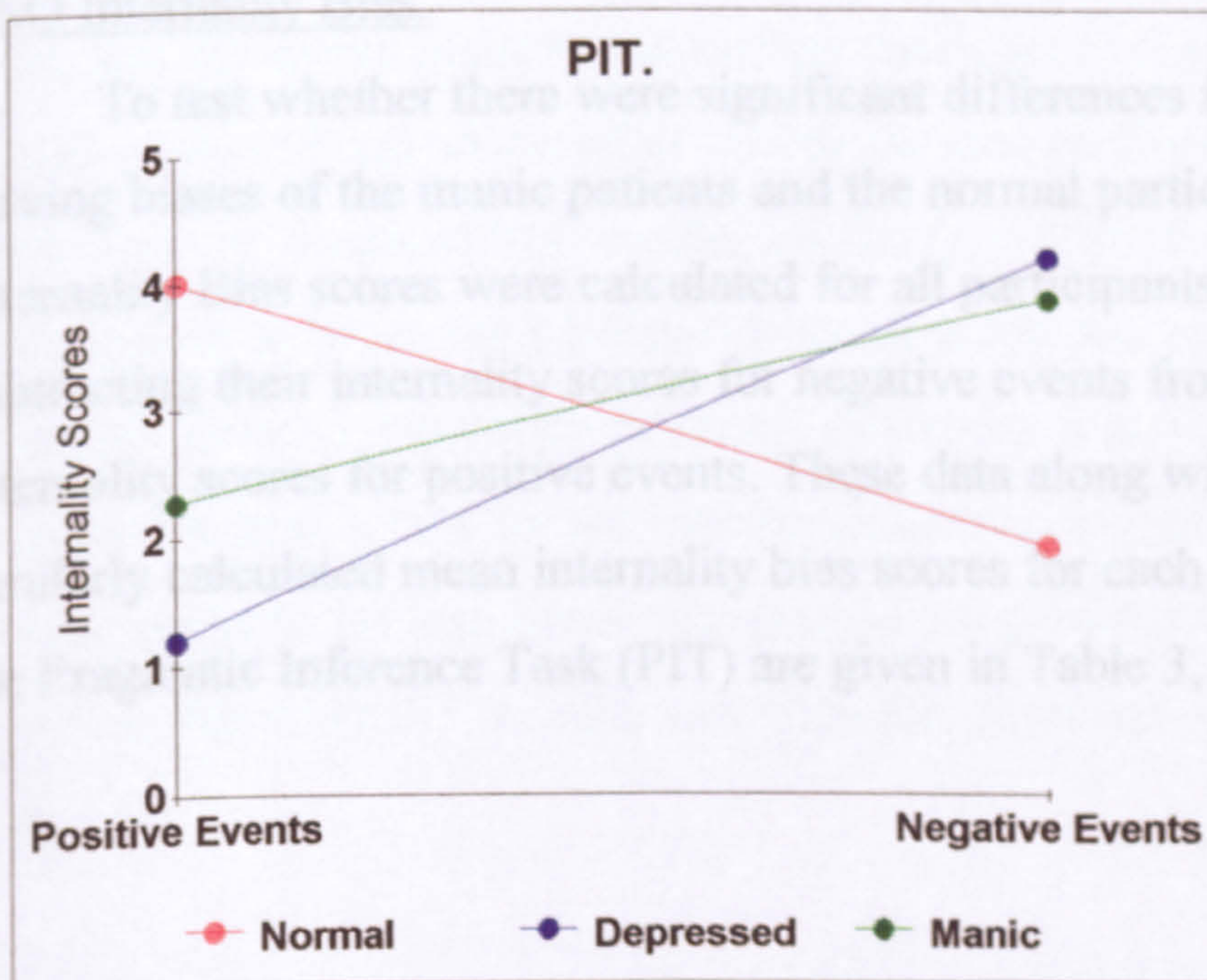
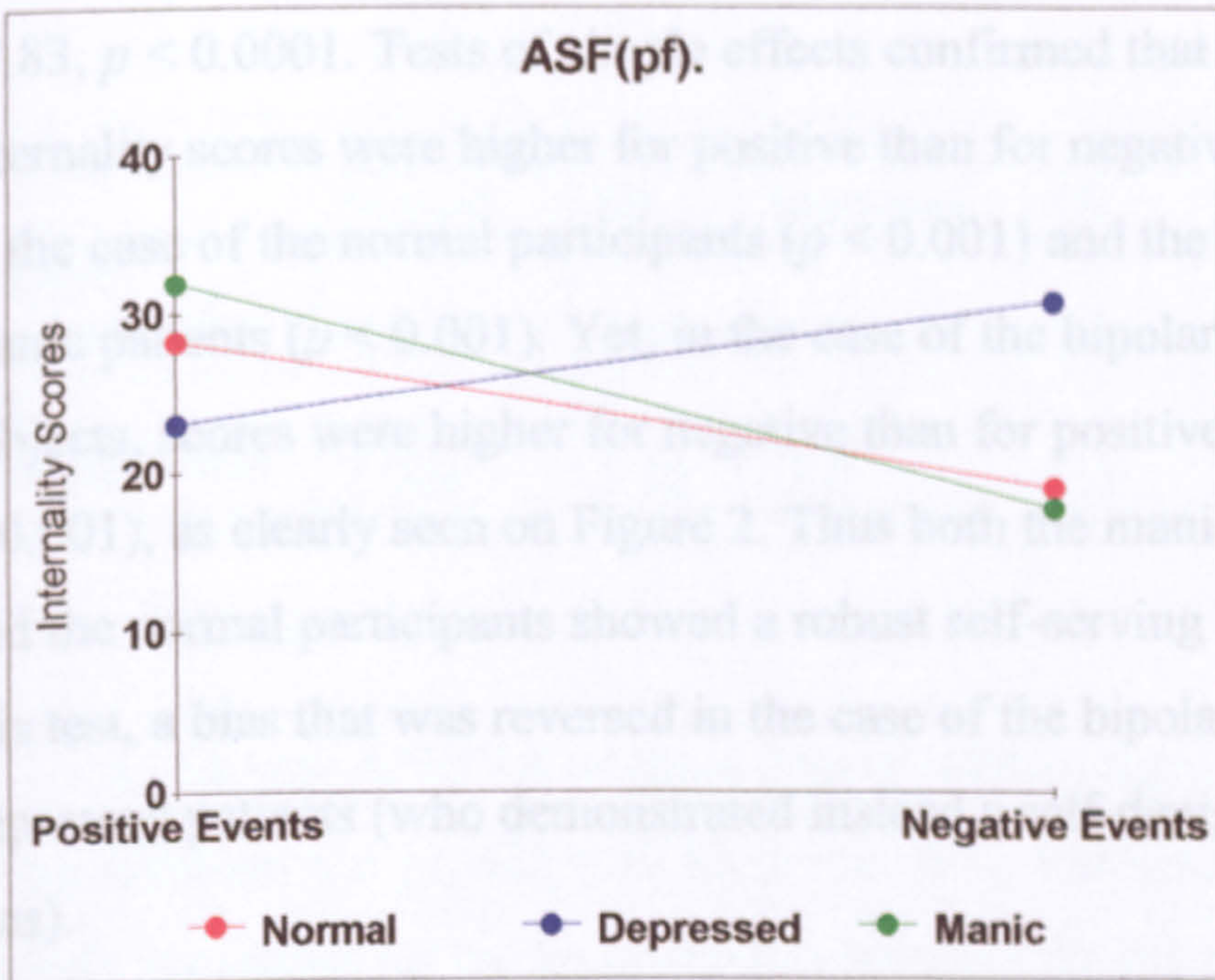


on this measure, attributing negative events more than positive events to internal causes. However on the PIT only the normal participants appear to show a self-serving bias, and both the bipolar-depressed and the manic patients seem to show a self-denigrating bias. Statistical analyses were used to formally test these observations.

A two way analysis of variance (ANOVA) on the ASQ(pf) internality data (groups x valence of events) revealed a significant main effect for group,  $F [2, 42] = 3.84, p < 0.05$  (ANOVA tables for selected data sets can be found in appendix 14). This main effect for group can be accounted for by reference to group means; (normals equal to 23.7, manics equal to 24.9, and depressed equal to 26.9) and has to be interpreted in light of the significant interaction, discussed below. Tukey<sub>a</sub> tests revealed that, averaging over positive and negative events the normal respondents had significantly lower internality scores than the depressed respondents ( $p < 0.05$ ). The manics did not score significantly differently from either of the other two groups and fell in between. The main effect for valence (positive v negative events) was also significant,  $F [1, 42] = 24.05, p < 0.0001$ . This was accounted for by higher overall internality scores for positive rather than for negative events across the three groups with means for positive events equal to 27.8 and for negative events equal to 22.5. This is consistent with the majority of participants showing a self-serving bias (attributing positive events to themselves and negative events to others or external causes).



**Figure 2. Mean Internality Scores on the Attributional Style Questionnaire (ASQpf) and the Pragmatic Inference Task (PIT) for Normal, Bipolar-Depressed and Bipolar-Manic Groups.**





However it is the group versus valence effect that is of most interest. This interaction was also highly significant,  $F [2, 42] = 37.83, p < 0.0001$ . Tests of simple effects confirmed that the internality scores were higher for positive than for negative events in the case of the normal participants ( $p < 0.001$ ) and the bipolar-manic patients ( $p < 0.001$ ). Yet, in the case of the bipolar-depressed subjects, scores were higher for negative than for positive events ( $p < 0.001$ ), as clearly seen on Figure 2. Thus both the manic patients and the normal participants showed a robust self-serving bias on this test, a bias that was reversed in the case of the bipolar-depressed patients (who demonstrated instead a self-denigrating bias).

### ASQ Internality Bias.

To test whether there were significant differences in the self-serving biases of the manic patients and the normal participants, Internality Bias scores were calculated for all participants by subtracting their internality scores for negative events from their internality scores for positive events. These data along with similarly calculated mean internality bias scores for each group on the Pragmatic Inference Task (PIT) are given in Table 3, below.

**Table 3: Means and SDs of Internality Bias Scores on the ASQ(pf) and PIT for Bipolar-Manic, Bipolar-Depressed and Normal Participants.**

	Group					
	Normal	SD	Depressed	SD	Manic	SD
ASQ(pf) Internality Bias	9.2	6.65	-7.6	7.62	14.133	7.20
PIT Internality Bias	2.066	1.16	-3.0	1.3	-1.6	2.77

A one way ANOVA of these data revealed a significant group difference,  $F [2, 42] = 37.83, p < 0.0001$ . However this was entirely accounted for by the difference between the bipolar-depressed participants (with a negative overall bias) on the one hand, and the manic patients and normal participants (both with an overall positive bias) on the other (as can be seen on Figure 5, p. 144). Tests of simple effects confirmed this ( $p < 0.01$ , for comparisons between the bipolar-depressed group and each of the other two groups). No such significant difference was observed between the bias scores for the bipolar-manic participants and the normal controls. Of less direct relevance to the present study are, however, the ASQ(pf) Stability and Globality measures reported next.



### ASQ Stability.

When a two-way ANOVA (groups x valence of events) was conducted on stability scores, the main effect for group failed to reach significance,  $F [2, 42] = 0.62, p = 0.94$ . There was however a significant main effect for valence,  $F [1, 42] = 29.54, p = 0.0001$ , accounted for by higher stability scores across the groups for positive events (mean equal to 27.7) than for negative events (mean equal to 21.5). Once again, this observation should be interpreted in consideration of the significant interaction,  $F [2, 42] = 22.82, p = 0.0001$ . This, the most interesting ASQ stability finding, is better illustrated by way of a graph in Figure 3 (p. 141). Tests of simple effects confirm that the normal participants ( $p < 0.001$ ) and bipolar-manic subjects ( $p < 0.001$ ) gave higher stability scores for positive events whereas the bipolar-depressed produced higher stability scores for negative events ( $p < 0.05$ ).

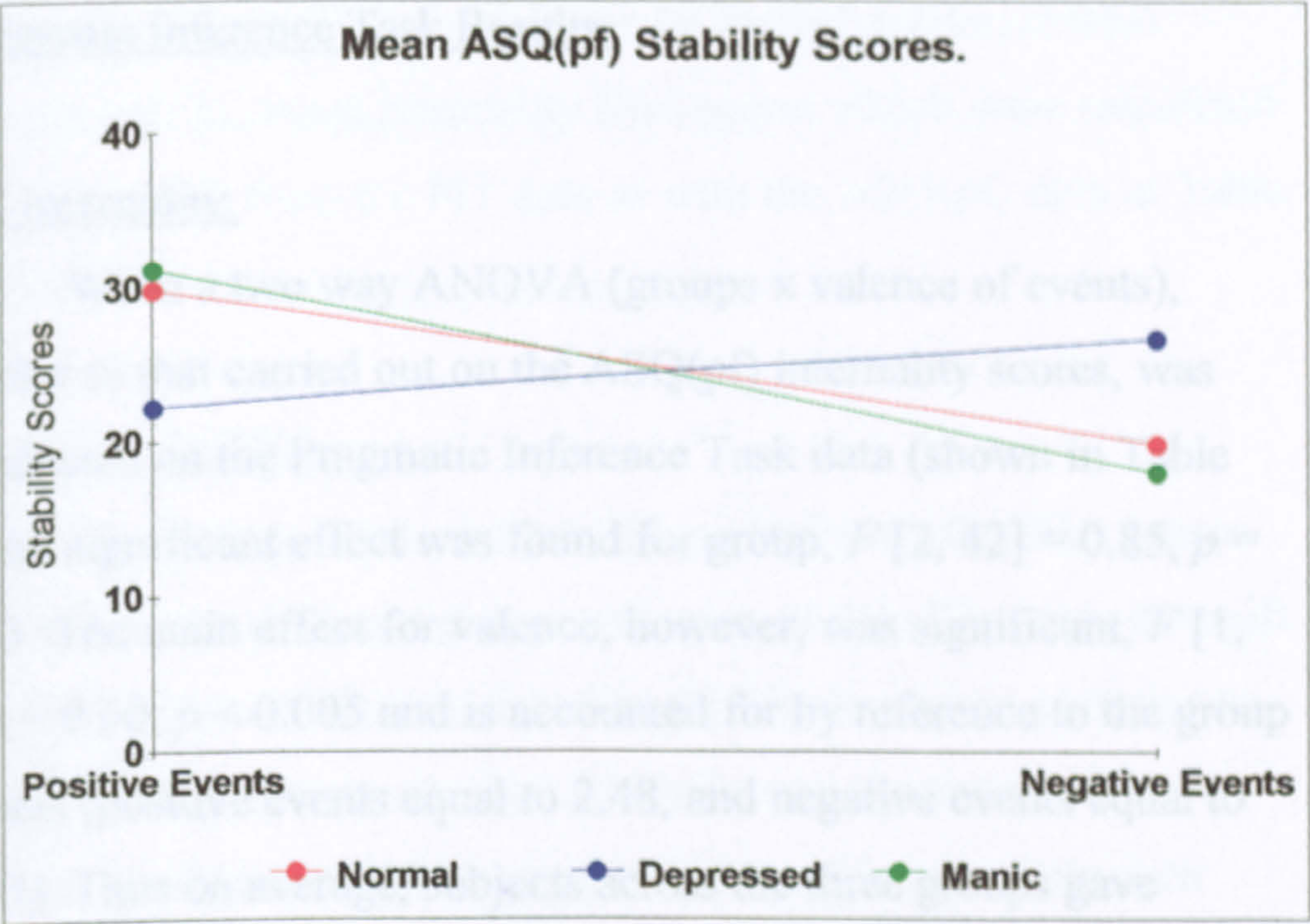
### ASQ Globality.

Finally, an analysis of the ASQ(pf) globality scores was conducted using a 2-way ANOVA (groups x valence) and again the group main effect was non-significant ( $F [2, 42] = 0.533, p = 0.59$ ). There was however, a highly significant effect for valence of events,  $F [1, 42] = 32.92, p = 0.0001$ , accounted for by higher globality scores across the groups for positive (mean equal to 25.6) than for negative events (mean equal to 19.7). The significant interaction effect ( $F [1, 42] = 25.1, p = 0.0001$ ) is illustrated in

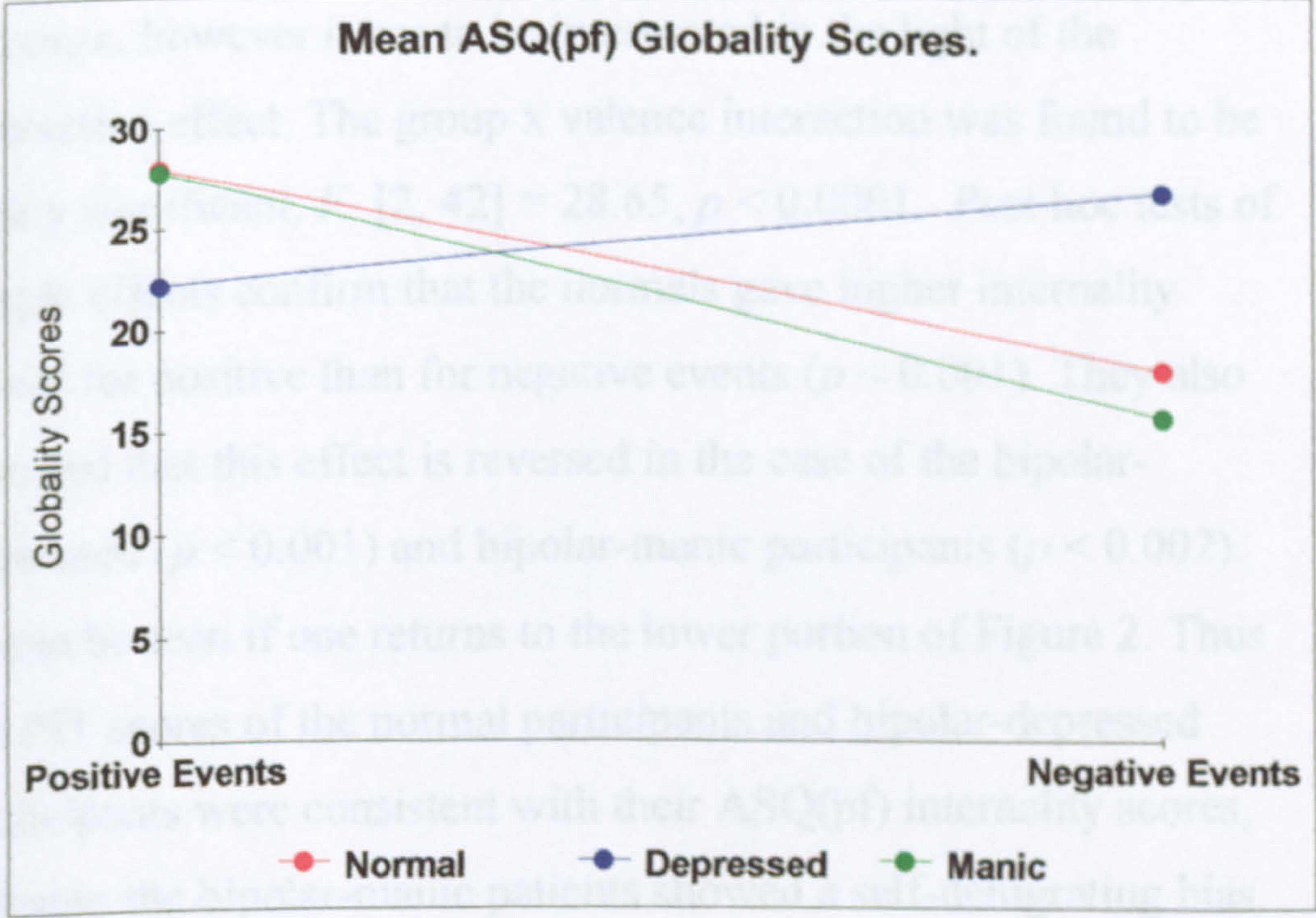
Figure 4 (p. 141). Post hoc tests of simple effects again confirm that the normal and bipolar-manic participants showed higher globality scores for positive events ( $p < 0.001$ , for both comparisons), whereas bipolar-depressed participants showed higher globality scores for negative events ( $p < 0.02$ ).



**Figure 3.** ASQ(pf) Mean Stability Scores for Normal, Bipolar-Depressed and Bipolar-Manic Groups.



**Figure 4.** ASQ(pf) Mean Globality Scores for Normal, Bipolar-Depressed and Bipolar-Manic Groups.





## Pragmatic Inference Task Results.

### PIT Internality.

When a two way ANOVA (groups x valence of events), similar to that carried out on the ASQ(pf) internality scores, was conducted on the Pragmatic Inference Task data (shown in Table 2), no significant effect was found for group,  $F [2, 42] = 0.85, p = 0.43$ . The main effect for valence, however, was significant,  $F [1, 42] = 9.00, p < 0.005$  and is accounted for by reference to the group means (positive events equal to 2.48, and negative events equal to 3.33). Thus on average, subjects across the three groups gave higher internality scores for negative events than for positive events on the PIT. This observation is fairly unremarkable as it was anticipated that 2 of the 3 groups would produce such a pattern of response, however it has to be interpreted in the light of the interaction effect. The group x valence interaction was found to be highly significant,  $F [2, 42] = 28.65, p < 0.0001$ . Post hoc tests of simple effects confirm that the normals gave higher internality scores for positive than for negative events ( $p < 0.001$ ). They also revealed that this effect is reversed in the case of the bipolar-depressed ( $p < 0.001$ ) and bipolar-manic participants ( $p < 0.002$ ), as can be seen if one returns to the lower portion of Figure 2. Thus the PIT scores of the normal participants and bipolar-depressed participants were consistent with their ASQ(pf) internality scores, whereas the bipolar-manic patients showed a self-denigrating bias on the PIT that was opposite to the self-serving bias previously



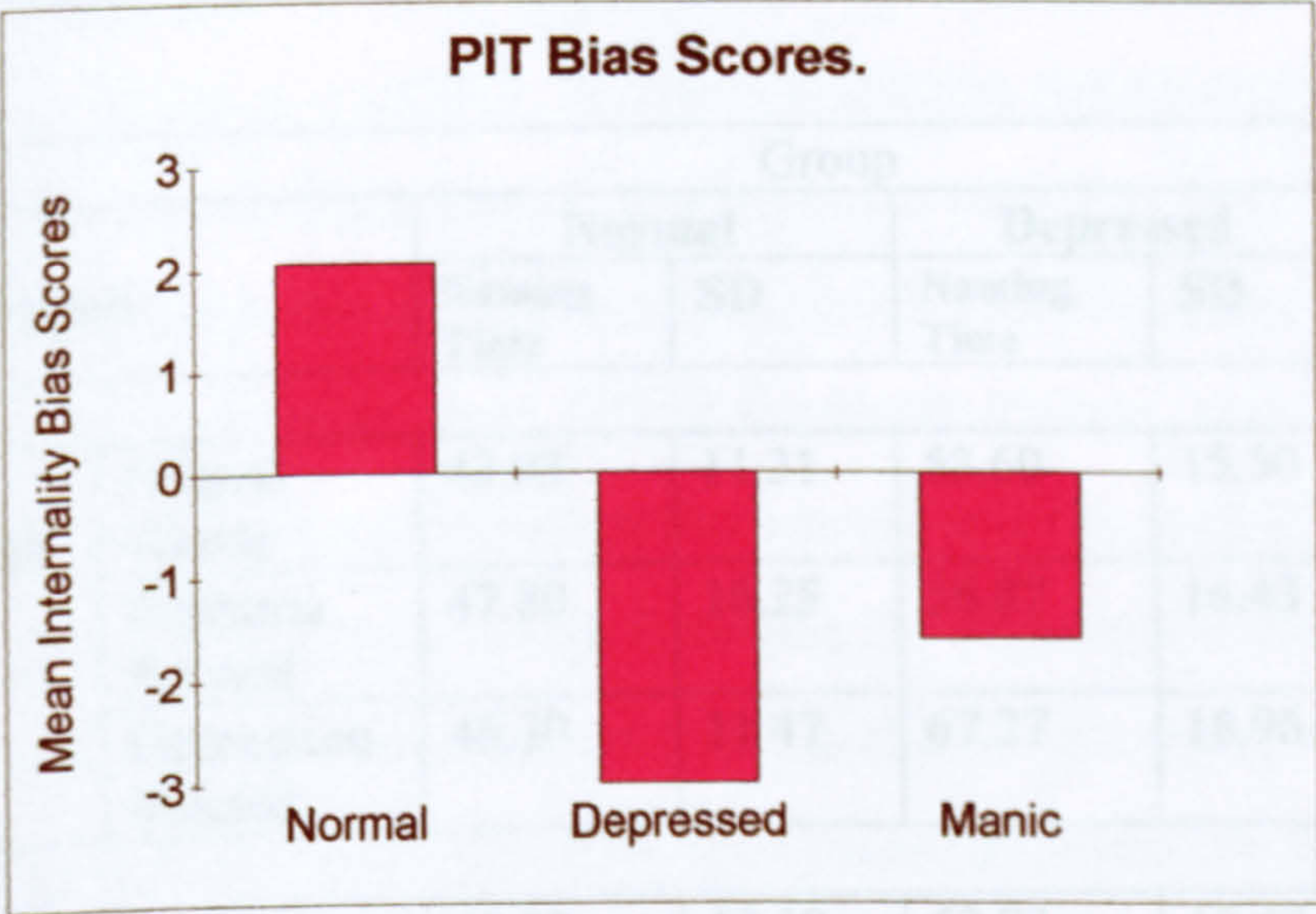
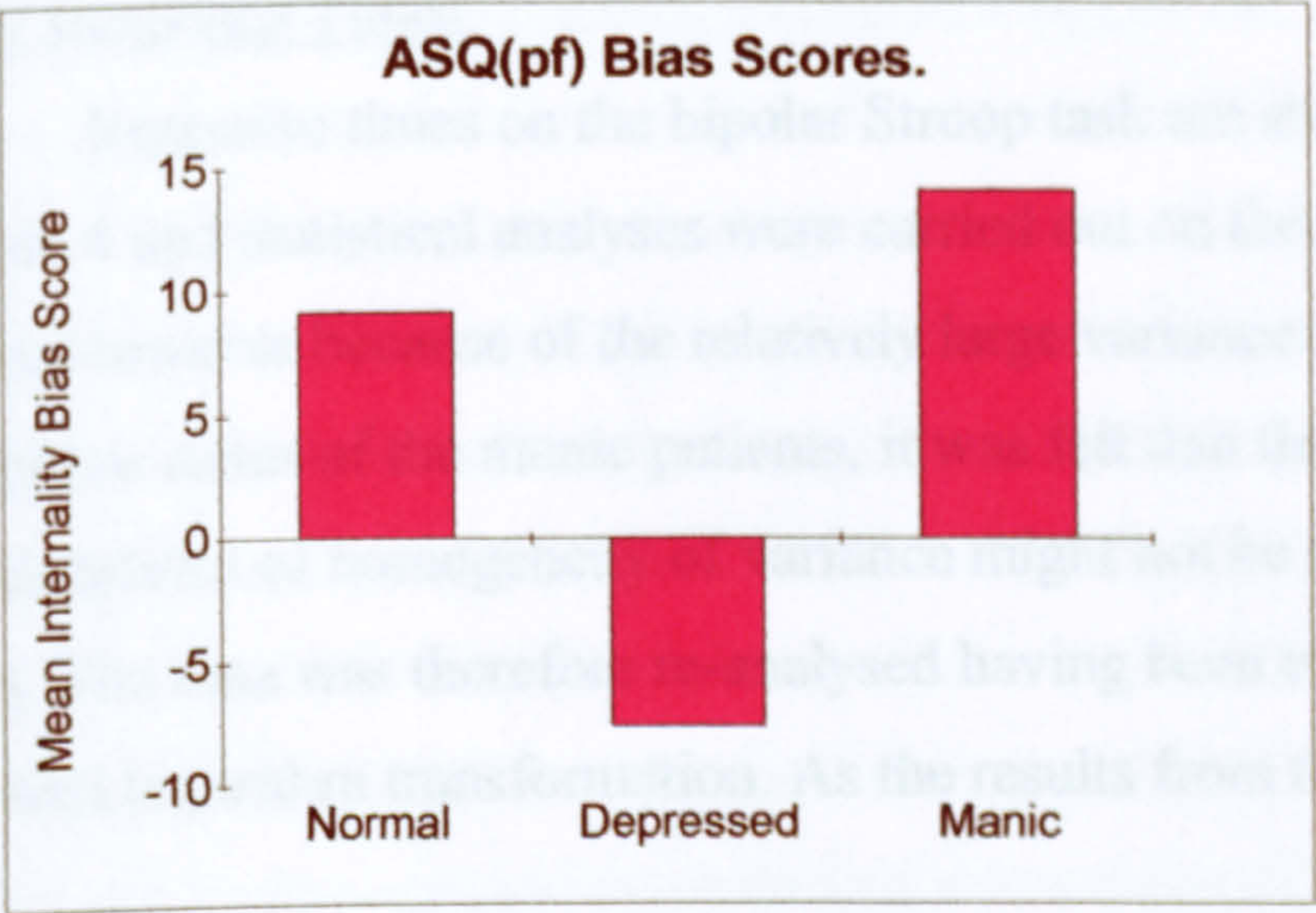
exhibited on the ASQ(pf). This can be further examined with reference to the mean Internality Bias scores which were calculated for each group from the PIT data as with the ASQ(pf) data in Table 3 above.

### PIT Internality Bias.

Figure 5 (p. 144) contrasts the mean Internality Bias scores attained by the different groups and more clearly illustrates the shift in bias exclusively demonstrated by the bipolar-manic participants when moving from an obvious to a more covert measure. The normal and bipolar-depressed participants appear to retain their relative positive and negative biases regardless of how their attributions were elicited.



**Figure 5. Mean Internality Bias Scores on the Attributional Style Questionnaire (ASQpf) and Pragmatic Inference Task (PIT) for Normal, Bipolar-Depressed, and Bipolar-Manic Groups.**





Emotional Stroop Test Results.

EST Response Times.

Response times on the bipolar Stroop task are shown in Table 4 and statistical analyses were carried out on the raw stroop data. However because of the relatively large variance in the response times of the manic patients, it was felt that the assumptions of homogeneity of variance might not be adequately met. The data was therefore re-analysed having been treated with a natural logarithm transformation. As the results from the both

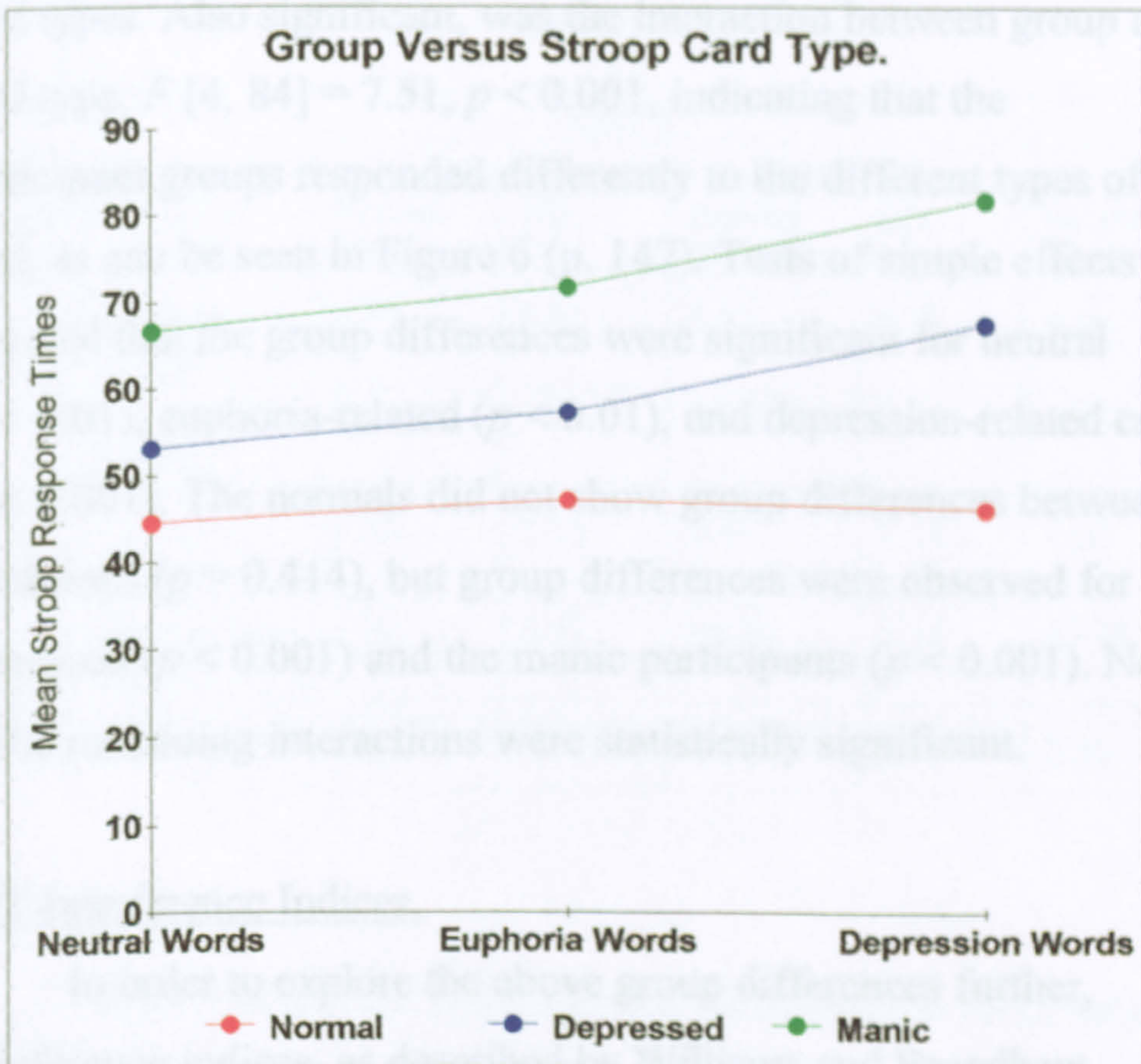
**Table 4: Mean Colour-Naming Times (and SDs) for Neutral, Euphoria-Related and Depression-Related Words on the Emotional Stroop Test (EST) for Bipolar-Manic, Bipolar-Depressed and Normal Participants.**

Group							
Stroop times		Normal		Depressed		Manic	
		Naming Time	SD	Naming Time	SD	Naming Time	SD
First Presentation	Neutral Words	43.93	11.31	53.60	15.30	64.60	29.40
	Euphoria Related	47.80	16.25	56.87	16.43	69.80	27.01
	Depression Related	46.20	11.47	67.27	18.96	80.67	30.43
Second Presentation	Neutral Words	45.33	12.30	52.73	13.40	68.80	29.48
	Euphoria Related	46.67	13.36	57.80	16.19	73.87	32.16
	Depression Related	46.33	14.41	67.93	18.20	83.20	36.36

analyses were very similar, the findings from the transformed data alone are reported here. A three-way ANOVA (group x card type x first versus second presentation) revealed a significant group main effect,  $F [2, 24] = 7.26, p < 0.001$ . The bipolar-manic patients took significantly longer to colour-name words across all conditions (mean response time of 74.80 seconds) than the normal subjects (46.04 seconds), with the response times of the bipolar-depressed falling between the two groups (59.36 seconds). A posteriori Tukey HSD tests revealed that the difference between the manic and normal participants alone was significant ( $p < 0.01$ ). The effect for order (first versus second presentation) was not significant,  $F [1, 42] = 1.75, p = 0.19$ , but a significant main effect was found for card type (i.e.; neutral, euphoric or depression related words),  $F [2, 24] = 36.00, p < 0.0001$ . Across conditions the neutral cards were colour-named significantly faster (mean response time of 55.83 seconds) than the euphoria-related cards (59.05 seconds), and depression-related cards (65.32 seconds). Tukey's tests confirmed that the differences between the neutral and the euphoria-related response means ( $p < 0.05$ ), between the depressed-related and the neutral response means ( $p < 0.01$ ) and between the means for the two emotion conditions ( $p < 0.01$ ) were all significant.



**Figure 6. Mean Response Times on Neutral, Euphoria and Depression- Related Stroop Cards for Normal, Bipolar-Depressed, and Bipolar-Manic Groups.**





That is, when all subjects were considered together, there were significant differences in mean response times for all the different card types. Also significant, was the interaction between group and card type,  $F [4, 84] = 7.51, p < 0.001$ , indicating that the participant groups responded differently to the different types of card, as can be seen in Figure 6 (p. 147). Tests of simple effects revealed that the group differences were significant for neutral ( $p < 0.01$ ), euphoria-related ( $p < 0.01$ ), and depression-related cards ( $p < 0.001$ ). The normals did not show group differences between conditions ( $p = 0.414$ ), but group differences were observed for the depressed ( $p < 0.001$ ) and the manic participants ( $p < 0.001$ ). None of the remaining interactions were statistically significant.

### EST Interference Indices.

In order to explore the above group differences further, interference indices, as described by Williams and Broadbent (1986), were computed for both emotion word (depression and euphoria related) test materials. These were calculated by subtracting response times for the neutral-word cards from the response times for the relevant euphoria-related and depression-related word cards. This also took into account order of presentation, thus the response times for the first presented neutral card was subtracted from those of the first presented euphoria and depression cards, etc. The interference indices for the three groups are presented in Table 5 (p. 149).

**Table 5: Interference Indices Calculated by Subtracting Times for Neutral Words from Times for The Euphoria-Related and Depression-Related Words on the Emotional Stroop Task (EST) for Each Group.**

Group							
Interference Indices		Normal		Depressed		Manic	
		Naming Time	SD	Naming Time	SD	Naming Time	SD
First Presentation	Euphoria Related	3.87	6.05	3.27	6.77	5.20	9.04
	Depression Related	2.26	4.80	13.67	9.73	16.07	6.81
Second Presentation	Euphoria Related	1.33	7.11	5.07	6.14	5.07	11.08
	Depression Related	1.00	6.42	15.20	8.14	13.73	13.11
Total Errors		0.87	0.99	0.87	1.19	2.47	3.96

When a three-way ANOVA was performed on these data a significant main effect was found for group,  $F [2, 42] = 9.24$ ,  $p < 0.0005$ . This can be explained by the following group means: normal controls equal to 2.45 seconds, bipolar-depressed participants equal to 9.3 seconds and, bipolar-manic participants equal to 10.02 seconds. Post hoc Tukey HSD tests revealed that both the depressed participants ( $p < 0.01$ ) and the manic participants ( $p < 0.01$ ) showed significantly more interference overall than the normals, but the difference between the manics and the depressed patients was not significant. A further main effect



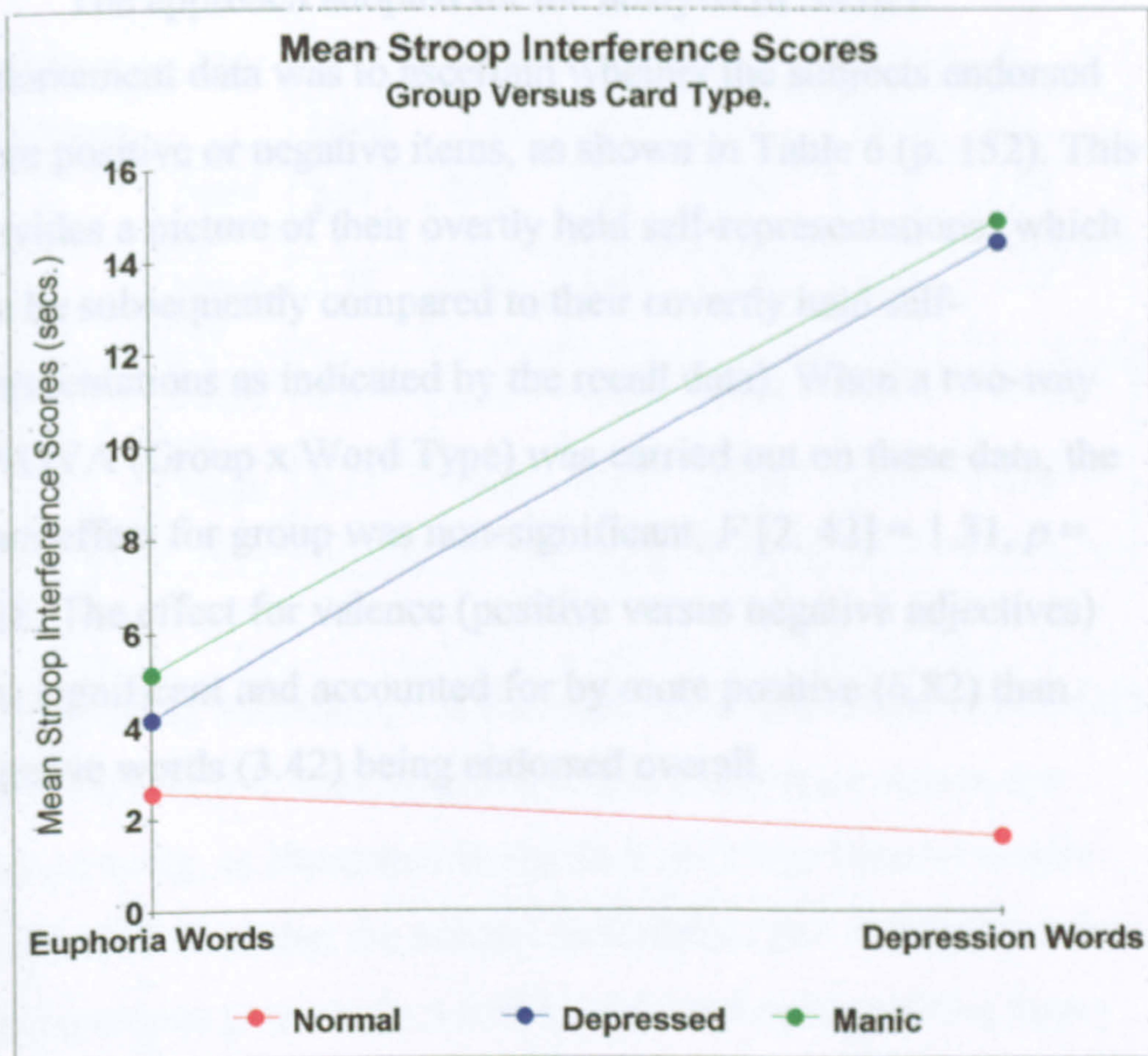
was found for card type,  $F [1, 42] = 45.69, p < 0.0001$ . This can be accounted for by the fact that the interference effect was much greater across the groups for the depression-related words than for the euphoria-related words as can be seen by the means; euphoria words equal to 3.97 seconds contrasted to depression words equal to 10.54 seconds. Once again it was the group x type interaction, which is of most relevance to the current investigation and this was also highly significant ( $F [1, 42] = 12.51, p < 0.0001$ ). This effect is demonstrated graphically in Figure 7(p. 151). Tests of simple effects reveal that the group differences are evident only for interference on depressive words ( $p < 0.001$ ) and not on interference for euphoric words ( $p = 0.535$ ). They also confirmed that both the bipolar-manic patients ( $p < 0.001$ ) and the bipolar-depressed patients ( $p < 0.001$ ), but not the normal controls ( $p = 0.860$ ), showed greater interference for the depression cards than for the euphoria related word cards as was predicted.

### EST Errors.

Stroop error data are also shown at the foot of Table 5. Because colour naming errors rarely occurred and were usually corrected spontaneously (which was therefore reflected in increased response times) it made little sense to analyse these data.



**Figure 7. Mean Stroop Interference Scores on Euphoria-Related and Depression-Related Stroop Cards for Normal, Bipolar-Depressed and Bipolar-Manic Groups.**





## The Self-Referent Incidental Recall Test Results.

### SRIRT Endorsements.

The approach adopted for the analysis of SRIRT endorsement data was to ascertain whether the subjects endorsed more positive or negative items, as shown in Table 6 (p. 152). This provides a picture of their overtly held self-representations (which can be subsequently compared to their covertly held self-representations as indicated by the recall data). When a two-way ANOVA (Group x Word Type) was carried out on these data, the main effect for group was non-significant,  $F [2, 42] = 1.31, p = 0.28$ . The effect for valence (positive versus negative adjectives) was significant and accounted for by more positive (6.82) than negative words (3.42) being endorsed overall.



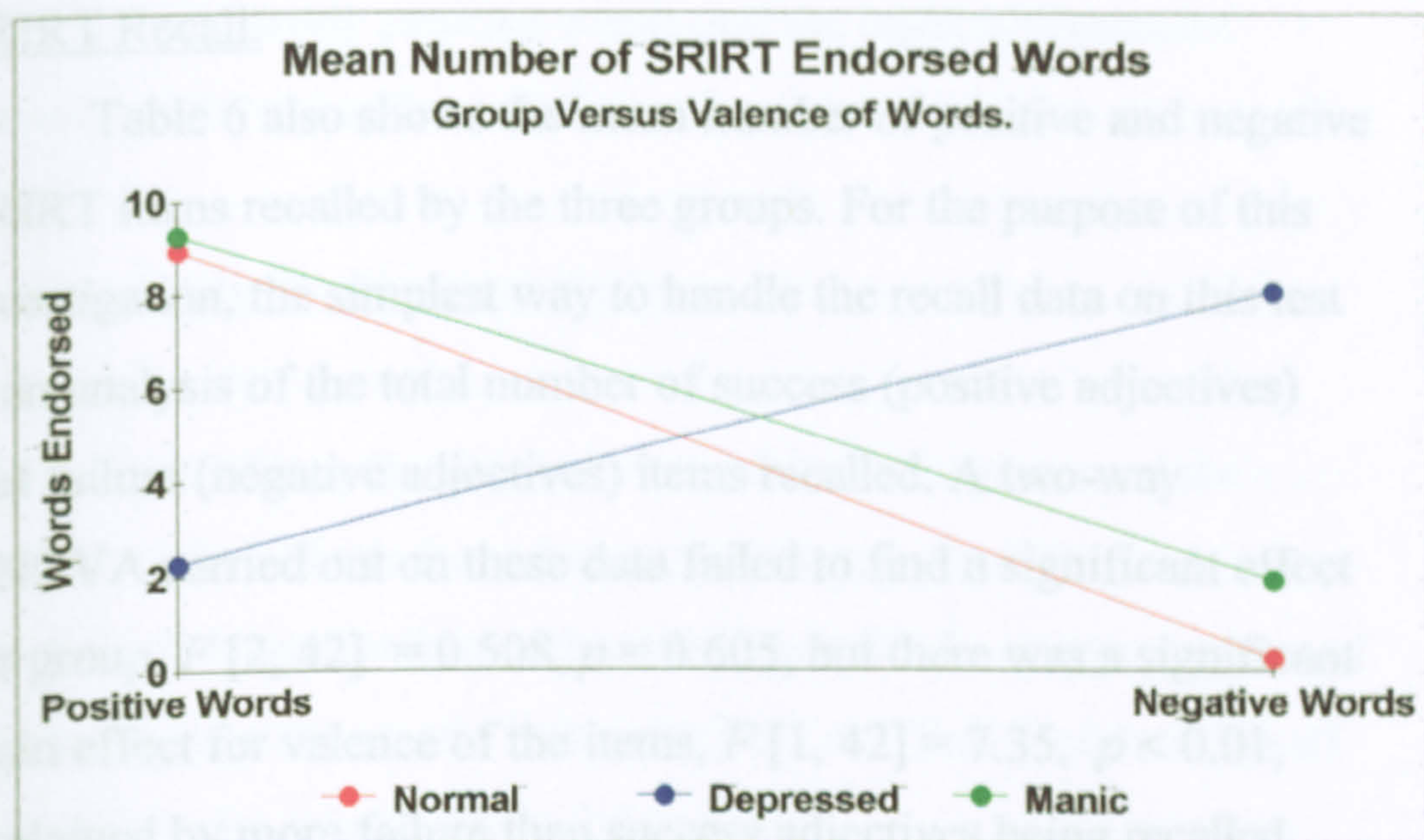
**Table 6: Mean Number of Positive and Negative SRIRT Trait Words Endorsed as True to Self and Mean Number of Positive and Negative Words Subsequently Recalled by Bipolar-Manic, Bipolar-Depressed and Normal Participants (together with SDs).**

		Group					
SRIRT Scores		Normal		Depressed		Manic	
		Mean	SD	Mean	SD	Mean	SD
Items Endorsed	Positive	8.93	2.37	2.27	1.62	9.27	2.58
	Negative	0.27	0.46	8.07	2.40	1.93	3.22
Items Recalled	Positive	5.33	2.09	1.73	1.10	2.93	1.16
	Negative	2.13	1.60	5.47	2.20	5.27	2.43

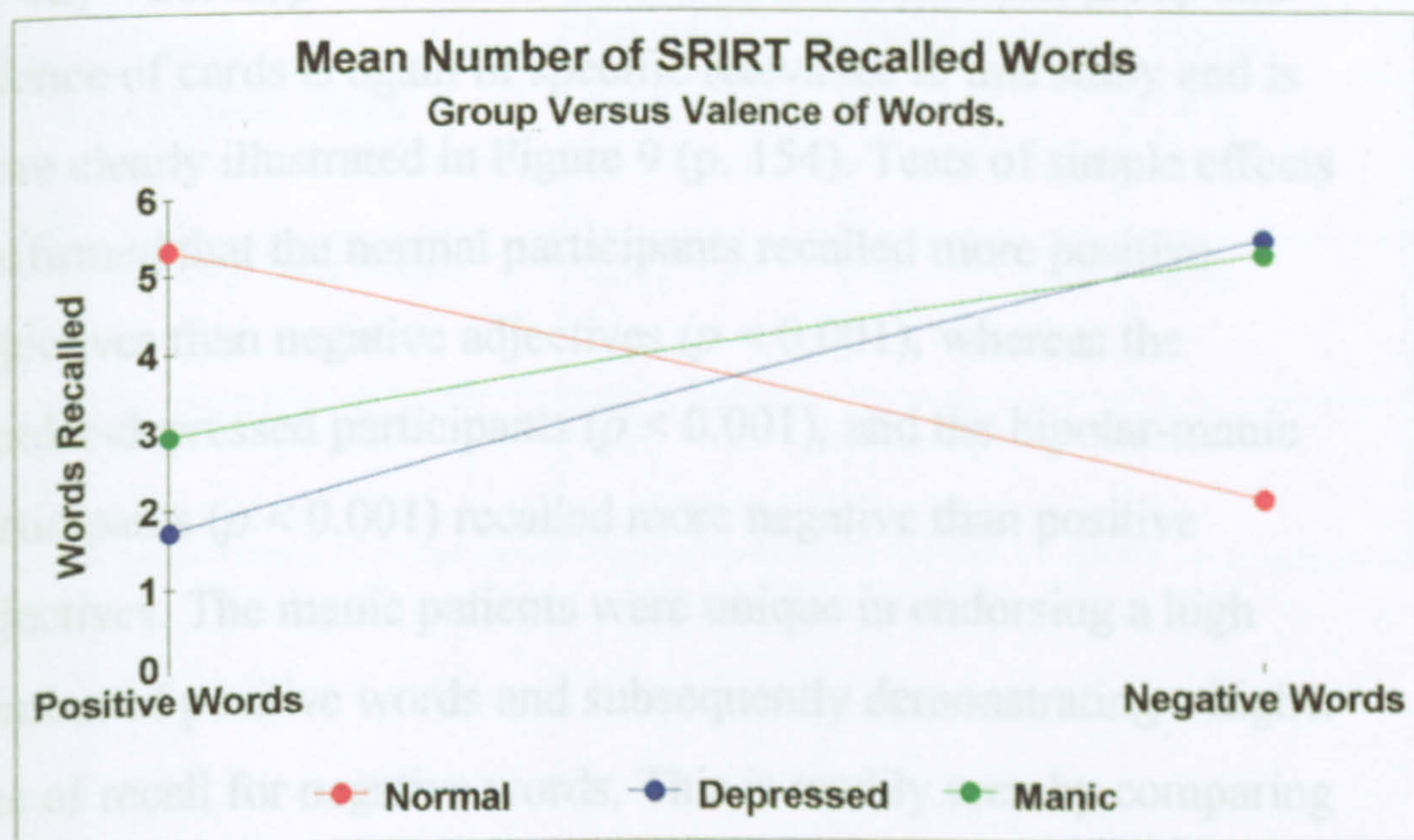
As previously intimated, it is the significant interaction effect,  $F [1, 42] = 102.46, p < 0.0001$ , that is of particular relevance to the present study, as illustrated in Figure 8 (p. 154). Tests of simple effect confirmed that the normal participants ( $p > 0.001$ ), and the bipolar-manic patients ( $p < 0.001$ ) endorsed more positive than negative items whereas the bipolar-depressed patients ( $p < 0.001$ ) endorsed more negative than positive items.



**Figure 8.** Mean Number of Positive and Negative Adjectives Endorsed on the SRIRT by Normal, Bipolar-Depressed and Bipolar-Manic Participants.



**Figure 9.** Mean Number of Positive and Negative Adjectives Recalled on the SRIRT by Normal, Bipolar-Depressed and Bipolar-Manic Participants.





### SRIRT Recall.

Table 6 also shows the mean number of positive and negative SRIRT items recalled by the three groups. For the purpose of this investigation, the simplest way to handle the recall data on this test is an analysis of the total number of success (positive adjectives) and failure (negative adjectives) items recalled. A two-way ANOVA carried out on these data failed to find a significant effect for group,  $F [2, 42] = 0.508, p = 0.605$ , but there was a significant main effect for valence of the items,  $F [1, 42] = 7.35, p < 0.01$ , explained by more failure than success adjectives being recalled across groups (mean recall of success items equal to 3.33, and recall of failure items equal to 4.29). This observation should be viewed in the context of the highly significant interaction effect,  $F [2, 42] = 36.08, p < 0.0001$ . This interaction between group and valence of cards is again of specific relevance to this study and is more clearly illustrated in Figure 9 (p. 154). Tests of simple effects confirmed that the normal participants recalled more positive adjectives than negative adjectives ( $p < 0.001$ ), whereas the bipolar-depressed participants ( $p < 0.001$ ), and the bipolar-manic participants ( $p < 0.001$ ) recalled more negative than positive adjectives. The manic patients were unique in endorsing a high number of positive words and subsequently demonstrating a higher rate of recall for negative words. This is readily seen by comparing Figures 8 and 9, which also demonstrate that neither the bipolar-depressed nor the normal respondents underwent such an



endorsement-recall shift. Rather the normal controls both endorsed and recalled mostly positive words and the bipolar-depressed patients endorsed and recalled mostly negative words.

Further Analysis of Implicit Tasks.

In an attempt to determine whether performance on the implicit tasks reflected a common underlying psychological process, data were collected from the specifically opaque aspects of the Stroop, the Pragmatic Inference Task and Self-Referent Recall task (as can be seen in Table 7, below).

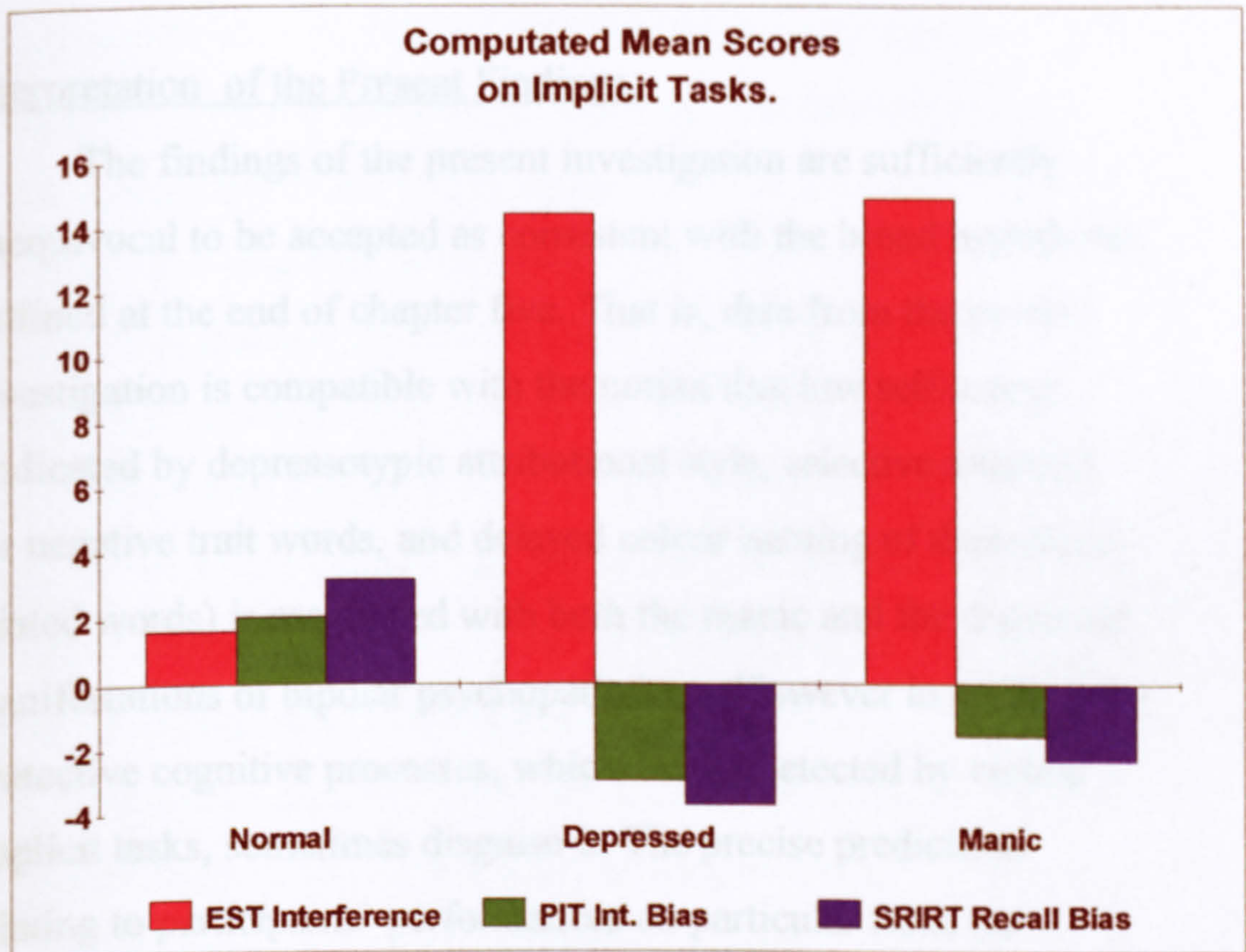
**Table 7: Mean Stroop Depression-Related Cards Interference Scores, PIT Internality Bias Scores, and SRIRT Recall Bias Scores for Normal, Bipolar-Manic and Bipolar-Depressed Participants.**

Implicit Tests	Group		
	Normal	Depressed	Manic
EST Interference Mean Scores for Depression-Related Words	1.63	14.44	14.9
PIT Internality Bias Mean Scores	2.07	-3	-1.6
SRIRT Recall Bias Mean Scores	3.2	-3.74	-2.34

Pearson correlations were calculated between the EST Interference Indices for depression-related words (averaged across presentation), the Internality Bias scores for the PIT (calculated by subtracting scores for negative items from scores for positive items, as already described), and the Recall Bias scores for the SRIRT (calculated by subtracting the number of negative items recalled from the number of positive items recalled). Presented graphically in Figure 10 (p. 158) these data reveal that on implicit tests the bipolar-mania and depression groups have very similar response profiles, which are in marked contrast to the normal control group performances. Significant correlations in the expected directions were found between Stroop Interference and PIT Internality Bias mean scores ( $r = -0.46, p < 0.001$ ), between Stroop Interference and SRIRT Recall Bias mean scores ( $r = -0.54, p < 0.001$ ), and between PIT Bias and SRIRT Bias mean scores ( $r = 0.65, p < 0.001$ ). However, none of these correlations approached significance when the data were analysed for each group alone.



**Figure 10. Mean PIT Internality Bias Scores, SRIRT Recall Bias Scores and EST Interference Scores (Depression-Related Words), for Normal, Bipolar-Depressed, and Bipolar-Manic Groups.**





## **DISCUSSION**

### **Chapter Nine.**

#### **Interpretation of Results and Ramifications Thereof.**

##### **Interpretation of the Present Findings.**

The findings of the present investigation are sufficiently unequivocal to be accepted as consistent with the broad hypotheses outlined at the end of chapter five. That is, data from the present investigation is compatible with the notion that low self-esteem (indicated by depressotypic attributional style, selective attention for negative trait words, and delayed colour naming of depression-related words) is associated with both the manic and the depressed manifestations of bipolar psychopathology. However in mania, protective cognitive processes, which can be detected by certain implicit tasks, sometimes disguise it. The precise predictions relating to participants' performances on particular tests, (as specified in chapter six) were also largely realised. These findings are discussed in more detail below.

##### **Bipolar-Depressed Participants' Results.**

The results obtained from the current bipolar-depressed participants are in accord with data from previous studies involving unipolar-depressed patients. The depressed bipolar respondents tested in the present study attributed negative events more than positive events to self on both the parallel form Attributional Style

Questionnaire and the Pragmatic Inference Task. That is, (like the healthy controls) they performed in a similar fashion on both transparent and opaque tests. This observation is consistent with Robin and Hayes' (1995) results from unipolar patients, with Winters and Neale's (1985) observations of remitted manic patients, and with the Alloy/Reilly-Harrington teams' depressed bipolar spectrum patients (Alloy et. al., 1999; Reilly-Harrington, et al., 1999). Also, the current bipolar-depressed participants made excessively global and stable attributions for negative events, a finding which is compatible with the earlier data from Robin and Hayes (1995), and showed slowed colour-naming for depression-related words as did the unipolar participants in the Gotlib and Hammen Stroop study (1995). Similarly, like previous unipolar-depressed respondents (Davenport et al., 1979; Dent & Teasdale, 1988; Hammen et al., 1985; Williams et al., 1990), during the self-referent recall tasks the current depressed bipolar group endorsed more negative trait words in comparison to healthy control subjects, and also recalled more negative trait words. That is, their performance was equally depressotypic on both opaque and transparent elements of the SRIRT.

These findings should not be taken to indicate that bipolar depression is identical to unipolar depression, since unipolar-bipolar discrepancies have been reported on a number of clinical variables, as previously discussed (chapter two). Differences in anxiety and agitation, (both of which are higher in depressed unipolar patients), and psychomotor retardation, mood lability and sleep disturbance

(which are all higher in depressed bipolar patients), have also been documented; see Goodwin & Jamison, 1990, chapter 3). However, it seems that bipolar depression, like unipolar depression, is characterised by a relatively negative self-schema, a corresponding tendency to blame the self for negative experiences, and a propensity to attend selectively to depression-related stimuli.

### Bipolar-Manic Participants' Results.

The data from the bipolar-manic participants are more complex. As predicted, on some of the tests (where the nature of the task was fairly obvious) their responses were similar to those of the normal controls, whereas on other, less obvious tests, their responses were much more similar to those of the bipolar-depressed participants. Of the three groups tested, the bipolar group alone demonstrated such a performance shift between obvious and non-obvious measures.

### Manic Participants' Response Shift; ASQ(pf) – PIT.

Like the healthy controls, the bipolar-manic respondents showed a robust self-serving bias on the ASQpf, attributing positive events more than negative events to internal, global and stable causes, a finding consistent with that of Alloy, et al.'s (1999) hypomanic students and Tracey et al.'s (1992) remitted bipolar participants. On the PIT (an opaque version of the same test) however, the manic bipolar candidates in the present investigation indicated a more deprecatory bias, attributing negative events more



to self, an effect that is consistent with Winters and Neale's (1985) PIT data from manic patients in remission.

### Manic Participants' Response Shift; SRIRT.

A further demonstration of this shift in response to the relative transparency of measures can be seen by comparison of data from the opaque and transparent components of the Self-Referent Incidental Recall Test (SRIRT). Here, the current bipolar-manic participants, like the healthy control participants, initially endorsed more positive than negative trait words, but when subsequently required to recall these words, they recalled more negative than positive words, this time like the bipolar-depressed candidates. Thus, it appears that the bipolar-manics overtly portray a positive view of themselves (as reflected in their pattern of adjective endorsements) whereas their recall data indicate that their self-schemata are perhaps much more orientated to failure, and are thus more akin to their bipolar-depressed peers.

### Manic Participants' Stroop Performance.

On the emotional Stroop (another non-transparent measure) the bipolar-manic respondents again performed like bipolar-depressed participants and hypomanic normal participants in previous studies (Bentall & Thompson, 1990; French et al., 1996). The current bipolar-manics showed delayed colour-naming for depression-related words but not for euphoria-related words. Interestingly, this group also demonstrated most variability in

Stroop times. Since this was evident across all conditions (neutral, depression, and euphoria-related words) this anomaly might reflect the extreme distractibility of actively manic patients frequently observed in clinical settings.

The radical shift in response patterns across the various opaque and transparent tasks, which was observed exclusively in the bipolar-manic patients, can be taken as clear support for both the functional nature of manic episodes and the capacity of such measures to penetrate this defence.

### Ramifications Of The Present Findings.

#### Bipolar Participants' Self-Representations.

The observation that some of the findings from the bipolar-manic individuals, (namely the PIT, the Stroop and the recall data from the SRIRT) match those from the bipolar-depressed individuals, suggests some degree of psychological continuity between episodes of depression and episodes of mania. Indeed, the results from all three of these opaque tasks suggest that, as in unipolar affective disorders, low self-esteem plays an important role in the genesis of manic symptomatology. However, by definition, none of these tests required subjects to make an explicit evaluation of self.

The suggestion of a fundamental continuity between manic and depressive bipolar symptomatology implicates the notion of a single construct of latent low self-esteem. However, as noted in

chapter five, some authors have opined that self-representations are much more complex or multifaceted (Higgins, 1987; Markus and Nurius, 1986). As such they cannot be reduced to a single construct of self-esteem and this principle may apply as much to latent representations of the self as to those which are more explicit. Thus, the nonsignificant within-group correlations between the implicit measures in the present study (i.e. the Stroop interference indices for depression-related words, the PIT internality bias scores, and the SRIRT recall bias scores), might be explained by supposing that they reflect different kinds of latent self-representations.

Alternatively, it is possible that these tests are differentially sensitive to a single latent domain of self-esteem but that other cognitive processes, as yet unspecified, also affect them. Further empirical exploration of this complex concept using instruments that access the different facets of the self is clearly warranted.

### Bipolar Mania and Unstable Self-Schema.

The positive views of the self elicited from the bipolar-manic patients on those tasks that required an explicit judgement of self-worth when contrasted with the findings from the implicit tests are broadly in accord with Neale's (1988) ideas of the manic defense (and those of his psychodynamic forefathers). According to Neale, the grandiose ideation of manic patients can be viewed as a defensive response to any intensification of low self-worth, which occurs in individuals who experience unstable self-esteem. Yet, the



current findings also suggest that some elaboration of his original account might be required.

### Elaboration Of Neale's Account and Implications Thereof.

According to Neale (1988), compensatory grandiose ideas are a strategy for achieving congruence between real and ideal selves and can lead to a fierce elevation of mood. Such grandiosity is precipitated by decreases in self-regard, and can lead to mania, which in turn serves to block distressing cognitions from the consciousness.

The data from the present study fits Neale's suggestion that instability of self-esteem might reflect the existence of an implicit negative self-schema that remains unprimed under most circumstances. From such a premise it could be argued that when implicitly negative self-schema are primed (for example by a stressful life event), bipolar individuals either consciously experience feelings of low self worth, in which case they become depressed, or the defensive responses are triggered and they become grandiose and manic. This elaboration of Neale's premise has two clear implications.

Firstly, remitted manic patients might be expected to behave in a manner similar to unipolar depressed and bipolar-manic patients on implicit, but not on explicit, measures of self-esteem. Data from remitted bipolar patients in Tracey et al.'s ASQ study, and Winters and Neale's (1985) PIT study are consistent with this.

So too are the Stroop results from hypomanic normal participants (Bentall & Thompson, 1990; French et al., 1996). Less compatible however are accounts of bipolar spectrum individuals responding in a depressive-like manner to the transparent ASQ (Alloy et al., 1999; Reilly-Harrington et al., 1999) although it is not clear if the bipolar patients were in remitted or manic phase during testing. Replication of the present study with the addition of a remitted bipolar participant cohort needs to be carried out to test this prediction further.

The second implication of Neale's account concerns the intermittent activation of the defence mechanism. As it presently stands, the functional account of mania lacks any precise explanation as to why the defense is sometimes triggered whereas at other times it is not. Clearly this may constitute a serious limitation to the manic-defence argument, the ramifications thereof are discussed next.

### Limitations of the Present Study.

#### Activation of the Manic Defence.

That mania only sometimes serves to protect against the consequences of primed negative self-schema implies some mechanism whereby the manic defense is switched on. What actually causes the manic defence to erupt? Patently, it does not automatically come into play whenever dormant feelings of low-self esteem are activated, since in some instances priming of an

implicitly negative self-schema renders the bipolar individual depressed. Thus on some occasions it appears that the defence fails, arrives too late, or is an ineffective defence against latent feelings of unbearably low self-esteem.

This fits with the unfortunately high rates of suicide in bipolar patients. However, a closer examination of the relationship between an individual's risk of suicide and the course of their bipolar disorder is largely consistent with the proposition that mania may have a protective function. Not only is suicide considerably more frequent in depressive bipolar episodes than in manic episodes, but it is most frequent in the initial stage of the illness when depression predominates (Dilsaver et al., 1994). Less consistent is the observation (made in chapter 1) that suicide is at least as high in a depressed bipolar group (whose population has recourse to the manic defence) as in a depressed unipolar group (whose population does not). One explanation for this might be that bipolar patients do not evolve other more appropriate coping strategies. Perhaps when their manic defence fails the depressed bipolar individual has few other options but to seek the permanent escape from intolerable psychic threat. As Motto points out,

“...minimum pathology in a suicidal person bereft of strengths may be lethal, while severe pathology in a person with unusual strengths may constitute only moderate risk”. (Motto, 1975, p. 239).



However, few are likely to describe the bipolar defence (a manic episode) as a “strength”, and such conjecture aside, we retain unresolved the question of the specific triggers of dysthymic or euthymic bipolar episodes.

### Interacting Processes.

It seems likely that both intra-organismic variables (such as the state of explicit self-esteem at the point of exposure to certain stressors, disruption of circadian rhythms), and extra-organismic variables (particular types of stressors – e.g. negative life events, inter-personal conflict) play a role in the intermittent activation of the manic defence. The picture could be further complicated by the reciprocal influences of these elements. For example, recent studies have begun an exploration of the interaction of depressogenic cognitive processes and life events as possible predictors of bipolar episodes (e.g. Alloy et al, 1999; Reilly-Harrington et al, 1999) but as yet have failed to identify specific mechanisms underlying the demarcation of depressed and manic episodes.

Fluctuations in bipolar symptom manifestations however may yet be found to reflect circular processes between various functions or abnormalities. Thus, even where mania is not apparent, it is possible that a defensive system is still occurring but the processes (of negative self-representation and attributional bias) are occluded by another coexistent mechanism (such as a deficit in meta representation or neuropsychological processing).

### Processes in Flux.

Just as the mechanism that activates, or fails to activate, the manic defence is unlikely to be a simple binary device, it is also unlikely to remain static over time. Unfortunately, cross-sectional research methodologies provide a mere snap-shot of the ever-changing processes of psychopathology. It is possible that the cognitive and affective processes which this (and related studies) have attempted to measure are in constant flux. Bentall (1999) has speculated that rapid fluctuations in psychological state might be typical of individuals with severe psychopathologies. This is consistent with the finding in bipolar patients of multiple mood changes with no clear pattern (Gottschalk et al., 1995, discussed in chapter 1). It may be that such apparent turmoil belies variations in symptoms that are in fact deterministic but too complex to identify or predict. Bentall (1999) suggests that under these conditions, changes over time conspire with other dynamic processes to produce a state of 'formal chaos'. Thus a pattern might exist but it is obscured in the maelstrom (or possibly is too complicated to identify with existing instruments). Nonlinear dynamic systems theory, which seeks to articulate these sorts of fluctuations (the chaotic interaction of even a few elements), has as yet not been adopted in the study of psychopathology as a whole (Bentall, 1999), although Ciompi (1998) has suggested that psychotic phenomena do perform in this manner. An ambitious attempt to portray these processes in bipolar disorder, entailing the construction of complex mathematical equations to describe chaotic behaviour, is presently

underway (Bentall, personal communication, Dec. 1999). Detailed longitudinal investigations will be necessary to disambiguate this intricate domain. Future research, whether through traditional or novel methodologies, could benefit from increased attention to the particular constructs under scrutiny, their interactions, and variations over time.

### Absence of Manic Defence in Unipolar Depressives.

The current evidence leaves unresolved several other questions. The first is closely related to the conundrum of what activates the manic defence in bipolar disorder.

It remains unclear why all depressions (unipolar as well as bipolar) do not become manifest as defensive manias. Why are unipolar depressed individuals unable to access this method of escaping psychic pain? Given the unipolar-bipolar similarities, there is no clear explanation for the total absence of recourse to the manic defense by individuals who are presumably in as much need of a protective strategy as their bipolar peers. One simple explanation might be that there exist more fundamental differences between unipolar and bipolar affective disorders. If this were the case it may challenge the notion of an affective spectrum overlap. Alternatively, it could be that some unipolar patients do activate a defensive strategy but that this phenomenon is obscured by the complex interactions of other dynamic systems (i.e. in line with nonlinear dynamic systems theory, already mentioned).



Neale's own explanation for this conundrum returns us to the issue of alternating symptomatic episodes in the bipolar spectrum of disorders. Just as bipolar episodes are not always manic, so there exists a hypothetical choice of manifestatory routes for unipolar depressives. Neale (1988) suggests that this discriminatory mechanism might be determined by the intensity of threat to the self. Perhaps a manic defense can only cope with so much. Thus an excess of negative affect could strip the individual of their protective repertoire to leave them overwhelmed by a depression. This takes us back to Garety and Freeman's (1999) criticism that motivational theories of mania are inadequate, although Freud himself preempted this observation by some 80 years. Freud (1917[1950]) admitted that this subconscious attempt to overcome inner turmoil or otherwise sustain self-esteem can be transitory and is rarely accomplished in full. Post-manic depression could therefore reflect the superficiality of the manic defence. Having concealed an interior distress through excesses of activity, or used denial and regression to cope with the loss of an ambivalently loved object, the defence is unsustainable over prolonged periods. The truth emerges and the individual manifests the symptoms of unmitigated depression (Healy & Williams, 1997).

Neale wonders whether vulnerable individuals are already predisposed to a manic defence by way of their idiosyncratic use of elaborate fantasy to avoid real or perceived hassle and threat (as outlined in chapter 3). Not only do these questions remain unresolved, but Neale's (1988) ideas remain untested as his

contribution to this debate was primarily theoretical, and inadequately supported by empirical evidence. Also unresolved in the context of bipolar defence mechanisms are the considerations of consciousness and neuropsychological functioning. Since such weighty issues are well beyond the present remit, the remainder of this chapter returns to a consideration of the similarities between mania and paranoia, with reference to the present bipolar data.

### Parallels Between Mania And Persecutory Delusions.

Comparisons between bipolar disorder and other psychotic disorders, and between the “manic-defence” notion of bipolar disorder and the “delusion-as-defence” contention in paranoia have already been made (see chapters 4 & 5 respectively). Some note must now be made of the similarities between the bipolar-manic participants in the current study and participants suffering from persecutory delusions studied in previous investigations.

Persecutory deluded (or paranoid) patients tested on the ASQ have consistently shown an excessively powerful self-serving bias (Kaney & Bentall, 1989; Candido & Romney, 1990; Fear et al., 1996, Sharp et al., 1997). However, Lyon et al. (1994) tested paranoid individuals with both the ASQ (pf) and the PIT and found that, like the bipolar-manic participants in the present study, they made self-denigratory responses on the opaque measure. Although Peters, Day, and Garety (1997) failed to replicate, these results were broadly consistent with the ASQ(pf) and PIT performances of

individuals experiencing a first-episode of paranoia reported in a recent study by Krstev, Jackson and Maude (1999).

Paranoid respondents have also been shown to exhibit abnormal responses on the emotional Stroop (Bentall & Kaney, 1989; Kinderman, 1994) and on the SRIRT (Bentall & Kaney, 1996), behaving akin to depressed participants in both instances. Moreover, paranoia and mania show some similarity at the level of phenomenology, as grandiose delusions are often noted in paranoid patients (Zigler & Glick, 1988) and persecutory delusions are not exceptional in mania (Goodwin & Jamison, 1990).

Not surprisingly, these observations are consistent with Zigler and Glick's (1988) hypothesis that paranoia and mania are the product of defences, which are closely related at a cognitive level. Comparison of the present data with that from previous studies of paranoid respondents indicates that the self-serving bias on explicit attributional measures may be more extreme in paranoid than manic patients, as paranoid respondents have consistently shown greater bias scores than healthy control participants. The evidence of abnormal self-referent encoding obtained from paranoid patients by Bentall and Kaney (1996) however, was more equivocal than findings obtained from the manic patients in the present study. Further investigation of the similarities and differences between these conditions is warranted.



### Towards a Synthesis of Models.

Since depression, mania, and paranoia all seem to involve abnormalities in the domains of attribution and self-representation, it may eventually be possible to construct a unified psychological model of these conditions. However, any such model would be likely to draw upon the ideas of all opposing camps and it is unlikely that the present manic-defence theory will suffice to account for all psychopathologies. Of course motivational and defect theories are not necessarily mutually exclusive and by Neale's own account the manic defence consists of a specific motivational mechanism resulting from a fundamental deficit in cognitive processing (Neale, 1988).

As Frith (1999) points out, differences in the prevailing cognitive accounts of delusions (outlined in chapter 5) are not improbable since each applies to a different aspect of false belief. Specifically, the defence model (e.g. Bentall et al., 1994; Kinderman & Bentall, 1997; Lyon et al., 1994) is based on attributional and self-representation anomalies and aimed at persecutory and grandiose delusions. The meta-representation hypothesis, or theory of mind deficit (e.g. Corcoran, Mercer, & Frith, 1995), applies to delusions involving the mental state of others which includes delusions of reference and of persecution. A separate theory of corollary discharge (or self-monitoring deficit) has been forwarded to account for delusions of control (Frith & Done, 1989). Finally, the reasoning deficit or perceptual processing

paradigms advocated by Garety and co-workers (Freeman et al., 1998) could be applied to all the many kinds of delusions (the above delusions of grandiosity, persecution, and reference, as well as delusions of control). It is consequently likely that the functional theory of cognitive processing abnormalities presently under review is adequate to account for some delusional states (that is delusions of persecution in schizophrenia, as attested by Lyon, et al, 1994, and delusions of grandiosity in bipolar mania as evidenced by the present results), but perhaps not all. The spasmodic appearance of manic episodes interspersed with depressed episodes could be viewed as consistent with such a weakened manic-defence hypothesis.

Alternatively these phenomena are not inconsistent with the idea that although functional forces are at play, sometimes they may be obscured by the complex or chaotic interrelationship of other cognitive or neuropsychological mechanisms (Bentall, 2000). It is thus possible that various psychopathologies (i.e. bipolar disorder, depression or paranoia) might reflect various combinations of similar abnormalities which interact to cause vulnerabilities to discrete symptom expression (Bentall, 1990). It thus seems not unreasonable that the further delineation of the complex processes and interrelations implicated in opposing models of psychological disorder (i.e. defence or defect paradigms) may contribute to an eventual "meta-model" of psychopathology. Such a synthesis eventually may prove capable of uniting the affective and psychotic continuums. Lofty aspirations aside, the next chapter concerns itself

with a more mundane task. Certain methodological limitations of the present study are highlighted and an attempt is made to address some of the obvious reservations.



## **Chapter Ten.**

### **Methodological Reservations.**

In general the present study could be viewed as being of potential conceptual and clinical influence in the nascent arena of bipolar psychopathology. As predicted, the non-transparent measures (the PIT, EST, and recall component of the SRIRT) succeeded in eluding the optimistic self-representations, by disguising the self-attributional (or self-endorsing) aspects of the task. Thus the opaque tests managed to activate the otherwise hidden pessimistic self-image of the bipolar-manic patients. However various methodological shortcomings, as outlined below, limit the conclusions that can be drawn from of the present investigation.

#### **Lack of Remitted Bipolar Control Group.**

First, the advantage of employing a remitted bipolar cohort in addition to the acutely ill group has already been alluded to. This may have helped tease out the aetiological significance of the social cognitions demonstrated by the manic bipolars. It would be useful to ascertain if their “pathological” attributions precede the symptomatic episode or disappear with recovery. If coincident with the illness rather than antecedent these cognitive abnormalities may be either part of the symptom cluster or epiphenomena. In this case, use of the overt tests on remitted manic bipolar patients (similar to the PIT study by Winters & Neale, 1985) to detect dysfunctional

attributions could be taken as partial support for the notion of latent self-schemata bias. Use of a depressed unipolar cohort as an alternative psychiatric control would also have been useful, although perhaps less crucial given the relative proliferation of attributional and self-representation studies with these patients already in existence.

As already indicated, it was initially proposed to empirically test the manic-defence hypothesis by way of a longitudinal study. This would have entailed enlisting bipolar participants to act as their own controls through repeated administrations of the test battery during the natural occurrence of mania and depression, and during periods of remission. Unfortunately such a project was deemed impractical and outwith available time constraints, although recent longitudinal research with subsyndromal bipolar patients (Alloy, et. al., 1999) appears to have undertaken a similar enterprise in a relatively discrete time frame. However, despite this optimal methodology, Alloy et al. have been unable fully to resolve the trait or state debate regarding observed phenomena. Also unaccomplished is the clarification of complex interrelationships between symptoms and cognitive processes, speculated in the previous chapter. If some of these processes appear immeasurable, the way forward might lie in the perfection of more precise measures.

### Participant Recruitment.

Perhaps the decision to opt for a less ambitious design for the present study was vindicated by difficulties in recruiting psychiatric participants. A longitudinal design would have necessitated a substantially increased number of bipolar participants at initial testing to offset anticipated rates of participant drop-out. Participant loss due to protracted cycle length or infrequent manic episodes would also be probable.

### Control for Chronicity or Cycle Length.

A within subjects longitudinal design, however, would have transcended the difficulty in controlling for chronicity or variation in cyclical patterns between groups. As it was, the author was unable to obtain sufficient information to control for between group differences in the clinical groups in terms of onset of psychopathology (late versus early), or cycle length (long versus rapid cyclers).

### Sample Bias.

This leads on to the question of sample bias. Given the issues of poor bipolar participant compliance discussed previously (chapter 7), it is possible that the results obtained from the bipolar-manic group in the current investigation are not entirely representative. It could be that they are confounded by inherent differences in manic refusers (who were perhaps not adequately sampled) and manic acceptors (who may have been over-



represented). In general however, the apparent 'wastage' of potential bipolar–manic participants was due to inadequate preliminary identification of potential manic candidates by mental health colleagues or ward staff, in conjunction with strictly adhered to inclusion and exclusion criterion, as outlined in the methodology.

### Sample Size.

An increased sample size would help reduce the effects of potential sample bias. However, this was beyond the scope of the present study and, from a statistical point of view the high levels of significance obtained on analyses suggests that the sample size was adequate.

### Psychometric Properties of the ASQpf.

A further limitation concerns the psychometric properties of the parallel form Attributional Style Questionnaire (ASQpf). Reviews of the psychometric properties of attribution questionnaires, particularly the ASQ, have persistently pointed to poor reliability of these scales (Reivich, 1995; Tennen & Herzemberger, 1985). More recently the internal reliability of the parallel form ASQ (and the PIT) have also been challenged (Krstev, Jackson & Maude, 1999). Unfortunately it is the internality dimension, of central importance to the proposition under consideration here, which is also the least satisfactory. Nevertheless the lack of viable alternatives has meant that these scales have continued to be used, as in the present study.

### The Internal Situational Attributional Style Questionnaire.

A solution to the above difficulties may be at hand. Recently Kinderman and Bentall (1996) have proposed a novel measure that distinguishes between two types of external attributions, those that implicate situational factors and those that implicate the actions of others. Their Internal, Personal and Situational Attributions Questionnaire (IPSAQ; Kinderman & Bentall, 1996) has good psychometric properties and has demonstrated reliable assessments of attributional style separate from the ASQ. Specifically, the IPSAQ provides measures of externalising bias (the inclination to attribute negative rather than positive events to external causes) and personalising bias (the tendency to make personal-external rather than situational-external attributions for negative events), biases which were associated with ASQ internality scores and with depression.

A more recent cross-sectional study using the IPSAQ found that depressed patients tended to attribute negative social events to internal and self-blaming causes. Individuals with persecutory delusions and healthy controls generally avoided such self-blaming proclivities. However where the nonpatient control participants tended to choose situational or circumstantial external explanations, the paranoid group tended to choose external attributions that located blame in other people (Kinderman & Bentall, 1997). Given that these external-personal attributions are linked to persecutory ideation (blaming others for misfortune), it would be worth

assessing manic patients on this transparent measure in the expectation that they would demonstrate a self-serving bias by attributing negative social events primarily to external-situational causes. Unfortunately this new test of explanatory style was unavailable when the present methodology was conceived and psychometric shortcomings aside, the inherent elegance of the current design relies on the direct equivalence of items from the PIT and ASQpf making them effectively opaque and transparent versions of the same test. This would not have been the case with the IPSAQ.

#### Other Methodological Shortcomings.

Methodological problems such as small sample size, and difficulties in matching participants for symptom severity are perennially encountered by authors who attempt to contribute to this corpus of research (Garety & Freeman, 1999). Other limitations of this (and previous studies) include the practicalities of controlling for the effects of prolonged use, and level, of medication, or disorder chronicity and length of hospitalisation. Increased attention to the severity of negative symptoms (given that the current battery of measures entailed substantial motivation and cognitive demand), or further screening for organic abnormalities might also be considered in future undertakings (Krstev, et al. 1999). However, since, every possible attempt was made to preserve the integrity of the restricted experimental design adopted, the difficulties encountered do not appear to be beyond those



typical when working with research participants who experience a severe psychopathology.

## Chapter Eleven.

### Clinical Implications of Current Bipolar Research.

“What mechanisms does the individual use to protect and defend his self-concept against the onslaughts that incessantly assail it?” Morris Rosenberg, 1997, p x.

The current evidence suggests that even in manic states, indirect tests of emotional functioning often produce profiles resembling those of depressive rather than euthymic individuals. This confirms the impression often obtained in clinical settings, that although self-esteem is especially impaired during episodes of bipolar-depression, it is often fragile or severely compromised in manic episodes as well (e.g. Kotin & Goodwin, 1972). This observation seems intricately linked with the bipolar patient's experience of self in the context of an illness that is often frightening, at times disconcerting, on occasion exhilarating, but invariably unpredictable. It is not uncommon for these individuals, when in remission or in the wake of a manic episode, to make explicit reference to a change in their sense of self (Goodwin & Jamison, 1990). Confusion between their manic, depressed and remitted personae, and problems identifying the “true” self are thus not surprising. This is especially so given the adverse experiences of emotional flatness or side-effects consequent on medication in contrast to the sometimes heady exuberance, sense of superiority, or

boundless energy and creativity encountered within some manic periods.

### Self and Illness in Bipolar Disorder.

Motivational theories of psychoses assume patients are cognisant of their illness but are motivated to deceive either themselves or others about it. That is, at some level of consciousness they attempt to defend their self-esteem, evade depression, or preserve a positive perspective. Lam et al. (1999) draw on clinical observations for their account of strategies typically employed by bipolar individuals to define themselves in relation to their disorder. They describe three extreme paradigms (use of denial, externalisation, or an exclusively medical model), all of which imply at least some paucity of insight and have inherent drawbacks for the afflicted individual. These strategies, outlined in detail below, lend themselves to motivational accounts and are broadly consistent with the ideas of unstable self-representation currently under review.

### Denial of Illness.

The observation by Ghaemi (1997) that poor insight is associated with grandiosity has some bearing on the first of Lam et al.'s hypothetical self-preservation strategies. This relates to the complete denial of the very existence of a disorder (or at least the individual's own disorder). Concordant with the manic state, individuals who adopt this strategy maintain that there is nothing



amiss, claiming rather that they have special powers and creativity, or are otherwise superior to others and, as such, do not have to abide by the same rules of conduct. Here it is important to reflect upon the link between creativity and leadership, an association already established in those with bipolar disorder.

### Creativity and Leadership in Bipolar Individuals,

There is strong scientific and biographical evidence to suggest that affective illnesses might have contributed to the very real accomplishments of countless historical figures. A list of those renowned for their literary legacy and believed to suffer from a bipolar disorder would have to include Charles Lamb, Ruskin, Samuel Taylor Coleridge, Edgar Allan Poe, Percy Bysshe Shelley (known at school as “Mad Shelley”) and his close friend Lord Byron (known as “Mad Lord Byron”). Not to be forgotten in this category is Virginia Woolf, who we already encountered in the review of bipolar suicide. Although composers tend not to demonstrate a full-blown bipolar disorder (or manic-depression), Robert Schumann, Peter Tchaikovsky, Gustav Mahler, Alexander Scriabin, and Sergey Rachmaninoff are all documented to have experienced symptoms within a bipolar spectrum. We now move from melody to mastery.

Great heroes of political or military leadership include Oliver Cromwell, Napoleon Bonaparte, Benito Mussolini, Lord Nelson, Abraham Lincoln, and Winston Churchill, all of whom were acutely bipolar (Goodwin & Jamison, 1990, chapter 14). It must be

stressed however that despite an increasing body of knowledge attesting to the connection between creativity or leadership and bipolar affective disorder, many eminent artists, writers and leaders are free of significant psychopathology. Unfortunately, and conversely, most bipolar individuals are not extraordinarily creative or masterful, although bursts of productivity or artistry can accompany bouts of mania (Lam, et al., 1999). Of course the advisability or reliability of retrospectively (and posthumously) diagnosing an affective disorder in historical figures should be questioned. Further, the labeling as manic-depressive all feats of unusual creativity, accomplishment, or eccentricity will inevitably both diminish the notion of artistic individuality and trivialise what is often a painful, disabling and sometimes fatal disorder.

Hopefully, the advent of modern prophylactic treatment which brings with it (for some) the option of an escape from the extremes of turmoil and despair, will not entirely extinguish bipolar creativity. However, as Goodwin and Jamison (1990, chapter 14) point out, such a choice was unavailable to the writers, musicians and leaders mentioned above and perhaps some suffering must always accompany great artistic achievement.

Conceivably related to this is the observation (in a clinical population) that medication non-adherence is particularly common in the first year of prophylaxis, and especially so for individuals who complain of missing the “highs” (Goodwin & Jamison, 1990, chapter 25). It also tends to be fear of depression rather than fear of mania that motivates compliance (Scott, 1995). Inevitably, the

reverse pertains for healthy relatives. They dread manic phases more than depressive phases (Targum et al., 1979), perceive the disruption of their relative's disorder as greater than do those directly afflicted, and it is usually they who most appreciate the benefits of treatment (Scott, 1995).

### Externalisation.

The second of Lam et al.'s self-representational strategies, like the first, is conceivably adopted to ameliorate the prevailing stigma attached to this (as to other) mental health problems. Lam et al. (1999) suggest that here the individual may attribute their problems to an external factor, such as substance misuse or the effects of prescribed medication. Explanations of their plight implicating stress or trauma, employed to enhance social acceptance would also broadly fit this category, which is closely akin to the self-enhancing attributional tendency evidenced by bipolar-manics on the ASQpf of the current study.

### Exclusively Physiological or Medical Model.

The final strategy described by Lam and co-authors is the zealous adoption of a medical model, which negates any psychological or environmental contributory factors. Clearly this strategy, like the previous ones, whilst attempting to protect against a confused sense of self, a threatened self-esteem or an imminent depression, may also result in disadvantages for the person so disposed. Such extreme approaches can be used as explanations for



dysfunctional conduct, or excuses for bad behaviour. Just as the aforementioned historical figures were both blessed with genius and cursed by bouts of insanity, one might speculate that they were less than conventional in behaviour and outlook. Perhaps their less notorious bipolar peers who rarely become rule makers also believe themselves above or beyond normal conventions of propriety (i.e. are rule breakers).

In terms of clinical implication, such dysfunctional self-representation might also serve to abnegate any responsibility for improvement. Denial of possible environmental or intrapersonal determinants is also likely to engender disempowerment, social isolation, or excessive pessimism. Such inflexible models may prove to be self-fulfilling and are likely to impede accomplishments. As antithetical to the rationales of modern cognitive therapies, which advocate psychosocial change, these beliefs may act as obstacles to rehabilitation. Assessment of the patient's self-representation (and the family's views of the disorder and its management), is thus a vital component of initial clinical evaluations, and modification thereof remains an ongoing therapeutic focus.

Lam et al.'s (1999) account of maladaptive self-enhancement strategies evoked by bipolar patients whilst in remission is consistent with the broad clinical implications of the present results. Given the presence of an enduring disturbance of bipolar self-representation, techniques to enhance or stabilise fluctuating self-

esteem should be given increased emphasis in the evolving psychotherapies.

### Cognitive Behaviour Therapy.

“The growth of biological reductionism and the dominance of the neuroleptics have been the key themes in the management of psychosis over the last 20 – 30 years. It is a matter of great pride that British clinical psychology and social psychiatry have been responsible for bringing the person and his or her social context back to centre stage, and for taking a lead role in developing effective psychosocial and cognitive treatments.”

(Max Birchwood, 1999, p. 315).

In the introduction to this thesis it was mentioned that there has been comparatively little research into the role of psychological processes in bipolar affective disorder. There is a comparable dearth of attempts to evaluate the effectiveness of psychological interventions (APA, 1994b) and thus relatively little empirical evidence in support of psychotherapy for bipolar individuals (Lam, 1991). This is surprising in the light of alarmingly high rates of medication non-response and non-compliance (discussed later).

Many bipolar patients may need specific therapeutic help to resolve the panoply of difficulties relating to their disorder, although the form, intensity, and focus of psychotherapeutic treatment will vary over time for each individual (APA, 1994b).

Most individuals and those close to them will struggle with some of the issues listed below.

- 1) Emotional consequences of periods of major mood disorder and diagnosis of a chronic mental illness.
- 2) Developmental deviations and delays caused by past episodes.
- 3) Problems associated with stigmatisation.
- 4) Problems regulating self-esteem.
- 5) Fears of recurrence and consequent inhibition of normal psychosocial functioning.
- 6) Interpersonal difficulties.
- 7) Marriage, family, childbearing and parenting issues.
- 8) Academic, occupational and financial problems that arise from reckless, violent, withdrawn, or bizarre behaviour that may occur during symptomatic episodes.

### Applicability of Cognitive Therapy.

Cognitive-behaviour therapy (CBT) might be considered as a candidate treatment, as it appears to be a successful therapy for both unipolar depression (Gotlib & Hammen, 1992; Williams, 1992) and the positive symptoms of psychosis (Chamberlain, & Dunn, 1994; Drury, Birchwood, Cochrane, & MacMillan, 1996; Freeman et al, 1998; Garety, et al., 1994; Tarrier, Beckett, Harwood, Baker, Yusupoff, & Ugarteburu, 1993; Tarrier, et al., 1998; Sensky, et al., 2000). Further, CBT can be delivered in individual or group format and with couples or families in the form of psychosocial interventions (Scott, 1996; Lam, et al., 1999). An



ambitious multi-centre evaluation of individual CBT treatment with bipolar patients is presently underway (R. P. Bentall, personal communication, December 1999) but as yet data from a large-scale randomised controlled trial of CBT for bipolar disorder is unavailable (Scott, 1995).

Whilst the advent of new pharmacological treatments “made psychotherapy possible”, in their turn, modern psychological interventions (such as CBT) herald novel strategies to address the perennial problems involved in the medical management of bipolar patients. Various authors (Scott, 1995, & 1996b; Lam et al., 1999) have considered the probable utility of CBT for bipolar disorder and have argued that this approach might be used to ameliorate depression, to enhance coping, to prevent relapse, and to improve compliance with medication. (The applicability of another candidate psychotherapy, Interpersonal and Social Rhythm Therapy (IPSRT), will be discussed later in this chapter.)

### Bipolar Specific CBT Tasks.

CBT interventions have been designed to address themes pertinent or specific to bipolar disorder, which correspond with those listed above. Cognitive-behaviour therapists encourage their bipolar clients to marshal their problems into three broad categories (Scott, 1996a) as follows.

Intrapersonal problems. Consistent with the present investigation, a primary target here is that of low self-esteem, although threats to self-perception related to life-long reliance on medication would fit

Interpersonal problems. These include lack of social support systems and difficulties in relationships with family members.

Basic problems. These tend to focus on biological or environmental factors, and include symptom frequency, severity or course of the individual's condition, early warning signs, and difficulties coping with work, or housing problems etc.

Organisation of problems in this manner emphasises the normal-pathological continuum, and moves away from a strictly medical model. Thus although many traumas and losses cannot be remedied by CBT, the therapist helps the bipolar client cope with them through the acquisition of both usual strategies (i.e. lifestyle enhancement, avoiding stressors) and specific strategies (i.e. time delay rules, identification of prodromal signature). This has several advantages. Whilst acknowledging the entire range of difficulties encountered by bipolar families, the therapist can avoid confrontation, encourage a psychosocial perspective and facilitate self-efficacy (Scott, 1996a; Lam, et, al., 1999). Further, the bipolar patient is less likely to confuse unrealistic losses (where the disorder or its treatment are inappropriately blamed for past or current difficulties) with realistic losses (Goodwin & Jamison, 1990, chapter 24). Amongst the various CBT strategies for bipolar disorder are education, facilitating adjustment, self-monitoring, lifestyle regulation, enhancing medication compliance and relapse prevention (detailed in Scott, 1995, 1996a, & 1996b).

### CBT as Adjunctive to Pharmacological Treatment.

**“... I cannot imagine leading a normal life without both taking lithium and being in psychotherapy. Lithium prevents my seductive but disastrous high, diminishes my depressions, clears out the wool and the webbing from my disordered thinking, slows me down, gentles me out, keeps me from ruining my career and relationship, keeps me out of hospitals, alive and makes psychotherapy possible. But ineffably, psychotherapy heals. It makes some sense of the confusion, reins in the terrifying thoughts and feelings, returns some control and hope and possibility of learning from it all. .... No pill can help me deal with the problem of not wanting to take pills; likewise, no amount of analysis alone can prevent my manias and depressions. I need both”**

**(A patient, Goodwin & Jamison, 1990, p. 725).**

**Given the undeniable physiological basis of bipolar affective disorder, it is hardly surprising that biological models and medical treatments still dominate the research agenda. However, this might also reflect the legacy of psychoanalytic writers who considered manic individuals as entirely inappropriate for psychotherapy. Fromm-Reichmann (1949), for example, is renowned for his characterisation of bipolar individuals as entirely lacking in complexity or subtlety. Scott (1995), however, points out that discouraging views relating to the efficaciousness of psychotherapy with bipolar patients were written in the pre-lithium era. Fortunately best modern practice encourages a holistic integration of biomedical**



and psychosocial accounts of disorders, both psychological and physical.

Consistent with this, there is no empirical evidence to suggest that bipolar patients can be helped by psychotherapy alone. Various authors contend that clinical experience indicates the advantages of sequential introduction of medication followed by the systematic use of CBT. Similarly, the use of psychosocial interventions as a necessary adjunct to prophylactic drug treatment is increasingly recognised (Scott, 1996a; Lam et al., 1999). Thus, the place of modern pharmacotherapy in the treatment of acute episodes remains unassailed.

Indeed CBT and other psychotherapies (e.g. IPSRT) for bipolar disorder actively endorse the importance of medication (Lam, et al, 1999). However, after acute symptoms have been assuaged and the patient's mood is stable, there is an urgent need to focus on the consequent devastation experienced by bipolar patients and their families (Scott, 1996a). Despite good premorbid functioning or apparently excellent interepisodic recovery, it is likely that those affected will require considerable assistance in coming to terms with having a chronic and recurring disorder requiring lifelong prophylactic medication (Scott, 1996a). Delivering such a service is not always easy; difficulties likely to be encountered by the novice therapist will be considered next.

### Obstacles to Treatment.

Alloy et al., (1999) confidently assert that treatment, with medication or psychotherapy, can remediate bipolar symptoms and also ameliorate, deactivate or otherwise militate against dysfunctional cognitive patterns. Despite this bold and optimistic view, various factors, not all peculiar to bipolar disorder, are likely to impede both medical and psychological therapies.

### Poor Medication Compliance and the Side Effects of Medication.

Most psychological studies of bipolar disorder fail to control for medication and treatment evaluations overlook their participants' actual rates of compliance. This area is sadly neglected and warrants further research in its own right. Unlike non-response, which has been extensively examined, poor adherence is potentially reversible by providing patients with enhanced management and the opportunity to explore compliance obstacles and strategies to overcome them (Goodwin and Jamison, 1990). An estimated 30 to 50 % of bipolar individuals do not comply with medication (Goodwin & Jamison, 1990; Basco & Rush, 1995; Kessler, et al., 1997), with one in five patients failing to adhere despite a good therapeutic outcome (Scott, 1996a).

Up to 75% of relapses are related to poor prophylaxis adherence (Scott, 1996a). This astounding estimate of non-compliance is probably related not just to issues of denial, stigma, or poor insight but also to the high incidence of noxious side effects. The APA (1994b) reports that up to 70% of patients on

lithium and up to 50% of those on carbamazepine experience side effects.

### Reinforcement Schedules Intrinsic To Bipolar Prophylactics.

Various factors appear to conspire to exacerbate the poor compliance to prophylactic medication. These include the swift disappearance of side effects on withdrawal from medication, the delayed return of symptoms, and patients' underestimate of the seriousness of their disorder. Goodwin and Jamison (1990, chapter 25) point out the special reinforcement schedules intrinsic to lithium and some other bipolar prophylactics. Patients are expected to remain on medication for an indeterminate time, much of it in a more or less normal state, with no immediate felt need for prophylaxis. Lithium, in particular, has no known intrinsic reinforcing qualities (immediate or delayed) but it is also devoid of any immediate negative reinforcer (relapse is rarely immediate on drug cessation). Medication prescription, on the other hand, is often paired with immediate adversity (e.g. hospitalisation, psychosis, interpersonal difficulties or side effects) and delayed negative events (e.g. lithium first prescribed for mania often predicts post-manic depression). Finally, as well as the escape from distressing side effects, prophylactic cessation can also be accompanied by almost immediate breakthrough hypomania; an immediate and positive reinforcer, which often contributed to non-compliance in the first place.



### Medication Non Response.

Even under optimal conditions, lithium prophylaxis will protect fewer than 50% of bipolar sufferers (Joyce, 1992) although underestimated partial or non-compliance may confound these estimates. Despite this apparently bleak picture, the advent of lithium and other drugs has undoubtedly enhanced the quality of life for many individuals (patients and those close to them), and pharmacotherapy remains the first line treatment for bipolar disorder (Scott, 1995). Thus the role of psychotherapy to enhance compliance cannot be overstated, and specific strategies to further this end can be found in Goodwin and Jamison (1990, chapter 24), Scott (1996a), and Lam et al. (1999, chapters 8 & 9). Cognitive therapies also aim to help bipolar patients achieve more structured lifestyles. Whilst such an approach can facilitate medication compliance, it is believed to have further benefits as outlined below.

### The Therapeutic Importance of Stable Routines.

The strategies of illness denial or externalisation (Lam et. al., 1999) outlined earlier will have obvious implications for the clinician attempting to engage the patient in either psychotherapy or pharmacotherapy. Adjunctive to the identification and modification of such maladaptive beliefs, cognitive therapists place a greater emphasis on the continuum between illness and normal thought and behaviour (Lam et al., 1999). Sleep hygiene, good diet, regular exercise, and other healthy regimes are recommended as a general

approach to improve self-esteem and quality of life, to enhance medication compliance, to foster a sense of empowerment, and to avoid over-stimulation. This is consistent with the argument that restoring circadian rhythms or establishing 'control' of biological factors (i.e. avoiding pervasive disruptions in sleep) is essential to the effective management of bipolar disorder (Healy & Williams, 1989; Goodwin & Jamison, 1999, chapter 25).

The notion that bipolar patients are particularly susceptible to disruptions in their social rhythms has given rise to a new form of adjunctive individual psychotherapy. Interpersonal and social rhythm therapy (IPSRT), developed by Frank et al. (1997 & 1999), was designed specifically to induce lifestyle regularity and interpersonal harmony in recovering bipolar disorder patients.

### Interpersonal and Social Rhythm Therapy.

Interpersonal and Social Rhythm Therapy (IPSRT) incorporates behavioural and environmental interventions to help stabilise irregularities of the sleep-wake cycle that are believed to trigger bipolar episodes (Frank et al., 1999). Based on interpersonal therapy for unipolar depression (Klerman, Weissman, Rounsaville, & Chevron, 1984) this treatment is strongly influenced by Goodwin and Jamison's instability model of bipolar disorder. This proposes that the basic (and largely genetic) vulnerability to symptomatic episodes is a susceptibility of the circadian system and an increased vulnerability to neurotransmitter system disruption (Goodwin & Jamison, 1990, chapter 19). IPSRT is also derived from the closely

related 'social zeitgeber' hypothesis (Ehlers, Frank, & Kupfer, 1988; Ehlers, Kupfer, Frank, & Monk, 1993), which asserts that disruptions of certain periodic signals in the environment are associated with affective disorder relapse. Ehlers, et al., believe that zeitgebers (i.e. dawn light or social prompts) which usually serve as time cues and entrain biological rhythms, if allowed to go awry, can disrupt circadian integrity and have a deleterious effect on susceptible individuals. IPSRT encourages bipolar patients to adopt regular routines for sleeping, waking, eating, exercising, and partaking of social interaction. Patients are encouraged to become vigilant with respect to maintaining this rhythmicity, and are helped to reduce the number and severity of interpersonally based stressors. It is thus hoped to enhance the regularity of circadian and social rhythms and avoid the over-stimulation believed to trigger relapse (Frank et al, 1997; 1999). As yet this approach has not been empirically validated with bipolar patients, although data from an ongoing randomised, controlled trial (RCT) appears to have important clinical implications, a point that will be returned to shortly.

### Similarities between CBT and IPSRT.

It is clear from the brief descriptions of CBT and IPSRT that despite differences in theoretical underpinnings, there exists a considerable overlap between the aims and techniques employed. Exponents of each nascent therapy might learn from one another to provide hope for a set of clinical problems previously thought of as



intractable. A good example of an “eclectic” psychosocial therapy which has harnessed a variety of therapeutic techniques, on the grounds of their “potency”, exists in the guise of dialectical behaviour therapy (Linchon, 1993) which has met with some success in the management of individuals with borderline personality disorder.

In the light of the present discussion, the IPSRT rationale of targeting interpersonal problems as both direct means (via autonomic arousal) and indirect means (via daily routines and mood) of maintaining circadian equilibrium could be adopted by CBT therapists to facilitate engagement where patients are reluctant to embrace a psychological model. Similarly, the central tenet of CBT work in the field of psychosis suggests that amelioration of symptoms is best achieved within the framework of an understanding of the individual’s enduring psychological vulnerability (Chadwick, Birchwood & Trower, 1996). Cognitive therapists working with a broad panoply of disorders increasingly assume that all patients are incessantly striving to construct a sense of self (Beck, 1996). Although IPSRT (in common with CBT) encourages remitted bipolar patients to explore their feelings about having the disorder and to grieve for the loss of their healthy self (Frank et al., 1997), its practitioners do not directly target issues of low self-worth.

Whilst a complete synthesis of psychosocial treatments is unlikely to be of particular benefit, it has recently been suggested

that radical change in treatment modality can have a particularly deleterious affect on remitted bipolar patients, as described below.

### Effect of Shift in Treatment Modality.

During the course of a preliminary evaluation of adjunctive interpersonal and social rhythm therapy (IPSRT), Frank et al. (1999) made a surprising discovery, albeit one that was largely consistent with the philosophy as to the benefits of stable routines.

Their randomised, controlled trial targeted individuals with a lifetime diagnosis of Bipolar I Disorder whom, at time of recruitment, were in at least their third discrete affective episode. Having first treated the symptomatic episode by way of appropriate medication, and (randomly assigned) IPSRT or intensive routine clinical treatment, the (by now) remitted participants (N = 82) were randomly reassigned to either IPSRT or clinical management, both in conjunction with pharmacotherapy, for ongoing preventative treatment. Analysis of data documented an overall 34% risk of symptom reoccurrence during the first 12 months of treatment with disappointingly similar relapse rates for the four treatment combinations.

However the authors also noticed that the portion of patients who were subjected to a change of treatment modality were fairing less well than those whose therapy remained constant through treatment and preventative phases of the project, both in terms of recurrence rates and higher levels of ongoing symptomatology. Recurrence hazard was not immediate (becoming apparent at about

25 weeks) but was highly related to the assignment of stable versus altered treatment, and occurred across conditions. Only 17.5% of participants from the stable treatment assignments (IPSRT or clinical management in both phases) experienced a recurrence of affective episode, compared to 40.5% assigned to the altered treatments. Survival curves were comparable for groups who 'lost' or 'gained' the structured psychotherapy (in altered assignments) and for groups who received either continuous psychotherapy or no psychotherapy at all (in stable assignments).

Since this was a rigorously designed study, therapist or medication changes did not account for these findings. Patients remained with their original therapist regardless of treatment reassignment. Although 30% of patients experienced an unplanned change of therapist or psychiatrist during the course of the year, therapist reassignment did not significantly affect recurrence hazard. Strict pharmacotherapy protocols for treatment and preventative phases of the study were applied and held constant across conditions and medication adherence was physically monitored throughout. Patient termination against medical advice did not contribute to recurrence risk. There was, however, an association between shorter survival times and patients who had to remain in the acute treatment phase for prolonged periods. Similarly, patients treated for a mixed episode (concurrent depression and mania) in the acute phase were significantly more at risk regardless of treatment modality. The higher relapse rates in



these groups were thought to be an artifact of their increased severity of psychopathology.

Frank and team proffer several explanations for these unexpected findings. Bipolar patients, they believe, are especially sensitive to environmental instability, and were thus unable to tolerate the disruption caused by a radical modification of their treatment. Nonetheless these results do not appear to bode well for the efficacy of IPSRT as a psychotherapeutic management of remitted bipolar patients. However, the authors maintain that although it was based on a routine psychiatric consultation, and envisaged as a control condition, the clinical management condition was in fact fairly intensive (comprising psychoeducative components as well as clinical status and symptom review), and was conducted by highly trained, warm, and empathic psychotherapists. As such, they argue that the clinical management condition could better be described as “non-specific”, or “low dose” psychotherapy. Frank et al. (1999) thus draw the conclusion that both ‘high’ and ‘low dose’ adjunctive psychotherapy are associated with relatively good outcomes. Further, since acceptance onto the study was dependent on successful symptom resolution within specified time constraints, participants assigned to different preventative modalities were effectively denied (what had been for them) a successful treatment. The failure of the new treatment was not immediately apparent as risk of recurrence increased with time. Although loss of IPSRT was not associated with significant

differences in survival rates, it did correspond with higher monthly symptom scores than those in the other three treatment combinations. Anecdotally, several participants dropped out of the research programme apparently through disappointment at not remaining in the IPSRT group.

Thus Frank and colleagues conclude, that transfer from an intensive structured psychotherapy to a nonspecific treatment may be particularly destabilising for bipolar patients. The wider implications of these findings are considerable. One might conclude that it is unwise to embark on a course of enriched psychotherapy with bipolar patients unless the treatment is viable over a prolonged period. Premature termination of a specific structured therapy could be deleterious and it may be better never to receive such therapy than to have it discontinued. However, further research, perhaps using a more conventional control group and involving other forms of psychotherapy are clearly warranted to test these assumptions empirically.

The remainder of this chapter considers the implications drawn from the present study for psychotherapy with bipolar patients.

### Implications of Present Data to Psychotherapy for Bipolar Patients.

The results of the present study might be taken to indicate various minor modifications to evolving forms of cognitive therapy for bipolar patients. The finding of similar implicit abnormalities in depression and mania suggest an enduring deficit of self-regard. Thus cognitive behavioural strategies to modify underlying negative self-schemas might be used to inoculate patients against both depression and mania. Attempts to enhance impoverished self-esteem, or to bolster unstable self-esteem, could usefully be applied to depressed and manic episodes. There are, however, several clear obstacles to psychotherapy provision during acute phases of mania.

### Obstacles to Psychotherapy with Manic Patients.

At present the pioneers of psychological treatments for bipolar patients generally focus their therapeutic endeavours either on episodes of depression or on the intervals between symptomatic attacks. Obstacles to structured psychotherapy during episodes of mania include the extreme disruption to therapeutic engagement caused by patients' distractibility, excitement, and hostility. Further, poor concentration and deficits in learning have been demonstrated in both manic and depressed bipolar states (Goodwin & Jamison, 1990, chapter 11). As Scott (1995, p.581) puts it, "reticence about trying to cure acute mania with a talking therapy is easily understood" and the difficulties of conducting structured therapy during an acutely manic phase of bipolar disorder are all too clear.



In an attempt to bypass these obstructions, therapy is often targeted at the interepisodic periods of relative calm, and strategies to cope with grandiose impulses (i.e. delaying tactics) are agreed and documented by the patient during periods of remission (Lam et al., 1999, chapter 8). Having transcended the more conspicuous difficulties of conducting psychotherapy with a floridly manic (or hypomanic) patient, therapy with remitted patients is not without its hazards also.

### Obstacles to Psychotherapy with Remitted Patients.

Other obstacles await the unwary therapist in the form of the remitted bipolar patient's poor insight into his or her own mental illness. An inexperienced therapist could be seduced by the recovering bipolar patient's sometimes over-optimistic subjective assessments of improvement. Apparently improved affect, in the guise of enhanced, more affective or spontaneous interpersonal relations, might reflect recovery but could also be the early warning signs of relapse (Scott, 1996a; Lam, et. al., 1999). In the light of the findings by Frank et al. (1999), a decision to relegate improved patients to routine medical management or otherwise terminate therapy prematurely could be particularly detrimental for remitted bipolar patients.

Variations in insight are associated with poor short-term outcome and medication compliance in this, as with other severe psychopathologies (e.g. McEvoy, Freter, Meritt, & Apperson, 1993; Ghaemi, 1997, Lam et al., 1999). It is hardly surprising that

someone who cannot recognise that they have a serious mental illness (whether for reasons of neurological deficit or motivation to preserve an optimistic view of one's self or world) is unlikely to believe that they need treatment.

### Inaccessibility of Negative Cognitions in Remitted Bipolar Patients.

Closely related to the above, the results of the present study anticipate another, less well-recognised impediment to therapy with remitted patients. This is the relative inaccessibility of enduring negative cognitions even during periods of remission. It is hard to imagine conducting a course of psychotherapy with remitted patients through some (as yet undiscovered) defence-penetrating medium. Indeed, in order to successfully participate in their own rehabilitation (i.e. to recognise and moderate dysfunctional cognitive material, or interpersonal relationships), remitted patients would be required to become more aware (or accepting) about themselves and their illness. Alternatively put, they would need to lower their manic defence.

Since one can speculate that the manic defence is there for good reason, or at least that it came about by way of powerful motivational forces, a direct onslaught is likely to encounter considerable resistance both from patients and their relatives. An alternative method of overcoming difficulties in accessing latent self-esteem distortions can be derived from the conclusions of the present study. Bipolar patients could be offered treatment specifically during episodes of depression. According to the manic-

defence hypothesis, at this juncture the attributional and self-representational abnormalities underlying bipolar affective disorder are most accessible both to the patient and the therapist. Given the ample evidence for successful psychotherapy targeted at episodes of unipolar depression (e.g. Gotlib & Hammen, 1992; Williams, 1992) this strategy might prevail. It is also consistent with the increasing acceptance by cognitive therapists that all patients (and perhaps all people) are embroiled in an ongoing struggle to attain a effectual sense of self (Beck, 1996).

Aside from the possible human benefits that might follow, the effectiveness of this approach would offer an exacting test of the manic-defence hypothesis. These arguments, together with those of previous commentators (APA, 1994b; Scott, 1995, 1996a, & 1996b; Lam et al., 1999; Frank et al., 1999) suggest that trials of CBT or other psychotherapies for bipolar patients should be pursued in the near future.

### Conclusion.

The present investigation provides compelling evidence in support of the notion of a motivational process in bipolar affective disorder. The radical shift in response pattern between opaque and transparent measures demonstrated by bipolar-manic participants supports the manic-defence hypothesis and the capacity of opaque measures to penetrate this defence.



Although this study hints at underlying processes, unfortunately it does not delineate the precise nature of the mechanisms at play. It does not explain how the defense is acquired and it fails to establish why some individuals are able to employ this mechanism and some are not. Most importantly it does not provide an adequate explanation as to why the manic defence is only intermittently activated, and even then is often insufficient to prevent breakthrough depressotypic symptoms. These limitations, aside, this study does demonstrate how cognitive methods can be used to investigate and account for 'psychodynamic' processes. It is however necessary to qualify these results as those of a preliminary study which require longitudinal replication using a larger subject population. The addition of a remitted bipolar control group and revised measures of attributional style and self-representation are also indicated. More extensive research will be necessary to describe fully the cognitive processes involved, and their interactions with other normative processes, such as intelligence and memory.

Appendices.

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Appendix 1. Participant Information – General  
Bipolar-Manic Group.

Pt	Location	Status	Diagnosis	Sex	Age	Marital status	Employment status	Previous Course of Illness
1	Hospt.	IP MHA2	BPD-Manic	F	59	Divorced	Unemployed	Rapid cycles
2	Hospt.	IP MHA3	BPD-Manic	F	63	Widowed	House person	1992 I diagnosed Hypomanic
3	Hospt.	IP MHA3	BPD-Manic	F	61	Single	Retired pt nurse	
4	Hospt.	IP MHA2	BPD-Manic	M	65	Single	Retired farmer	Rapid cyler
5	Hospt.	IP MHA2	BPD-Manic	M	21	Single	Student	2nd manic episode
6	Hospt.	IP	BPD-Manic	F	53	Single	Care-assist, Sick Leave (SL)	3 previous manic episodes.
7	Hospt.	IP MHA3	MD-Manic	F	66	Divorced	PT vol. Oxdam	History of Bipolar episodes
8	Day Hospt.	OP	BPD-Manic	M	31	Single	Brick-layer, SL	3 manic episodes in 7 yr. Recently discharged Previously detained MHA3, Recently discharged Previous. Manic episodes
9	CMHT	OP	BPD-Manic	F	40	Divorced	Unemployed	
10	Hospt.	IP	BPD-Manic	F	28	Single	PT vol / carer	
11	CMHT	OP	BPD-Manic	F	35	Divorced	Unemployed	3 previous Manic-depressive episodes (MDE)
12	Hospt.	IP	BPD-Manic	F	74	Married	House-person	
13	Hospt.	IP MHA2	MD-Manic	M	52	Divorced	Retired seaman	Bipolar history
14	Hospt.	IP MHA5/2	Manic	F	33	Cohabits	Unemp. Previously a nurse	Previous MDE
15	Hospt.	IP	Manic	M	22	Single	Unemployed	Previous depression & manic episodes



Depressed Group.

Pt	Location	Status	Diagnosis	sex	Age	Marital	Employment status	Previous Course of Illness
1	Support- ed House	OP	BPD-Dep.	F	40	Single	Unemp. previously a nurse	Rapid cycles
2	Hospt.	IP	BPD-Dep.	F	54	Divorced	pt vol. Work	BP history
3	Home	OP	BPD-Dep.	F	55	Married	Retired teacher	History of manic episodes.
4	Day Hospt	OP	MD- Psychosis depressed	M	49	Married	Previously a factory worker	Rapid cycler
5	Home	OP	BPD-Dep.	M	48	Divorced	Taxi driver, sick leave	BP history
6	Day Hospt	OP	BPD-Dep.	F	42	Divorced	Matron, sick leave	BP history
7	Home	OP	BPD-Dep.	M	72	Single	Retired navy	Previous episodes
8	Day Hospt	OP	BPD-Dep.	M	54	Single	Retired teacher	BP history
9	Hospt.	IP	BPD-Dep.	F	44	Married	House person	BP history
10	CMHT	OP	Manic depressive	M	48	Married	Teacher, sick leave	long cycle lengths
11	Hospt.	IP MHA3	Manic depressive psychosis	F	38	Divorced	Unemployed	rapid cycles
12	Hospt.	IP	BPD-Dep.	M	49	Married	Retired decorator	prev. manic episodes.
13	Hospt.	IP	BPD-Dep.	F	31	Single	Unemp. previously a nanny	prev. manic episodes.
14	Home	OP	BPD-Dep.	F	20	Single	Unemployed	rapid cycles
15	Hospt.	IP MHA2	BPD-Dep.	F	23	Cohabits	Unemployed	2 manic episodes in past

Healthy Control Group.

Pt	Location	Status	Diagnosis	sex	Age	Marital	Employment status	Previous Course of Illness
1	Work	N/A	N/A	F	51	Single	Health visitor	N/A
2	Work	N/A	N/A	F	53	Single	Health visitor	N/A
3	Home	N/A	N/A	F	62	Married	House person	N/A
4	Home	N/A	N/A	M	19	Single	Unemployed	N/A
5	Work	N/A	N/A	F	53	Single	Student counselor	N/A
6	UCNW	N/A	N/A	M	47	Cohabits	Student	N/A
7	Home	N/A	N/A	F	48	Sep.	Unemployed	N/A
8	Home	N/A	N/A	F	68	married	Retired bar person	N/A
9	Home	N/A	N/A	M	32	cohabits	Builder	N/A
10	Home	N/A	N/A	F	74	married	House person	N/A
11	Work	N/A	N/A	F	32	married	Medical secretary	N/A
12	Work	N/A	N/A	F	44	cohabits	Secretary	N/A
13	Work	N/A	N/A	F	34	married	Teacher	N/A
14	Home	N/A	N/A	M	35	single	Unemployed	N/A
15	Home	N/A	N/A	M	45	divorced	Labourer	N/A

## Appendix 2. Participant Information – Medications.

### Bipolar-Manic Group.

<b>Pl</b>	<b>Medication (not clear whether past or current)</b>	<b>Notes</b>
1	Atenol Carbamazepam Zimovane	
2	Lithium Thyroxin Imipramine Ventolin	
3	Prothiadine Stelazine Procycladine Droperidol	
4	Lithium Thyroxin (Insulin)	Diabetic
5	Trifluoperazine (procycladine)	
6	Lithium	
7	Haloperidol Diazepam	
8	Lithium Trifluoperazine Procycladine Temazepam	
9	Carbamazepine Thioridazine? Procycladine??	Illegible notes
10	Lithium Halperidol Nitrazepam	
11	Lithium	
12	Lithium Zimovane	
13	Tremazepam Lithium Stelazine Zimovane	Poor compliance
14	Lithium Procyclidine Droperidol	
15	Lithium	

### Bipolar-Depressed Group.

<b>Pl</b>	<b>Medication</b>	<b>Notes</b>
1	Lithium (Carbamazapine in past ?)	Poor compliance
2	Lithium	
3	Lithium Depixol Petofryn?	Illegible notes
4	Lithium amitriptyline stelazine Zimovane	
5	Lithium (an SSRI?)	
6	Lithium Prozac Tegretol	
7	Lithium Droperidol	
8	Desipramine Lithium	
9	Prozac Evening Primrose	Refuses other Med.s
10	Gamanil Lithium Prothiaden Zimovane	
11	Lithium Asendis?	
12	Sertraline Zimovane Lithium	
13	Carbamazapine	
14	Lithium	
15	Lithium Zimovane	



### Appendix 3. Participant Recruitment Flyer.

HAFOD LAS  
LLAWR Y DREF  
LLANGEFNI  
LL77 7TS  
TEL: 0248 750191/750292  
FAX: 0248 750896

#### REQUEST FOR BIPOLAR DISORDER RESEARCH PARTICIPANTS

As you may be aware I am presently undertaking research to examine possible cognitive abnormalities in individuals with a Bipolar Affective Disorder (manic depression).

I am looking for men or women who are either currently manic (~~DSM-IV~~ Bipolar Manic) or currently depressed (~~DSM-IV~~ Bipolar Depressed). Those who consent to testing will be given a variety of psychological instruments designed to measure information processing and attributional style. The test battery (including a full debriefing) will take 60-90 minutes and previous participants report they have found it interesting and enjoyable! I can arrange to see people at Hergest or in the community. Unfortunately there is no payment or other immediate benefit, although hopefully this work will contribute towards the development of psychological treatments for Bipolar Affective Disorder.

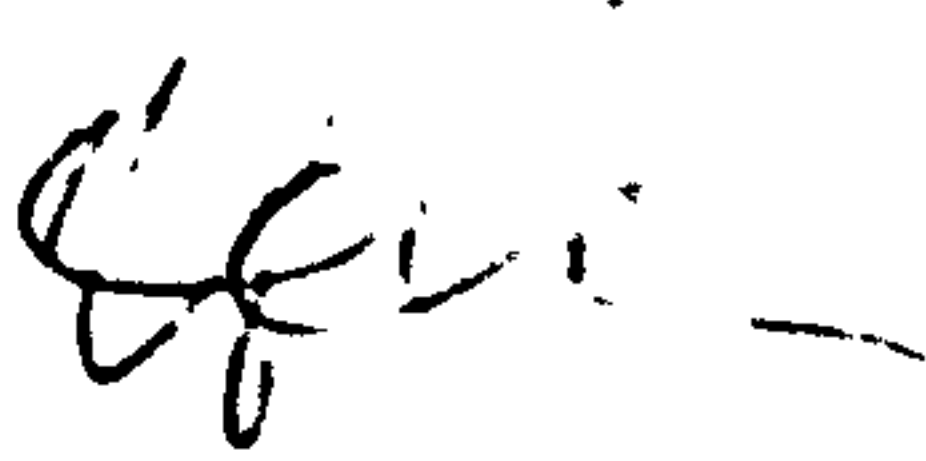
This research has been approved by the Ethics Committees of Gwynedd and Clwyd Health Authorities and U.C.N.W. (Psychology Department).

If you would like to assist with this project, please contact me with the name and locations of any potential participants over the next 18 months, on the above number or via Sheila Thomas, Psychology Department, Hergest Unit (Tel: 184296).

I will be happy to discuss this research in more detail.

With many thanks in anticipation of your help.

HELEN LYON C. PSYCHOL.  
Principal Clinical Psychologist  
Hafod Las C.M.H.T. Ynys Mon.

  
Sent to: Hospital Consultants  
Clinical Assistants  
Junior Doctors  
C.M.H.T. in Gwynedd  
Hospital Wards

*Happy to help*  
*T.H. Smith*  
-8 SEP 1994

## **Appendix 4. Modified Diagnostic Interview Schedule (DIS).**

### **DIANOSTIC INTERVIEW SCHEDULE**

**Amended for use with Cognitive Performance Measures**

**JULY 1990**

**Modified Diagnostic Interview Schedule Continued.**

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## Appendix 5. The National Adult Reading Test (NART).

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## **Appendix 6. The Beck Depression Inventory (BDI).**

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## **Appendix 7. The Rosenberg Self-Esteem Questionnaire (RSEQ).**

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**Appendix 8. The Attributional Style Questionnaire (ASQp).**

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**Appendix 9. The Pragmatic Inference Task (PIT).**

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**Appendix 10. The Emotional Stroop Test (EST).**

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## **Appendix 11. The Self-Referent Incidental Recall Task (SRIRT).**

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## Appendix 12. Participant Consent Form in English.

### CONSENT FORM

You are being asked to take part in a study which may help us understand the causes of bipolar affective disorder (manic depression). The study involves you taking a number of tests designed to tell us how you concentrate and think about yourself and things in the world. You will receive no direct benefits from participating in the study, although we hope that you will find the tests interesting. I would like you to spend approximately 90 minutes to participate in 6 tests. You do not have to take part and you will be free to withdraw at any time for any reason.

If you do decide to take part any information you give us will be treated in the strictest confidence. You will not be identified in any publications which may result from the research. You will be given the opportunity to discuss any aspect of your experience of being a participant.

Please sign this form if you understand what is being asked of you and if you agree to take part.

I agree to take part in this study and have had adequate time to read this form.

Signed:..... Date:.....

## Appendix 13. Participant Consent Form in Welsh.

### FFURFLEN CANIATAD

Gofynnir i chi gymeryd rhan mewn astudiaeth a fydd efallai yn ein helpu i ddeall achosion ahwylder affeithiol deubegwn (manic-depression). Fel rhan o'r astudiaeth byddwch yn cael profion a luniwyd er mwyn rhoi gwybodaeth inni sut rydych chi'n canolbwyntio a sut rydych chi'n meddwl amdanoch eich hun ac am bethau yn y byd. Ni chewch unrhyw fantais uniongyrchol o gymeryd rhan yn yr astudiaeth, er ein gobaith yw y byddwch yn gweld y profion yn ddiddorol. Bydd y 6 prawf unigol yn cymeryd tua 90 munud i'w cyflawni. Nid oes rhaid i chi gymeryd rhan a byddwch yn rhydd i dynnu'n ol ar unrhyw adeg am unrhyw reswm.

Os penderfynwch i gymeryd rhan cedwir unrhyw wybodaeth a roddir gennych inni yn hollol gyfrinachol. Ni enwir chi mewn unrhyw gyhoeddiadau sy'n debygol o ddeillio o'r ymchwil hwn. Bydd cyfle i drafod unrhyw agwedd o'ch profiad o fod yn rhan o'r gwaith ymchwil.

A fydddech chi gystal ag arwyddo'r ffurflen hon os ydych yn deall yr hyn a ofynnir i chi ei wneud ac os ydych yn cytuno i gymeryd rhan?

Cytunaf i gymeryd rhan yn yr astudiaeth yma, ac 'rwyf wedi cael digon o amser i ddarllen y ffurflen yma.

Arwyddwyd:..... Dyddiad:.....



Appendix 14. Selected ANOVA Tables.  
Presented in order of appearance in the text

ASQ(pf) Internality data.  
2-way ANOVA (groups x valence of events)

Effect	df	SS	MS	F	p	Epsilon
g		163.822	81.911	3.844	.0293	
Error		895.000	21.310			
v		618.844	618.844	24.047	.0000	
gv		1947.289	973.644	37.834	.0000	
Error		1080.867	25.735			1.00

ASQ(pf) Stability data.  
2-way ANOVA (groups x valence of events)

Effect	Df	SS	MS	F	P	Epsilon
g	2	2.996	1.470	.059	.9431	
Error	42	1056.867	25.211			
v	1	864.900	864.900	29.544	.0000	
gv	2	1336.067	668.033	22.820	.0000	
Error	42	42	1229.533	29.275		1.00

ASQ(pf) Globality data.  
2-way ANOVA (groups x valence of events)

Effect	Df	SS	MS	F	p	Epsilon
g	2	43.489	21.744	.533	.5908	
Error	42	1713.800	40.805			
v	1	786.178	786.178	32.921	.0000	
gv	2	1198.822	599.411	25.100	.0000	
Error	42	1003.000	23.881			1.00

**PIT Internality data.**  
**2-way ANOVA (groups x valence of events)**

Effect	Df	SS	MS	F	p	Epsilon
g	2	2.156	1.078	.852	.4338	
Error	42	53.133	1.265			
v	1	16.044	16.044	8.953	.0046	
gv	2	102.689	51.344	28.651	.0000	
Error	42	75.267	1.792			1.00

**Stroop Reaction Times.**  
**3-way ANOVA (groups x type of card x order of presentation).**

Effect	df	SS	MS	F	p	Epsilon
Group	2	37276.541	18638.270	7.264	.0020	
Error	42	107767.956	2565.904			
Type	2	4190.785	2095.393	35.995	.0000	
G x t	4	174.904	436.976	7.506	.0000	
Error	84	4889.978	58.214			.94
Order	1	118.670	118.670	1.758	.1920	
G x o	2	174.674	87.337	1.294	.2848	
Error	42	2834.489	67.488			1.00
T x o	2	13.541	6.770	.253	.7768	
G x t x o	4	69.948	17.487	.654	.6255	
Error	84	2245.178	26.728			.96

**Stroop Interference Scores.**  
**3-way ANOVA (groups x order x type of card)**

Effect	<i>df</i>	SS	MS	<i>F</i>	<i>p</i>	Epsilon
Group	2	2093.811	1046.906	9.239	.0005	
Error	42	4759.433	113.320			
Order	1	3.200	3.200	.036	.8495	
G x o	2	84.100	42.050	.479	.6227	
Error	42	3686.700	87.779			1.00
Type	1	1947.022	1947.022	45.690	.0000	
G x t	2	1066.211	533.106	12.510	.0001	
Error	42	1789.767	42.613			1.00
O x t	1	.022	.022	.001	.9768	
G x t x o	2	43.744	21.872	.844	.4371	
Error	42	1088.233	25.910			1.00

**SRIRT Endorsed Items.**  
**2-way ANOVA (groups x valence of adjectives)**

Effect	<i>Df</i>	SS	MS	<i>F</i>	<i>p</i>	Epsilon
g	2	15.089	7.544	1.314	.2794	
Error	42	241.067	5.740			
v	1	260.100	260.100	55.584	.0000	
gv	2	958.867	479.433	102.457	.0000	
Error	42	196.533	4.679			1.00

**SRIRT Recall Data.**  
**2-way ANOVA (groups x valence of adjectives)**

Effect	<i>Df</i>	SS	MS	<i>F</i>	<i>p</i>	Epsilon
g	2	4.022	2.011	.508	.6053	
Error	42	166.267	3.959			
v	1	20.544	20.544	7.354	.0097	
gv	2	201.622	100.811	36.086	.0000	
Error	42	117.333	2.794			1.00



**Appendix 15. Short Form Of Present Thesis As Published In  
Journal Of Abnormal Psychology, 1999.**

# Social Cognition and the Manic Defense: Attributions, Selective Attention, and Self-Schema in Bipolar Affective Disorder

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Manic patients, depressed bipolar patients, and normal controls were compared on measures of social cognition. Manic patients showed a normal self-serving bias on the Attributional Style Questionnaire, but depressed patients attributed negative events more than positive events to self. On an implicit test of attributional style, both patient groups attributed negative events more than positive events to self. Both patient groups showed slowed color naming for depression-related but not euphoria-related words. Manic patients, like normal controls, endorsed mainly positive words as true of self but, like the depressed patients, recalled mainly negative words. Findings from the implicit tests indicate a common form of psychological organization in manic and depressed patients, whereas the contrasts between the scores on the implicit and explicit measures are consistent with the hypothesis of a manic defense.

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