Affect on the Prestige Landscape:

The Prestige Model of Spectrum Bipolarity

James M. Le Bas

Under the supervision of Prof. David Castle and A/Prof. Richard Newton

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Abstract

**Background:** Bipolar spectrum conditions arose during the Pleistocene epoch, where social inclusion was maintained through prestige – the investment of the group in the individual. This thesis seeks to investigate the interplay between bipolarity and prestige.

**Methods:** A case control study categorised 228 adult participants into a seven node bipolar spectrum. (1) Structural differences in prestige approach motivation (MSPaM) between the nodes were assayed. (2) A dynamic model of bipolar pathogenesis was tested by means of correlation and path analysis. (3) Binary logistic regression was utilised to examine the relationships between MSPaM, bipolar family history, perceived childhood relational trauma (PCRT) and bipolar disorder. (4) Contour plots of affective change on the plot of MSPaM versus prestige were drawn. (5) An exploratory analysis was undertaken.

**Results:** (1) After controlling for mood variables, bipolar I (S1), bipolar II (S2) and (bipolar family history-positive) pseudounipolar (S3) groups were found to have higher mean MSPaM scores than controls (S7). (2) There was good evidence to support the modelling for the dynamic model stem and depressive (withdrawal) branch, while support for the approach arm was ambiguous; the relationship for tension and prestige only being evident in moderate mood elevation. In hypomania MSPaM correlated strongly with prestige ($r_p = 0.51$). While the bipolar depressed cohort demonstrated a raised MSPaM, the unipolar depressed cohort did not. The pseudounipolar (S3) node had both elevated MSPaM and prestige when compared with the unipolar (S6) node. (3) MSPaM predicted BD (OR 6.8), but only in the absence of bipolar family history, and the converse trend was evident. (4) The contour maps showed three affective zones – depressive, elevated and euthymic.

**Conclusions:** Developmental, genetic, and social-prestige variables relate dynamically to the bipolar spectrum, where those with bipolar disorder have a greater drive to social inclusion. Mood disorders may have arisen in evolutionary time through prestige competition in the context of relative social marginality; bipolar depression being a means of ostracism avoidance and hypomania serving to raise social value (prestige) through mood elevation and prestige approach motivation. The contour plots provide us with a landscape of bipolar mood states, promoting the view that they may be evolutionarily stable strategies in the ancestral competition for prestige. There was a complex interaction of MSPaM and bipolar family history with respect to bipolar causation, suggestive of at least two pathways to bipolar disorder. Childhood relational trauma (PCRT) appeared to be a further aetiological factor. The prestige model offers a new conceptualisation of affective disorders and has received preliminary support.
Keywords

Prestige, social investment, bipolar spectrum, MSPaM, self-esteem, hypomania, mania, depression, evolution
Declaration

This is to certify that:

i. the thesis comprises only my original work towards the PhD except where indicated in the Preface,

ii. due acknowledgement has been made in the text to all other material used,

iii. the thesis is fewer than 100,000 words in length, exclusive of tables, maps, bibliographies and appendices.

..........................................................

James Le Bas

Date: 27.2.15
Preface

This thesis represents the sole work of the author under the supervision of Professor David Castle and A/Professor Richard Newton. Short drafts written by the author for this thesis were extracted and revised by the author with Professor David Castle, A/Professor Richard Newton, Ms Rachel Sore and Dr Denis O’Loughlin and published in *Prestige and Bipolarity* and *The Prestige Model of Spectrum Bipolarity*. The author’s contribution to these papers was of the order of seventy five per cent. The source thesis drafts were then modified to the extent of rectifying statistical errors, as advised by Ms Rachel Sore.

The sections The Evolution of Emotion (Affective Pathogenesis) and Play (Affect) were written solely by the author in the year before candidature and have not been used or published elsewhere.

Acknowledgements

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Academic Presentations

- Paper: Prestige and Bipolarity: An Evolutionary Perspective.  

- Paper: Prestige and Bipolarity: An Evolutionary Perspective.  

- Paper: Prestige and Bipolarity *Australasian Society for Bipolar & Depressive Disorders* 3-5 October 2013 Melbourne Australia. With D. Castle, R. Newton, R. Sore, D. O’Loughlin.


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Introduction

“At the core of evolutionary medicine is recognition that diseases need both proximate explanations of bodily mechanisms and evolutionary explanations of why natural selection has left the body vulnerable to disease.” (Nesse & Stearns, 2008, p. 30)
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Problem

Bipolar spectrum disorders are highly prevalent and disabling conditions, being amongst the top ten causes of non-fatal disease burden in the world (Ayuso-Mateos, 2000). Though we do not understand their causal mechanisms, they are vexing to doctors as they cause significant suffering and sometimes death. This situation may stem in part from a lack of knowledge about general processes of affectivity, alongside a particular ignorance of the proximate bipolar substrate. Despite empirical data on the relationship between such things as life events, cognition, social support, self-esteem, and mood states, integrative models are lacking. Furthermore, we have little idea of how these disorders arose during evolution. This thesis aims to derive and test an exploratory model of bipolar spectrum pathogenesis based on the notion of social investment (prestige).

Aim

Therefore, a literature review on extant psychosocial factors involved in bipolar disorder will be undertaken and a dynamic model of bipolar spectrum pathogenesis – centered on the notions of self and group investment – will be developed. A schema of affectivity based on both Panksepp (1998) and Mithen (2005) – Hedonic Energetic Social Engagement (HESE) – will support this work. This will lead to a bipolar spectrum model based on clinical and genetic parameters, illustrating a gradient of bipolarity. This model will be evaluated for its structural and dynamic properties through hypothesis testing and exploratory analysis.

Scope

The scope of the Measurement of Prestige Factors (MOPF) study is to research affective and prestige factors relevant to a notional bipolar spectrum. Prestige is defined as the investment of the group in the individual and the project draws heavily on this construct. The data for this research were collected from a case control survey given to two populations – a tertiary mental health service (over twelve months) and a general practice clinic (over consecutive days). Both were located on the Mornington Peninsula Melbourne, Australia. Two hundred and twenty eight surveys were analysed; the primary analysis centred on the relative score for prestige
approach motivation (MSPaM) between spectrum nodes and the correlation of variables in the Dynamic analysis. The spectrum groups were created by combining self-nominated clinical diagnoses of bipolar disorder and depression (along with “no diagnosis”) with bipolar family history, history of hospitalisation or psychosis and ratings by means of the Mood Disorder Questionnaire and two mood scales. Exploratory analysis was wide-ranging and took form as the project proceeded and ideas arising iterated with clinical experience. This research was undertaken in the context of ongoing public and private clinical work dealing with a range of individuals with bipolar spectrum conditions. The study aims to achieve a proof of concept synthesis of the relationship between prestige and bipolarity.

In order to commence this work, extant information was investigated through the interrogation of the following databases both collectively and singularly; Pubmed, Psychinfo, Psychiatry Online, EBSCO, Web of Science, Scopus, Google Scholar, Proquest, Discovery and Trove. Search terms included "bipolar spectrum" and "review", "self-esteem" and "bipolar", "life events" and "bipolar", "genetic" and "bipolar disorder", "temperament" and "bipolar disorder", "maladaptive schema" "bipolar disorder" and "review", "behaviour" and "bipolar", "social support" and "bipolar", "prestige", "evolution" (in various guises) along with an number of other terms. Because of the large scope of the investigation, some searches were ad hoc and constrained to limited databases and to review articles.

The survey is largely delimited to genetic and psychosocial factors pertinent to the bipolar spectrum, though further biological aspects are incorporated. There is already a large amount of biological research into bipolar disorder with only slow progress toward finding a proximate substrate. I have endeavored to maintain an evolutionary focus due to a desire to delineate ultimate causal factors. Because there is a paucity of instruments available to the investigation of prestige, new and untested scales were developed. Similarly, there was a lack of explanatory modelling in relation to subjects such as arousal, and for this reason new models were tentatively developed, albeit being based on extant empiricism and theory. The case control method was chosen because of its simplicity and applicability to a proof of concept method. The survey was undertaken in a clinical context because of the direct and
current applicability to “real” mood syndromes, rather than university student samples, for example. Self-nomination of diagnosis was used at the first level of the diagnostic algorithm rather than using a structured diagnostic instrument, because this method was complementary to clinical interview and not disruptive to the patient. In addition, the study was entirely self-funded. The sample chosen was largely angloceltic by reason of the region and aged 18-64, though is likely to be representative of the general Australian adult population, albeit one with affective and care-seeking enrichment.

The study is based on the correlational method and therefore causal attributions are speculative. The newly conceived survey instruments MSPaM, MSIS, SIPS and TENSION scale were created to mensurate the population for factors relevant to the task. While they were subject to measures of internal reliability and, in two instances convergent validity, they have not been subject to test-retest procedures. The Likert method was utilised extensively and parametric statistics applied to these summed results. However, both the SISE and TENSION scales were nominally ordinal, but were found suitable for parametric analysis.

Being a survey, there is no validation by observer-rated instruments. Because certain groups of those with a family history of bipolar disorder have a relatively low representation in the population, two nodes of the bipolar spectrum (the pseudounipolar (S3) and pure family history (S5) had small numbers, limiting statistical inference. Because of the self-nomination method, there is some ambiguity and approximation in diagnostic allocation – as would befit a spectrum – though the empirical differentiation of bipolar I individuals from controls is substantiated, for example through triangulation. Similarly, it was not initially possible to separate cases with categorical mood elevation into hypomanic and manic groups and for this reason the category (hypo)manic was used. Later inferences which drew on tension level, prestige and depressive symptoms enabled approximate differentiation.

Though two a priori hypotheses drove the research (the structural and dynamic models), many of the findings are constrained by their reliance on an
exploratory analytic method. The study makes no findings about related conditions such as schizophrenia and schizoaffective disorder.

As a psychiatrist, I have biases in my work and the way I conceptualise conditions. My career in psychiatry began with a strong biological emphasis, but with time the psychological, and later social, dimensions became more relevant to me. This includes a preference for the “meaning” of symptoms as they arise in the psychological and social dimensions. Biological factors, I believe, are a mechanism for illness, but not the sole determinants. As such, some illnesses will be genetic accidents, while others may have distal causation and I look to evolutionary theory to fill these gaps. My professional orientation may have therefore led me to overlook issues. Similarly, I was a novice to the world of research at the formative stages of the study and had to quickly learn aspects of the field.

**Overview**

This work is a 9 chapter project which is interspersed with three patient narratives aiming to exemplify aspects of the developing theory. The first five chapters seek to set a frame for the research design and results. Chapter 1 considers the context of the predispositions to bipolar disorder with an emphasis on self-esteem. The biopsychosocial backdrop for the development of bipolar conditions is considered in Chapter 2, while Chapter 3 examines the interwoven evolution of affect, self-esteem and prestige. Social marginality is depicted as the crucible for the bipolar responses to real and perceived prestige.

In Chapter 4 the concept of the strategic motivational affect system (SMAS) is introduced and exemplified in *Hedonic Energetic Social Engagement (HESE)*, which draws on the models of Panksepp (1998) and Mithen (2005). The evolutionary origins of social engagement are traced and linked to dimensions of normal and pathological affective states, based in prestige selection. In Chapter 5 the historical origins of the bipolar spectrum are followed and found to be based on Platonic notions of *collection* and *division*.
The Prestige Model of Spectrum Bipolarity is described in Chapter 6 and two a priori hypotheses are mooted – the structural and the dynamic models. Thence the results are presented in Chapter 7, followed by a discussion, which finds that the results are in general support of the models. A wide ranging exploratory analysis is undertaken. The work concludes with the view that there is proof of concept evidence for the importance for prestige in the distal and proximate development of bipolar spectrum disorders.
Narrative - Naomi

Naomi\(^1\) is a 35 year old woman, separated from the father of her devoted young boys. She works hard as a gym instructor, cleaner and shop assistant in order to keep afloat. She came to consultation from her general practitioner with mood lability; a feeling that there were "two of her". One of these "selves" was Naomi, and the other was tagged "Nellie" by her friends. These were not alter egos, but states of mind with continuity. Naomi was depressed and constantly sad and saw herself as "crazy" – on the brink of being taken away. “Nellie”, on the other hand hyped-up markedly in the company of people and nothing seemed an effort. On autopilot, she pushed the balance between success and failure, being happier and energetic, helpful and magnanimous. Her speech was loud and fast and she was oriented for the good, whilst her energy had her "self-medicating" on people. These states were an involuntary response to social settings and there was such a period that lasted eight months after she guiltily left her partner; at the time she had lots of social playdates with the children and was involved in personal training. Her intent was to survive and not to give in. She was capable of great things, such as cutting her hair off and raising $6000 for a bike marathon.

There was no history of mania or psychosis. Naomi was not taking any medication, though she had been on the antidepressant sertraline for a number of years from the age of 19. She drinks minimal alcohol and takes no illicit drugs. She has not always followed through with medical advice. She has comorbid OCD and her half brother is said to have bipolar disorder, which she sees as a "copout". Her parents separated when her mother was pregnant with her and she has one a full brother and three half siblings.

Developmentally, Naomi’s early life was reasonably settled. There had been no obstetric complications and her milestones were good. She has good memories of being well-mothered and her stepfather was described as “wonderful”, though he left the family when she was ten. She had felt very affected for one year. She was a clever

\(^1\) All clinical material has been de-identified in this work.
girl who did not use her potential. She had a number of stable relationships from 19 and entered a union with the father of her children at around 25.

From seventeen she described “spiraling”, with exuberant energy and feelings of being almost indestructible. At 22 she had a profound depression without clear trigger and she was treated for a short time in the public sector. Since that time her moods have continued to sweep up and down with little remission. At times she speaks quickly with an up-tempo rhythm, whilst sometimes her mood is a mixture of depression and elevation. Her depressions are quite prototypical with dull and deadened feelings and self questioning – ”it feels like a physical thing”. Her thoughts may be retarded or accelerated, depending on mood. Naomi has been troubled with suicidal ideation over a long period. At one point she had ideas of driving into a wall and avoided using the car for two weeks to avoid this. Recently she laid her head on a train track as an “experiment” without intent to die. She is restrained from death by the existence of her children.

Mental State examination found a woman appropriately dressed without make-up and somewhat unkempt hair. She engaged well and communicated significant distress. Apart from sadness there was a degree of apprehension, whilst there was no sign of enjoyment or amusement. Her speech was within normal limits and there was no formal thought disorder. Naomi’s thoughts centred on her distress and self blame. She was concerned she would be seen as a "loon". Naomi appeared quite insightful. Subsequently at interview she displayed significant variation in her mental state; from melancholia to accelerated agitated presentations which she described as "antsy" and then back to sombre psychomotor retarded states. After an elevated discussion of a small crisis in which she had briefly left her children at home, she described feelings of failure and unworthiness. This led to a notable slowing of psychomotor activity and then a feeling like she wanted to cry. Sometimes she would swing from this presentation to an intensely labile and distressed state.

Naomi has always managed her situation with dignity and determination, concerned principally with the well-being of her boys. Naomi has bipolar disorder type II.
Development is a somatic branch, cutting through ontogenetic time and intersecting the domains of society, culture and ecology. Within this bough genes thread, turning proteins on and off in precision. Genes, joined by social filaments, intertwine and the environment sends shoots – feeding and containing growth in epigenesis. Memories form as synaptic buds – linked through time by meaning. Narrative serves as scaffold; bond between self and self; self and society. Time pushes on, molecules intersect and the domain of mind is instantiated. Change perspective; slice the threads across momentarily and view – face on – the multidetermined, time-locked, phenotype.
Chapter 1: Disposition to the Bipolar Illnesses

Introduction

Bipolar spectrum disorders occur within the substrates of biology, personality and society. Though only partly understood, these dimensions constitute both the germ and the ecology of dimensional bipolarity, of which bipolar disorder forms but a part. Bipolarity, it will be argued, is a response to the challenge of social inclusion in a competitive ancestral environment. This chapter seeks to summarise what is known about the temperamental dispositions to the bipolar spectrum conditions – dispositions which are subject to life events and the milieu of the family. Do people with these conditions have particular cognitive patterns and does their self-referential thinking pertain to the social environment? The discourse around creativity and Theory of Mind (ToM) will lead to an elucidation of the dissonance between the “inner” and the “outer” in bipolar spectrum conditions. Bipolar genes may be spread in the wider population, meting both advantage and disadvantage as multiple thresholds are passed (Kelsoe, 2003). Do they subserve any ecological mandate? Life events act on temperamental vulnerabilities to trigger pathologies and the cognitive patterns which subserve this relationship will be explored. Pathologies may be perpetuated by second order processes such as sensitisation and kindling. It will be argued that self-esteem cannot be explored without reference to societal prestige.

Temperament

Temperament is the manner of thinking, feeling and behaving characteristic to the individual. The word connotes composition toward disposition, yet consideration of temperament raises many epistemological questions, too diverse to consider here. Our temperament is clearly not a tabula rasa, on which development and the environment may write.

Temperament is a substrate dictated by genes, occupying an ecology which is impacted upon by developmental events. Organisms approach objects or withdraw from them, doing so with varying degrees of energisation, modulated by circadian and other biological rhythms. Persons follow a trajectory, both deterministic and
intentional and affective regulation plays a large part in how persons respond to their environment. Memory shapes knowledge, leading to goal-directedness and increasing efficacy within the environment; it may be difficult or relatively easy, while the person who fails at this task may internalise or somatise, rather than locating their self within the world. Temperament is not an issue for the solitary organism; as Cooley (1922) has said "the imaginations which people have of one another other are the solid facts of society" (p. 87). Temperament is the launching pad for social inclusion and a structure through which narrative ideation may shift. It denotes both commonality and uniqueness. Temperament accrues dispositions, which over time form character. Needless to say, temperament is a constellation, placing the individual in a singular position to prosper or become ill.

Thomas, Chess, and Birch (1968) and Chess (1997) described nine temperamental traits in a longitudinal study of newborn children. Four of these traits (activity level, tendency to approach or withdraw, response intensity, and mood quality) are conceptually related to affective dimensions. Similarly, the differentiation between "easy" and "difficult" temperamental types parallels research which describes the relationship between neuroticism and affective disturbance.

Cloninger, Svrakic, and Przybeck (1993) named novelty seeking, harm avoidance, reward dependence and persistence as four independently heritable temperamental dimensions. The first two of these may be conceived as extensions of Thomas and Chess's approach and withdrawal dispositions. Reward dependence and harm avoidance (Engström, Brändström, Sigvardsson, Cloninger, & Nylander, 2004; Huynh, Guile, Breton, Desrosiers, & Cohen, 2010; Loftus, Garno, Jaeger, & Malhotra, 2008) have been consistently found to be traits associated with bipolar disorder. Cloninger et al. (1993) cite four dimensions of personality which involve automatic, genetic and pre-conceptual responses to perceptual stimuli – the model is notable for its correlation with clinical conditions.

As currently evidenced, there is considerable overlap between the various personality models, ranging from Approach Motivation to Behavioural Activation to Approach and Avoidance and Extroversion/Introversion, to name but a few (Acton,
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An association has been found between bipolar disorder and novelty seeking and also lower self directedness (Huynh et al., 2010; Loftus et al., 2008). Harm avoidance was correlated with residual depressive symptoms (Loftus et al., 2008) and bipolar I patients possess personality traits significantly different from control individuals (Loftus et al., 2008). It remains difficult, however, to separate trait from state differences in the studies quoted.

Papolos, Mattisb, Golshanc, and Molaya (2009) have proposed that a Fear of Harm phenotype is reliably associated with paediatric bipolar disorder and an early onset course, with a severe form of the condition. In a correlational study of euthymic bipolar persons versus controls, Engström et al. (2004) found a modest association between harm avoidance, as well as reduced reward dependence, in bipolar disorders. The relationship of bipolar disorder to reward dependence was subsequently not supported by Tunca et al. (2004).

An endophenotype is an intermediate trait between the genotype and the illness phenotype. Gottesman and Gould (2003) nominated the following criteria for endophenotypy:

1. The endophenotype must be associated with the illness in the relevant population.
2. The endophenotype must be largely state independent, manifesting in the individual during both periods of health and illness.
3. The endophenotype must be heritable.
4. Within families, the endophenotype and illness should co-segregate.
5. The endophenotype found in affected individuals should be found in non-affected family members at a higher rate in the general population.

Savitz and Ramesar (2006) formed the opinion that there was good evidence that the cyclothymic and depressive temperaments were associated with bipolar I disorder. With respect to factor analytic techniques, bipolar disorder may associate with negative affectivity (neuroticism/harm avoidance), while more evidence was required to link bipolar disorder to Cloninger's other temperaments such as novelty seeking. Thus Gottesman and Gould's first criterion is met.
Studies of euthymic patients found that both negative affectivity traits and extroversion were elevated in bipolar disorder. The authors reached the conclusion that the aforementioned bipolar-related temperaments were state independent as Gottesman and Gould had required (Savitz & Ramesar, 2006). Personality traits, as tested by psychometric models, have been found to have a heritability between 41% and 61% (Jang, Livesley, & Vernon, 1996). Criterion 3 is therefore notionally supported. No single temperament is pathognomonic of bipolar disorder and for clinical and epidemiological reasons Savitz and Ramesar (2006) found that it could not be proven or disproven that personality and bipolar disorder co-segregate in families. Judgement on Gottesman and Gould’s criterion 4 is therefore reserved. In a recent study of bipolar probands and their siblings versus healthy controls, however, Almeida, Nery, Moreno, Gorenstein, and Lafer (2011) found that harm avoidance and self directedness co-segregated in the studied families. The evidence supports the association of bipolar illness and the cyclothymic temperament in the population, though it is less clear that there is an association between other temperament types as found in unaffected family members. Therefore criterion 5 is tentatively supported. Savitz and Ramesar (2006) concluded that "there is significant evidence to suggest that temperamental peculiarities constitute a more penetrant expression of the emotional dysregulation that characterises affective illness" (p. 334).

Hagop Akiskal has worked over several decades to define a number of temperamental profiles related to affective disorders. Temperamental dysregulation is seen as the constitutional foundation out of which affective episodes arise (Akiskal, 1996). The temperaments are proximal behavioural phenotypes arising in the premorbid course of illness – psychosocial events intervening between temperament and affective episodes. The cyclothymic temperament encapsulates what might now be termed cyclothymic disorder (American-Psychiatric-Association, 2013). Akiskal (1996) describes moody, impulsive, erratic and volatile people with interpersonal difficulties who demonstrate features of depression and hypomania with brief cycles.

The hyperthymic temperament is generally found in successful individuals with intermittent hypomanic features and a habitually reduced need for sleep. Akiskal sees this temperament as adaptive; affected persons being driven and self-assured,
sometimes diagnosed with narcissistic personality. The depressive temperament, on the other hand, is marked by a gloomy disposition and a propensity for worry and pessimism. Such persons are introverted and self critical, yet socially dependable (Akiskal, 1996). In DSM-5 terms (American-Psychiatric-Association, 2013) these individuals would probably be conceived as suffering from dysthymic disorder. In European and Japanese circles the term typus melancholicus has been applied. This term emphasises rigidity, drivenness, and social conformism. It is thought to be an endophenotype for unipolar disorder. Akiskal (1996) notes that affective episodes of different polarity may arise from the substrate of the "opposite" temperament. It has been suggested that a large proportion of those with an affective temperament will progress to full blown affective episodes (Akiskal, 1996).

In an Australian study, Murray, Goldstone, and Cunningham (2007) modelled personality correlates of the predisposition to bipolar disorder. Trait depression and trait mania could be usefully described as separable but correlated aspects of predisposition. They surveyed a student youth sample on dimensional scales and found that trait depression was determined exclusively by neuroticism, while trait mania related to extraversion and negative agreeableness. Whilst a single conventional bipolar trait model provided a good fit, they opined that it had less heuristic power than a two dimensional model. They concluded that predisposition to bipolar disorder may be understood by two reciprocally related dimensions of vulnerability related to personality correlates. They noted that neuroticism correlates most strongly with bipolar disorder, specifically its depressive pole. Sociability and goal-pursuit, as part of extraversion, map to the phenomenology of mania, though other studies have been mixed in this regard. Extraversion was found to be a significant contributor to trait mania. Their data suggests that depression contributes significantly to mania, while mania contributes modestly to depression (Murray et al., 2007). A later study by Quilty, Sellbom, Tackett, and Bagby (2009) broadly supported the findings of Murray and colleagues. The authors utilised a psychiatric population which was minimally inclusive of bipolar disorder. Because both studies did not specifically examine bipolar subjects, their results should be viewed tentatively.
Tijssen et al. (2010) investigated (hypo)manic experience in a cohort of youths. They found that the experience of manic and hypomanic symptoms was a common adolescent phenomenon which did not generally predict future use of mental health services. They followed adolescents aged 14 to 17 for up to 10 years. The cumulative lifetime incidence of either a hypomanic or manic episode was 7.6%. When hypomanic symptoms were looked at dimensionally, 26.5% of their cohort experienced four or more symptoms during the study. There were no sex differences for hypomanic symptoms, though manic episodes were significantly more common in males. They hypothesised that a dimensional representation usefully describes the (hypo)manic phenotype. Only a small percentage of the “afflicted” were in psychiatric care and the co-occurrence of (hypo)manic episodes with depression was lower than the published literature. Clinical (hypo)manic categories were robustly associated with earlier childhood disorders and male sex, though cross-sectional comorbidity was low. The incidence of (hypo)manic episodes was highest before age 22, decreasing markedly from that time onward. A dimensional continuity between subclinical and clinical categories was found. The results suggest that (hypo)manic symptoms may reflect a relatively common – and often subclinical – phenomenon in the youth population.

A number of studies have found deficits in Theory of Mind (ToM) functioning in euthymic bipolar disorder (Bora et al., 2005; Olley et al., 2005; Samamé, Martino, & Strejilevich, 2012; Shamay-Tsoory, Harari, Szepsenwol, & Levkovitz, 2009; Wolf, Brüne, & Assion, 2010). In a small Israeli study Shamay-Tsoory, Aharon-Peretz, and Perry (2009) found that affective empathy was increased in bipolar persons. Poor cognitive empathy correlated with performance in cognitive flexibility tasks. They speculate that the affective ToM enhancement in bipolar disorder may be related to a failure to inhibit emotional response, hence a "hyperempathy". A case is made that emotional empathy is a distinct simulation system, encompassing emotional contagion, personal distress, emotion recognition and empathic concern. The core cortical structure of the system is the human homologue of area F5 in the chimpanzee. From a phylogenetic perspective, emotional empathy is more related to the paleomammalian brain, as opposed to the cognitive empathy system, which arose in the ancestor common to monkeys and humans (neomammalian brain). Nummenmaa,
Hirvonen, Parkkola, and Hietanen (2008) noted that emotional empathy involves limbic (paleomammalian) areas such as the thalamus and insula, as well as cortical areas which involve the mirror neuron system. Corticolimbic connections are functionally coupled and mirroring is more vigorous than with cognitive empathy. Because it involves emotions, emotional empathy is more directly connected to approach and withdrawal action preparation systems (Nummenmaa et al., 2008).

In an intriguing study Harkness, Washburn, Theriault, Lee, and Sabbagh (2011) found that women with a maternal history of depression had enhanced ToM functioning. This was greater the younger the mother was when she first experienced depression, explaining a large amount of the variance in ToM in the offspring. The authors noted that remitted MDD and subthreshold depression were associated with enhanced ToM, while current state melancholia was associated with significant impairment. Mild to moderate depression correlated with a hypersensitivity to subtle social phenomena and this may have been a means of managing threatened relational value. The finding of Harkness et al. (2011) that young depressed mothers beget daughters with enhanced ToM suggests a genetic aetiology, as early age of onset of depression is related to genetically enriched bipolar spectrum conditions. Further, given that the syndromally depressed mothers may developmentally engender less ToM in their children (sensu Jenkins, Turrell, Kogushi, Lollis, & Ross, 2003) the converse finding bolsters a predominantly genetic contribution to their observation. These observations, along with the finding of enhanced emotional empathy in bipolar patients (Shamay-Tsoory, Aharon-Peretz, et al., 2009), raises the possibility that affective genes, in limited quantities, may enhance social cue receptivity.

Euthymic bipolar persons report more intense emotions and describe higher affective lability, suggestive of an inter-episodic trait (Henry et al., 2008). This result was replicated by Gruber, Harvey, and Purcell (2011), who reported increased positive emotion reactivity in bipolar disorder. Yet bipolar adults, ill youth and youth at risk for bipolar disorder perform more poorly than controls on facial expression recognition tasks. The evidence is consistent with the presence of neurally mediated social cognitive deficits – and dysregulation – in persons both with bipolar disorder and those at risk (McClure-Tone, 2010).
It is difficult to retrospectively ascertain premorbid temperament in those who have already developed an illness. Another approach is to assess the psychopathology in offspring of those with bipolar disorder, as undertaken by Diler et al. (2011). This study found that even non-bipolar offspring of parents who themselves had bipolar disorder had more severe dimensional psychopathology than controls. Using the Child Behaviour Checklist, the authors found a broad range of psychopathology; of internalising, externalising, anxiety, and depressive type. There was notably no increase in social disturbance in the offspring of bipolar parents, though aggressive behaviour was increased. The article does not consider the relative contribution of development in this doubly at risk population.

Andreasen (1987) studied the Iowa Writers Workshop and noted that eminent artists appear to be overrepresented with bipolar conditions. There is evidence to suggest that milder forms of spectrum conditions – and indeed those with simply a family history – have greater creative proclivities than those with full-blown conditions. In a psychoanalytically informed paper *Conformity and Achievement in Remitted Manic-Depressive Patients*, Spielberger, Parker, and Becker (1963) reviewed propositions that manic depressives are rigidly conventional and subject to authoritarian and achievement-related values. The manic-depressives parents are said to use their child’s performance to promote social position and prestige, thereby devaluing the child as a person. The emphasis is on what the child does rather than what s/he is. Social success through pleasing others becomes paramount, in this view. Bipolar persons were tested by the authors on several psychometric scales, which found an overemphasis on dependence-related and stereotyped achievement.

In a paired study Johnson and Carver (2006) investigated extreme goal setting and vulnerability to mania in young adults. They reported that mania proneness was related to the endorsement of high ambitions for political influence, wealth and fame. This is a little curious, given that, while the correlation for fame (Study 2) was 0.44, those for political influence and wealth were relatively weaker. Similarly, the scale used – the WASSUP (Willingly Approached Set of Statistically Unlikely Pursuits) - utilises almost *impossible* goals (eg. “someone will want to write a book about your life”) and asks university students to endorse these on a five point Likert scale. Were
the students faking responses? Apart from reliability and factor analysis, is the scale valid? Does it measure long-term success expectations or something else? Nevertheless, the correlation was strongest only in those with a suprathreshold Hypomanic Personality Scale. Johnson, Murray, et al. (2012) – after a number of replications – opine that "in each study, mania-proneness was related to extrinsic oriented ambitions to achieve the recognition of others, through popular fame and financial success" (p. 7). The work of Coryell, Endicott, Keller, and Andreasen (1989) around the higher rates of achievement in first degree relatives of those with bipolar disorder is consistent with the multiple threshold model of polygenic inheritance which Kelsoe (2003) promotes. Johnson, Murray, et al. (2012) note that the personality traits of impulsivity and openness to experience relate to both bipolarity and creativity. Activated positive mood states, as they occur in bipolar disorder, are related to creativity when not extreme. In a related vein, a study by MacCabe et al. (2010) found that exceptional scholastic ability was associated with bipolar disorder. Creativity, and possibly intelligence, is therefore more prevalent in bipolar spectrum conditions.

In a broad ranging review of psychosocial factors leading to the ascent into mania, Mansell and Pedley (2008) note cognitive abnormalities in bipolar persons. In particular, they cite retention of more general – relative to specific – memories and an increase in traumatic remembering.

There is evidence to suggest that people with bipolar disorder have higher Behaviour Activation System (BAS) sensitivity than controls (Alloy, Abramson, Urosevic, Bender, & Wagner, 2009). Another finding has been that goal-striving events (e.g. sitting an exam) may be associated with an increase in hypomanic symptoms in vulnerable individuals (Nusslock, Abramson, Harmon-Jones, Alloy, & Hogan, 2007).

In summary, abnormalities of temperament are common in bipolar disorder. These pertain to approach (reward dependence, novelty seeking) and withdrawal (harm avoidance). These temperaments may be endophenotypes for the condition. Cyclothymia and the depressive temperament associate with bipolar I disorder, as
does neuroticism and extroversion. The problem remains as to why these temperaments are related to the condition – what intrinsic function do they serve – or are they incidental? It is possible that bipolar disorder may be constituted not by a single temperamental diathesis, but by separable and interrelated trait mania and trait depression. Tijssen has found that unipolar (hypo)mania may be a relatively common developmental phenomenon. Deficits in ToM are common in bipolar disorder, while affective empathy may be increased. Children of women with a depressive disorder may have an enhanced ToM, suggesting that, in some circumstances, affective genes – or the experience of a depressed mother – may confer advantage with regard to group process. Bipolar disorder is not just an episodic condition, but one marked by temperamental abnormalities and affective changes. Whilst Diller's study does not rule out developmental contributions to psychopathology in the offspring of bipolar probands, it does suggest that the genetic diathesis affects a broad range of psychic domains. Paralleling Harkness and Shamay-Tsoory’s findings of selected advantage in bipolar conditions, it is documented that creativity is more closely associated with the familial propensity to bipolar disorder, than with the disorder itself. The inclination of bipolar persons to seek conformity and social prestige – and its possible developmental contribution – is consistent with the argument pursued in this thesis.

**Developmental Life Events**

Life events interact with temperament to influence the course of bipolar disorder and Figure 1 illustrates the myriad interactions which are possible. Parenting characterised by low care and high overprotection may be associated with bipolar disorder, though the early studies are inconsistent and suffer from methodological limitations (Alloy et al., 2005). Subsequently, in a complex and comprehensive cross-sectional study of persons with bipolar spectrum conditions compared to controls, Neeren, Alloy, and Abramson (2008) found that lower levels of acceptance/warmth from mothers – and high levels of negative psychological control from both parents – were associated with bipolar spectrum diagnoses. The authors controlled for family history of bipolar disorder as well as current mood state. They found that emotional and physical maltreatment by parents correlated with bipolar spectrum diagnoses. In a controlled study of 60 bipolar outpatients Watson et al. (2013) found that emotional
neglect was the only Childhood Trauma Questionnaire subscale associated with bipolar disorder.

The nascent literature on maltreatment histories as risk factors is less consistent, but worthy of further investigation (Alloy et al., 2005). In a review of the relationship between childhood physical and sexual maltreatment and adult bipolar disorder, Alloy, Abramson, Walshaw, Keyser, and Gerstein (2006) found mixed support for the association. Leverich et al. (2002) found an association between early maltreatment and clinical dimensions of bipolar disorder. Childhood abuse is associated with an earlier age of onset (Daruy-Filho, Brietzke, Lafer, & Grassi-Oliveira, 2011; Leverich et al., 2002; Post & Leverich, 2006), more rapid cycling (Leverich et al., 2002), suicide attempts (Daruy-Filho et al., 2011; Etain, Henry, Bellivier, Mathieu, & Leboyer, 2008; Fisher & Hosang, 2010), personality disorder, alcohol and drug abuse (Daruy-Filho et al., 2011; Leverich et al., 2002) and a higher rate of psychosocial stressors in bipolar outpatients (Leverich et al., 2002). Causality remains a major problem as apparent psychosocial antecedents may be concomitants of bipolar disorder symptomatology (Neeren et al., 2008) – see Figure 1. Neeren et al. (2008) found that sexual maltreatment did not associate with bipolar spectrum illnesses, while the use of a retrospective self-report measure limits the interpretation of their findings.

In a Swiss epidemiological study, Angst, Gamma, Rossler, Ajdacic, and Klein (2011) found that sexual trauma and conduct problems were unrelated to chronicity in bipolar and unipolar mood disorders. Childhood family problems, on the other hand, were found to be strong risk factors for the chronicity of unipolar and bipolar mood disorders. Childhood adversity was associated with chronicity in bipolar disorder, as was childhood/adolescent anxious personality (Angst, Gamma, et al., 2011). Low self-esteem and poor mastery in early adulthood were associated with chronicity, also, suggesting to the authors that early trait anxiety may mediate the relationship between childhood adversity and mood disorder (Angst, Gamma, et al., 2011).

In a recent critical review, Fisher and Hosang (2010) found significant methodological problems with the extant studies on the relationship between bipolar
disorder and childhood maltreatment. Only six of twenty nine studies utilised psychometrically tested measures, diagnostic interviews, a normal control group and included at least one hundred affected individuals. The rates for childhood maltreatment, though tentatively higher, varied and they found inconsistency in the association with clinical expression. Concurring with Etain et al. (2008), they noted that all studies were cross-sectional, ruling out the ascertainment of aetiology. They concluded that "given the limited evidence available, it is not possible to draw any firm conclusions about the association between childhood maltreatment and the onset or clinical characteristics of bipolar disorder" (Fisher & Hosang, 2010, p. 9). They call for more scientifically rigorous approaches, utilising prospective longitudinal designs, detailed standardised instruments, representative samples, control groups and adequate statistical power. Evidence is lacking that the majority of cases with bipolar disorder have experienced childhood maltreatment; therefore they conclude that there does not seem to be evidence that most individuals exposed to childhood maltreatment will develop bipolar disorder. Other biological, psychological and social factors are likely to be involved in the aetiology of the condition and should be explored alongside childhood maltreatment (Fisher & Hosang, 2010).

Etain et al. (2008) on the other hand, cite growing evidence to support the association between childhood trauma and bipolar disorder. They found that their Childhood Trauma Questionnaire (CTQ) trauma scores were notably higher for bipolar than for healthy subjects. Emotional abuse was more frequent in the bipolar group than in the control cohort. Etain et al. conclude that “childhood trauma is associated with a more severe clinical expression and course of the disease” (Etain et al., 2008, p. 869). They found an association between bipolar disorder and affective lability and intensity (sensu Henry et al., 2008) and concluded that these were core dimensions of the disease during inter-episodic euthymic periods. Recall bias remained a possible explanation for the association of relational trauma and bipolar disorder, as emotionally disturbed adults might over-report emotional abuse (Etain et al., 2008) – this consideration applies to my MOPF study. This highlights the possibility that genetic characteristics – affecting the parents and their offspring (Figure 1) – may lead to an increase in the reporting of childhood trauma. Further to the genetic hypothesis, intergenerational transmission of trauma may contribute to the
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association between traumatic events and bipolar disorder. Seeming to contradict their previous statements, Etain et al. (2008) opine: "However, no study has definitively demonstrated aetiological causality between childhood trauma and bipolar disorder.” (p. 872)

Hammersley et al. (2003) drew on a psychotherapy patient sample to ascertain any association between reports of childhood sexual abuse and reported hallucinations. Though they report a significant association between general trauma and auditory hallucinations in bipolar disorder, their sample was small and does not obviate the possibility of a reporting bias in the therapists. They advised that this association warranted further investigation.

Post and Weiss (1998) applied the constructs of sensitisation and kindling to mood and other disorders and noted that psychiatric illness often demonstrates progression over time, due to putative experience-dependent modifications of the gene. Both sensitisation and kindling result in increased physiological responsivity to the same stimulus with repeated administration. Kraepelin (1921) had noted that the course of untreated illness is marked by shorter remissions between episodes and a shift from loss-induced episodes to episodes that occurred more spontaneously. Environment, whether it be one of deprivation or enrichment, interacts with acute and chronic stressors to have ongoing effects on neural development (Post & Weiss, 1998). The authors note that "vulnerability for each of these syndromes could be set either by an inherited genetic mechanism or by an experience-dependent, stressor-mediated impact on gene expression in critical periods of brain development in pre- and perinatal development and childhood, or by both mechanisms in combination, providing a 'double hit' like that required in the development of some cancers." (Post & Weiss, 1998, p. 197). They further note that "psychosocial stressors related to work, family, and social interactions, with important threats to self-esteem unbalanced by psychosocial support and compensatory adaptive mechanisms (Post et al 1996b; Post and Weiss 1997), could be sufficient to trigger or reactivate an episode of recurrent affective illness (Paykel 1979; Brown et al 1986)." (Post & Weiss, 1998, p. 198).
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Though it is often genetically mediated, the role of environment in bipolar illness may be significantly underestimated, as around 50% of bipolar patients do not have a family history of bipolar disorder in first degree relatives (Post & Leverich, 2006). Animal models have shown that the severity, quality, and timing of environmental events each play a critical role in neurobiology and behaviour (Post & Leverich, 2006). The studies of Post and colleagues found that bipolar adults with a history of severe environmental adversity had a more complicated course of illness (Post & Leverich, 2006). They note an iterative effect between childhood abuse and a family history of bipolar disorder and with both factors present the age of first symptoms is significantly lower than all other groups. According to Post and Leverich (2006) physical and sexual abuse interact to increase the occurrence of suicide attempts more than either stressor alone. In accord with Angst, Gamma, et al. (2011), Post and Leverich (2006) found that an early abuse history correlated with an increase in proportion of time ill in those with early abuse histories. Early childhood adversity is associated with negative life events in adulthood (Leverich et al., 2002), though it is unclear which intervening variables account for this association. Parental affective – and other – illnesses may impact for both biological and developmental factors.

In summary, developmental life events are part of an interwoven system of variables contributing to bipolar disorder. There is good evidence that parental care characterised by low acceptance and high control is associated with bipolar disorder, though it is unclear whether this is causal or reflective of other relationships. Maltreatment may not cause bipolar disorder, but it certainly appears to affect its course and prognosis. Complex interrelationships between family culture, genes, trauma, and maltreatment are likely to affect the pathogenesis and pathoplasty of bipolar spectrum conditions, though form and function require elucidation. Life events may lead to physiological changes in the central nervous system, contributing over time to the initiation of mood episodes by less intense life events.
Contributory variables associated with relational trauma and bipolar pathogenesis

**Figure 1**. Contributory variables associated with relational trauma and bipolar pathogenesis

**Cognitive Patterning**

Affective and motivational temperaments interact with life events through the aegis of cognition. Cognition may be a cause and consequence of both mood and behaviour and it has been found that cognitive styles in the bipolar disorders are equally as negative as those found in unipolar disorder (Jones et al., 2005; Lam et al., 2004, as cited in Reilly-Harrington et al., 2010; Scott & Pope, 2003).

Studies utilising the Dysfunctional Attitudes Scale (DAS) generally find that bipolar patients score more highly in terms of perfectionism, sociotropy (dependency) and need for approval (Scott, Stanton, Garland, & Ferrier, 2000). Persons with bipolar disorder tend to hold extreme personalised appraisals of self and internal states (Mansell & Pedley, 2008). Core self-belief states pertaining to social inclusion – those associated with *helplessness* and those associated with *unlovability* (Beck, 1995) – may be aberrant. These core beliefs may be organised into interwoven structures –
cognitive schemas – which both codify and process reality (Beck, Rush, Shaw, & Emery, 1979). Social contexts may be both aggravating factors and buffers to depressive cognitions (Beck et al., 1979). Common status claims include "I am helpless" and "I am a failure", while relational core beliefs involve self statements such as "I am unworthy" and "I am unattractive" (Beck, 1995). Core negative beliefs centre on the self as perceived by others, a concept later examined under the rubric of prestige.

Bipolar individuals tend to endorse high ambitions for wealth, fame and political influence (Johnson & Carver, 2006). Bipolar cohorts have been found to be highly self-critical, or else self-affirming. These cognitive styles tend to predict ongoing manic symptoms. Scott et al. (2000) studied euthymic bipolar patients and controls and found that the bipolar patients performed poorly on social problem solving and had an over-general recall of autobiographical memory. Greater cognitive abnormalities in the bipolar disorder group were noted in persons with a longer duration of illness and more frequent episodes of disorder. The possibility that causality might run in either direction was canvassed.

Alloy, Abramson, Flynn, et al. (2009) found that persons in the bipolar spectrum had higher rumination and private self-consciousness. Private self-consciousness refers to self-directed thoughts, moods, and motives. Public self-consciousness, on the other hand, indicates observable aspects of the self as seen by others. The authors found that depressive symptoms were significantly correlated with rumination, private – and public – self-consciousness, and social anxiety. Depressive rumination prospectively predicted the number of major depressive episodes, whilst private self-consciousness indicated a likelihood of onset of (hypo) manic episodes over 3.5 years. Though the correlation was only 0.24, bipolar individuals had significantly higher levels of public self-consciousness than did controls, which correlated significantly with depression and rumination. Therefore, the bipolar spectrum is associated with a sensitivity of the self to internal critique and external scrutiny.
In a study of high scorers on the hypomanic personality scale it was found that Entitlement/Grandiosity and insufficient Self-Control/Self-Discipline positively predicted bipolar risk. Vulnerability to Harm or Illness also predicted group membership and Entitlement/Grandiosity indicated narcissism and excessive goal striving. A lack of affective and behavioural inhibition is further suggested, consistent with bipolar spectrum phenomenology (Hawke, Provencher, & Arntz, 2011). There is also evidence of maladaptive cognitive and coping styles in those at genetic or behavioural risk for bipolar disorder (Alloy, Abramson, Flynn, et al., 2009).

In summary, a broad but relatively consistent pattern of cognitive disposition has been found in bipolar disorder, though in some areas this does not differ from unipolar disorder and it is notable for implicit negativity. Bipolar persons tend to be more perfectionistic and goal oriented and they are prone to more extreme self appraisals which are often of dichotomous type. They may need more approval and tend to be sociotropic, and as Alloy, Abramson, Flynn, et al. (2009) have found, there is an increase in private and public self-consciousness and rumination. The study of Hawke et al. (2011) is indicative of increased narcissism and a number of researchers have pointed to deficits in Theory of Mind processing. These patterns of cognition coalesce to extreme views of the self which, in general, tend to those of self failure and lack of relational value.

Self-Esteem

“One of the most important and durable problems that is posed to an individual by his insertion into society is to find, create and define his place in these networks. It is reasonable to assume that both his ingroup and outgroup attitudes and behaviour must be determined, to some extent at least, by this continuing process of self-definition.” (Tajfel, 1974, p. 67)

Coryell et al. (1989) undertook a study comparing first degree relatives of unipolar depressive, bipolar I and II probands for social achievement. Previous studies had found a link between high socioeconomic status and affective disorder. The authors found that membership of bipolar pedigrees (especially bipolar I) was associated with educational and vocational advantage (Coryell et al., 1989). Goodwin and Jamison (2007) further described increased occupational levels in the families of
bipolar individuals. Tsuchiya, Agerbo, Byrne, and Mortensen (2004) found that higher educational and financial level in parents was associated with increased risk for bipolar disorder in offspring. Milder forms of the illness may be associated with high achievement, but the distal expression of illness in offspring may be more severe (Goodwin & Jamison, 2007). "The presence of achievement, success and creativity in the ‘well’ relatives of bipolar patients indicates that the ‘dilute’ genotypes of the illness may have evolved to subserve adaptive functions such as exploration and risk taking." (Akiskal, 2008, p. 10).

High self-esteem is known to be correlated with hedonic feelings (Baumeister, Campbell, Krueger, & Vohs, 2005; Buss, 2012), better sleep, increased resilience and better social integration (Myers, 2007). Low self-esteem, on the other hand, may be associated with unhappiness and depression. In 1995 a series of five experiments, Leary, Tambor, Terdal, and Downs (1995) turned the discourse on self-esteem on its head, suggesting that – rather than being a cause or consequence of psychological events – self-esteem was instead a monitor of relational value which served to avoid social devaluation and rejection. They termed this concept sociometer theory, echoing the contention of Cooley (1922) that we live in the imaginations of others. Leary, as cited in Buss (2012) outlines the evolutionary logic that our ancestors lived in groups and needed each other to survive and reproduce. Thus the motivation arose to seek company, develop social bonds, and curry favour within the group – selection favouring a mechanism which declared the degree of acceptance by conspecifics. The mechanism to facilitate this, Leary (1999) contends, is self-esteem, arising as a function of the sociometer. The sociometer "(1) monitors the social environment for cues that are relevant to the individuals relational value to other people; (2) it responds with negative affect (and typically lowered self-esteem) prompting a conscious assessment of the situation; and (3) it motivates behaviours intended to enhance relational value or at least forestall further declines. Thus, the sociometer consists of three separate but interrelated processes that involve a monitor, the output from the monitor, and a motivator..." (Leary, 2005a, p. 88). Research has confirmed that higher perceived social inclusion is linked to higher self-esteem (Buss, 2012; Leary et al., 1995). Leary concludes "By focusing on the monitor rather than on what the monitor measures, we have been distracted from the underlying interpersonal
processes and the importance of social acceptance to human well-being” (Leary, 1999, p. 35).

Is self-esteem a factor in bipolar disorder? A number of studies have found that euthymic persons with this condition score normally on self-reported measures of self-esteem (Knowles et al., 2007; Pardoen, Bauwens, Tracy, Martin, & Mendlевич, 1993; Winters & Neale, 1985). On the other hand it has been found that self-esteem is lower on implicit measures, supporting earlier psychodynamic theories (Winters & Neale, 1985). The dichotomous self concepts of bipolar persons were indicated by the inclination of bipolar subjects to make tacit personal assumptions of success, while overtly conceding a personal role in failure scenarios (Knowles et al., 2007; Winters & Neale, 1985). As long ago as 1928 Rado, as cited in Lyon, Startup, and Bentall (1999) opined that bipolar persons were at risk of low self-esteem by virtue of intense narcissism. While depressive subjects’ self-esteem hinges on distressing internalised feelings of inadequacy, worry and guilt, it has been found that the self-esteem of bipolar persons relates to social, leisure and conflict situations (Blairy et al., 2004; Pardoen et al., 1993) – that is an external (prestige) locus. Knowles et al. (2007) found that the self-esteem and affect of bipolar persons were unstable compared with unipolars and controls. Self-esteem theoretically relates to rejection sensitivity, an accompaniment to bipolar atypical depression (Thase, 2009) and this may be a predictor of relapse in bipolar disorder (Scott & Pope, 2003). Therefore, a range of studies have found abnormalities of self-esteem in bipolar disorder.

**Conclusion**

"The SOCIAL SCIENCES rest on simple psychologies.... Anthropological man hungers not so much the money and material goods as for prestige. Whenever anthropologists discuss competition and jealousy, speak of potlatches or counting coups, describe displays of ritualized boasting and ostentation or even the emergence of social stratification, they are making a basic assumption: men seek prestige and status, deference and respect. Such an assumption calls not for challenge – there is no reason to suspect its validity – but for analysis. This paper seeks to explore the idea that, everywhere, men require prestige to maintain self-esteem" (Barkow et al., 1975, p. 553)
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We have seen that the template for the bipolar disorders is wrought from temperament, overlaid by development and shaped by the social milieu. Such a complex iteration is unlikely to give up its secrets easily. Nevertheless, there is emerging data that illuminates the particular relationship between bipolarity and the social world. Our first evidence was the suggestion that bipolarity may be rooted in temperament itself, with the caveat that this disposition may be tilted toward harm avoidance and sociotropy. Further, there is support for the notion that bipolar disorder carries with it an endophenotypic “stamp” which may run in families. This trait may confer social, cognitive and creative benefit. The bipolar disposition is socially directed, increasing the motivation to achieve the recognition of others. This proclivity may be augmented by early familial difficulties. Sociometer theory has opened up new understandings of self in the bipolar disorders. Complemented by Barkow’s work, we can now see the “aboutness” of self-esteem; that it is a dynamic internalisation of societal prestige. Bipolar persons may have an external locus of esteem and a sociometer akin to a “smoke alarm”. Bipolarity may, therefore, warn of relational threat and launch the diatheses which over millennia have served social inclusion, survival and reproduction.
Chapter 2: Social Contexts of the Bipolar Spectrum

Introduction

The previous chapter depicted bipolarity as a temperamental template, onto which life experiences may accrue. Clearly, this process does not occur in a vacuum, as the social matrix encompasses individuals and provides a recognisable form to bipolar disorder. Yet, what is the relationship of society to bipolarity? This chapter seeks to describe bipolarity from a social perspective and to provide a model for the relationship of self to life events, social support and mood state. The relationship of bipolar individuals to society will be examined and demographic features will be explored. A history of thinking on social support – prestige – will be elaborated and the powerful effects of social affiliation on mental and physical health will be detailed. The role of life events in the genesis of bipolar mood episodes is investigated and found to be a matter of some complexity. More chronic forms of stress, as exemplified by expressed emotion, are discussed with relation to bipolar disorder. A summation of the evidence follows and draws together a model of the factors involved in illness pathogenesis, with reference to the unifying construct of self-esteem.

Society

The term society refers to a multi-dimensional fabric of interdependent institutions and subgroups, based within shared – and fluid – cultural and geographical boundaries. Society inhabits the realm of macrostructures, such as politics and religion, to what Berkman, Glass, Brissette, and Seeman (2000) refer to as the mezzo and midi of social groups and institutions, finally extending its reach into the micro level of the individual and his or her biology. It is an intangible web that binds, nourishes and sometimes punishes individuals, as they interact with others, locating persons through multiple identities and providing linkage through moieties such as language. Individuals live a finite time within their social node, but society persists, incessantly evolving mores and methods. Society shapes mind, providing a context for disorders of the bipolar spectrum.
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There are rich systemic associations between biology, the individual, bipolarity and society at large. Social support and life events may contribute to the illness burden of the bipolar spectrum, whilst societal bipolarity reflexly determines health policy and community perception. In addition, a subgroup of individuals with bipolar spectrum conditions has contributed to societal creativity and culture, even though persons with mental illness form a disadvantaged social substratum overall. Bipolar mood episodes may either generate or diminish prestige and these effects in turn shape social leadership and social capacity.

Bipolar conditions carry a significant direct and indirect cost in terms of health care expenditure, work absence and short-term disability (Shippee et al., 2011). In the United States, the bipolar disorder population is more likely to be unmarried, poorer, less educated, unemployed (OR 4.6, 95% CI 3.52, 6.04) and living alone. They are 3.56 times more likely to miss at least two weeks of work per annum (95% CI 2.12, 6.04) and 40.8% of them acknowledge a limitation in the domains of work, household and school compared with 4.8% of the non-mood disorder population (Shippee et al., 2011). Bipolar disorder represents not only a clinically unique subset of affective symptoms, but also a demographically different population (Shippee et al., 2011). As has been already noted, authors such as Akiskal and Akiskal (2005) and Kelsoe (2003) have argued that more dilute phenotypes of affective disorder confer adaptive advantages, both to individuals, and their social group. A proclivity to leadership and creativity may therefore arise as a subgroup of the disadvantaged community and their relatives which Shippee et al. (2011) have here described.

Social relationships provide the context and the substrate for most of Maslow’s hierarchy of needs (1943), ranging from physiological needs, to safety, love and belonging, self-esteem-respect and self-actualization. In her doctoral dissertation: Successful Management of Bipolar Disorder and the Role of Social Support, Buila (2005) traces the history of thinking on social support, quoting Cassel in suggesting that social support acts as a buffer to stress. Cobb as cited in Buila (2005) saw social support as constructed of information, which conveys – in effect – Maslow’s esteem, love and belonging needs, moderating stressful events. Social affiliation has powerful effects on mental and physical health and on mortality, actualising Durkheim’s
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objective to explain individual pathology in terms of social dynamics, where anomic suicide was triggered by society’s incapacity for integration (Berkman et al., 2000). Berkman et al. (2000) argue that social networks act through providing social support, social influence, providing engagement and attachment and via access to resources and material goods. These, in turn, influence the stress response, psychological states and traits, health-related behaviours and disease exposure.

It therefore appears that the relationship between the individual and his or her society impacts broadly upon psychological wellbeing. Does an event, such as migration, alter the relationship of self to society and thereby affect psychiatric morbidity? Swinnen and Selten (2007) have undertaken a meta-analysis to examine this question, drawing on studies which generally used a retrospective case-note design, concerned with first psychiatric hospitalisation. The relative risk of bipolar affective disorder in migrants, excluding African-Caribbean people living in the United Kingdom, was found to be 1.75 (95% CI 0.94, 3.28), which was a non-significant result. They found no conclusive evidence that there was a large increment in the risk of mood disorders among migrants, though they conceded a relatively small increase. This may, in part, be due to differences in treatment and hospital admission practices between ethnic groups (Swinnen & Selten, 2007).

In a comprehensive international study on the prevalence of bipolar spectrum conditions across 11 nations, Merikangas et al. (2011) found significant differences between countries. The United States had a lifetime prevalence of bipolar spectrum disorders of 4.4% and a twelve month prevalence of 2.8%, contrasting with India, which had only 0.1% in each group. While under-recognition, stigma and study implementation factors may account for an under-reporting of prevalence in some third world countries, it remains possible that there may be actual differences between nations, accounted for by social factors.

Social capital has been described as the aspects of social organisations that facilitate action and cooperation for mutual benefit (Putnam as cited in Stafford, De Silva, Stansfeld, & Marmot, 2008). Various authors have investigated the link between social capital and psychological morbidity. Stafford et al. (2008) found evidence for a link between social capital and common mental disorders, but only for
neighbourhoods in deprived circumstances, supporting Cassel’s buffering hypothesis (as cited in Buila, 2005). Two types of social capital are recognised: bonding and bridging, each divided into structural and cognitive forms. Bonding social capital acts at a micro level involving the individual and household, whilst bridging social capital operates at the macro level of community groups and organisations (Almedom, 2005). Some authors have found a deleterious effect between bonding social capital and mental distress (Mitchell and LaGory as cited in Almedom, 2005). Giordano (2011), on the other hand, found that in multivariable models, generalised trust was the sole social capital variable with a robust relationship to psychological health over time. Helliwell (2004) found that increased social capital and higher levels of trust were associated with reduced suicide rates compared between nations, as well as being associated with high levels of subjective well-being. These findings were replicated by Kelly, Davoren, Mhaoláin, Breen, and Casey (2009). Denney (2010) combined a large health interview survey database with suicide mortality figures to find that, at a family level, increased social support and integration reduces the risk of suicide.

Society is therefore a living multifaceted web which evolves over time in response to chaotic fluxes. It contains pockets of advantage and disadvantage, metering persons’ needs through a process of information flow. The self in society is part of a dynamic process, of which an essential part is the perceived and material support individuals procure from those in their network.

Social Support

“In summary, current results suggest that social support is an important aspect of course within bipolar disorder. Coupled with results from the expressed emotion literature, it appears that both positive and negative aspects of relationships are important determinants of bipolar symptoms.” (Johnson, Winett, Meyer, Greenhouse, & Miller, 1999, p. 570)

There are various ways to conceive of social support and selected definitions include: (a) the provision of a resource, (b) a description of the positive outcomes of support, (c) an implied positive outcome (d) a description of relationship between the provider and the recipient, (e) social integration, (f) social environment or climate, (g) network resources and (h) reciprocity (Hupcey, 1998). Typically, each of these terms
implies the presence of several others and social support may be viewed as a holism. Hupcey (1998) goes on to describe the structural features of social support: from the perception of need and the motivation to take well-intentioned action to a conspecific, leading to a positive change in the recipient.

Pratchett (2010) researched social support in bipolar disorder as a part of her doctoral dissertation. She found that lower levels of perceived total social support correlated with greater subsyndromal symptoms of depression and mood elevation at baseline. Reduced support was associated with the persistence of symptoms at six months. She suggested that perceived social support may be a variable associated with trait subclinical mood state. The direct effect model of social support suggests that a stable social role and regular positive experiences are operant in maintaining euthymic mood. The alternate model is that of the stress-buffering effect, though both appear to operate as protective factors. Pratchett (2010) concurred with other authors that perceived support is more relevant than actual support in terms of its predictive value for health outcomes. Various social support functions have been identified and these include emotional, informational, appraisal and instrumental support (Langford as cited in Pratchett, 2010). Cohen as cited in Pratchett (2010) has added self-esteem and belonging support to this definition. Social support may impact on disorders comorbid with bipolar disorder, such as anxiety. The majority of studies suggest that social support is generally lower among bipolar patients, even when euthymic, compared with the general population (Pratchett, 2010).

Studies such as that of Johnson et al. (1999) have found a correlation between social support and the course of bipolar I mood symptoms, noting a relationship between low social support and increased symptoms of depression. Social support was measured by reference to self-esteem and belonging support, along with tangible variants. Not all patients in this study were euthymic and thus mood level may have contributed to perceived social support. Cohen, Hammen, Henry, and Daley (2004) studied stable bipolar I patients and found that low social support predicted the recurrence of depressive, but not manic episodes. Contrasting with the study of Johnson et al. (1999) – which had found a polarity affect for depression only – the study of Pratchett (2010) found that greater levels of mood elevation correlated with low social support. Previous studies have looked at bipolar I disorder only, whereas
bipolar II and bipolar NOS were included in her study. A persistence of subsequent mood symptoms was found to correlate with low social support. Of course low mood will alter perception of social support, making attributions of causality difficult.

The review by Alloy et al. (2005) found reasonably consistent evidence to conclude that positive and negative interactions with family and friends impacts on the course of bipolar disorder. Bipolar individuals with poor social support recovered more slowly, had a higher likelihood of relapse, and had a higher impairment than individuals with high social support. Interventions designed to enhance relatives’ social support decreased the risk of mood episodes. The authors caution that much of the literature in this area has significant methodological limitations. A further study by Depp and Meeks (2002) in 109 middle and old age individuals with bipolar disorder found that, while controlling for time point 1 symptom levels, social network attrition predicted elevated manic symptoms and functional impairment at time point 3. A comparison group with major depression did not reflect these findings, which suggested to the authors a diagnosis-specific social network effect in late-life bipolar disorder. Romans and McPherson (1992) found a similar attrition of social interaction with increased age and duration of illness, in probability related to more deleterious manic episodes.

It is worth noting that social support may also have a negative valence, as described in the literature on expressed emotion (EE), which is described as a significant predictor of the course of bipolar disorder (Miklowitz, 2011). EE is a familial phenomenon, referring to the expression of hostility, critical attitudes or emotional over-involvement amongst caregivers of individuals in an acute episode of psychotic or mood disorder. Individuals living in families with high EE have higher rates of relapse – or more extensive symptoms – than those with caregivers expressing low EE (Miklowitz & Johnson as cited in Miklowitz, 2011). Psychosocial interventions, especially those with a focus on the family and social relationships, are linked to both better psychosocial functioning and a more rapid recovery (Miklowitz, 2011). Miklowitz (2011) describes findings from the STEP-BD study which examined bipolar patients randomly assigned to one of three psychotherapy treatments (plus pharmacotherapy) versus a three session psychoeducational plus
pharmacotherapy comparison group. Patients receiving one of the psychotherapies, in addition to pharmacotherapy, recovered from their depressive episode in a mean of 169 days, as against 279 days in the control group. Overall functioning, relationships, and life satisfaction were improved by psychotherapy.

**Life Events**

Negative life events affect the experience of an individual with respect to his or her world, bending social relationships and creating detriments. The relationship between major life events and affective disorder has focused principally on unipolar depression, as evidenced in the meagre attention paid to bipolar disorder by Paykel (2003) – who noted that mania itself may produce life events; necessitating particular care to define events which are independent. Of course, depression may also trigger life events through irritability, hopelessness and social withdrawal. These phenomena have been formalised under the concept "stress generation" (Bender, Alloy, Sylvia, Urosevic, & Abramson, 2010). As of early this century, Paykel (2003) noted that prospective studies have found mixed evidence for the role of life events and mood episodes. By 2009 authors such as Miklowitz and Johnson (2009) were able to describe bipolar disorder-relevant life events of three types: negative, goal attainment and social-rhythm disrupting. They noted that several longitudinal studies found robust predictive power in relation to life events, especially with regard to bipolar depression. Chronic stress, which exclusively involved family and romantic relationships, was related to increased depression in bipolar adolescents (Kim et al. as cited in Miklowitz & Johnson, 2009). They found little evidence for a direct effect on mania, however. Miklowitz (2011) later concluded that family conflict and criticism, as well as life event psychosocial stressors, were strong predictors of recurrence timing and the degree of recovery from episodes.

Alloy et al. (2005) opined that "the life events literature has been fairly consistent in suggesting that bipolar individuals experience increased stressful events prior to onset or subsequent episodes of the disorder" (p. 1047). This statement may cause some confusion by implying that most, or all, bipolar patients experience episode-precipitating events consistently. Johnson (2005a), writing in the same year,
found significantly more ambiguous data (with small effect sizes) to support the link between life events and relapse. She indicates that many depressions are unrelated to the occurrence of negative life events, being precipitated by other variables. She notes the problem with what is in effect effort after meaning, when research arises in cross-sectional and post hoc analysis. Johnson (2005a) subsequently stated that "in sum, it would appear that independent, severe negative life events are as common before bipolar depression as they are before unipolar depression..." (p. 1015). This was qualified by noting that two of three studies indicate that there is no clustering of negative life events before depressive episodes and "on the other hand, life events are not that common before bipolar depression..." (p. 1016). Yet it was Miklowitz and Johnson (2009) who found that there was "robust predictive power for life events" (p. 284). Alloy, Miklowitz and Johnson therefore appear to be making strong claims for the relationship between life events and bipolar depression, based on relatively meagre evidence.

Proudfoot, Doran, Manicavasagar, and Parker (2011) give more tentative support to the link between stressful life events and the precipitation of bipolar (hypo)mania – noting some contradictory studies. They favour the kindling notion that severe life events tend to trigger earlier rather than later illness episodes. The retrospective nature of most studies limits interpretation, necessitating further prospective research.

Cohen et al. (2004) have undertaken a study of 52 individuals with bipolar I disorder, structuring their research sensibly with a prospective design, albeit with a small number. They found that higher levels of stress – along with reduced levels of social support – independently predicted the recurrence of depression over a one year period, controlling for clinical history and medication compliance. Social support, however, was found not to moderate the impact of stress. They admit that the causal direction between support, life events and disorder cannot be determined definitively in their research. Negative life events were, however, rated post hoc at six monthly intervals which may have led to a recall bias and the privileging of macro over minor events. There was an implicit assumption in their calculation that the numbers of socially supportive relationships was more important than the quality of that support.
Though the $p$ values for the effects of total network support were significant, the odds ratios for recurrence are modest at around 0.92. The study raises the important possibility that morbidity and social support may be reciprocally interacting, rather than social support simply predicting relapse. Morbidity may reduce support, which may in turn drive morbidity up further.

Miklowitz and Johnson (2009) reviewed the literature on goal-attainment life events and bipolar episodes, arguing for a connection between basal ganglia dopaminergic reward sensitivity and mania. Eisner, Johnson and Carver, as cited in Miklowitz and Johnson (2009) note that individuals with bipolar disorder demonstrate augmented responses in confidence in response to small success experiences, controlling for current symptomatology. They take the view that increased reward sensitivity in bipolar disorder results in a greater sensitivity to life events which involve success, in turn elevating confidence and promoting goal engagement. This pathway may lead to mania. Support for this hypothesis is found also in the review of Proudfoot et al. (2011).

**Synthesis**

“It seems reasonable to conclude that vulnerability to bipolar symptoms is robustly associated with unstable self-esteem. The more extreme differences in self-esteem and related measures observed in different phases of the disorder (Ashworth et al., 1982; Bentall et al., 2005) suggest that these changing perceptions of the self are a core feature of the condition.” (Bentall et al., 2011, p. 407)

Within society, individuals are like buoys in the ocean; their stability governed by the ebb and flow of the societal network. Intermittent major life events perturb this surface, testing the social fabric and impacting on persons and their needs. Yet, this metaphor does not completely capture the relationship of the bipolar individual to society, being focused on the *macro* and not the *micro*. Despite her summation, Johnson (2005a) found only modest support for the effects of negative life events on the depressive phase of bipolar disorder and found little support for an effect on (hypo)mania. Perhaps stressors for bipolar episodes are constituted not directly by cataclysmic events, but by the day to day fluctuations of life as they impact on cognition and mood? Miklowitz and Johnson (2009) have documented a strong
association between EE and mood episodes; chronic stress is like a *microclimate* for mood state. As already noted, it was chronic stress which involved intimate others, that was related to increased depression in bipolar adolescents (Kim et al. as cited in Miklowitz & Johnson, 2009). There is, furthermore, a robust association between social support and subclinical mood state (Alloy et al., 2005; Cohen et al., 2004; Pratchett, 2010).

Havermans, Nicolson, and deVries (2007) noted that there is a general ignorance about the way in which bipolar patients perceive daily uplifts and hassles. Current depression, along with an increased number of affective episodes, was found to associate with greater levels of stress. Whilst the overall reactivity of negative and positive affect was similar in bipolar individuals and controls, the subgroup of bipolar persons with subsyndromal depression showed increased negative affect sensitivity to daily hassles (Havermans, Nicolson, Berkhof, & deVries, 2010). Johnson et al. (2008) have conducted a study on the mood trajectories which follow daily life events, to find that depressive moods last typically six to nine hours after an index event. They found that a history of mood disorder did not moderate the duration of mood downswing within, or across, days. However, whilst 40% of their university sample was diagnosed with mood disorders, unipolar and bipolar groups were aggregated in the analysis, making it impossible to ascertain the relationship between bipolar disorder and mood downswings. Equally, over a span of seven days in a substantially non-clinical population, the likelihood of the onset of a sustained depressive episode seems low.

Figure 2 depicts a proposed relationship between life events, social support, cognition, response style, mood state and morbidity. The evidence cited in this chapter supports this synthesis, though the pivotal role of self-esteem has not yet been substantiated. Could a unitary construct unite these apparently dissimilar concepts?
Scott and Pope (2003) found that decrements of self-esteem predict relapse, especially into bipolar depression. Johnson et al. (1999) found that social support influenced the onset of depression but not mania. The social support measure utilised was the ISEL, including self-esteem and belonging items, suggesting that the internalisation of societal prestige to self-esteem is pivotal to the onset of depression. Depressed bipolar patients display low self-esteem by having a significant gap between their self-actual and self-ideal domains, when compared to controls and persons in manic, hypomanic or remitted phases of the disorder. There is a similar discrepancy between their estimations of self and their perception of the way others evaluate them (Bentall, Kinderman, & Manson, 2005). As demonstrated by Knowles et al. (2007), persons prone to hypomania (as indicated by the Hypomanic Personality Scale), show significant fluctuations in self-esteem. This was considered by Bentall et al. (2011) to be a robust sign that self-esteem is a core feature of bipolar disposition,
though their study did not examine the duration of self-esteem and affective response to stress. Similarly, the study of Havermans et al. (2007) did not include internal events, nor analyse mood longitudinally. The studies cited all employed a between group methodology rather than a longitudinal idiographic one, thereby obscuring any meaning that individuals may derive from the intersection of life events, behaviour, social support and mood. This represents a serious shortcoming in the research to date. The study by Vachon, Doron, Thomas-Ollivier, and Fortes-Bourbousson (2012) represents a significant improvement in self-esteem research, with respect to its prospective and electronically measured methodology. The authors examined the evolution of self-esteem in one bipolar patient twice daily on an analogue scale over three months, accompanied by audio recordings. As Knowles et al. (2007) and Bentall et al. (2011) had found, self-esteem was low and quite unstable, indicting two processes: the first implied state dependence and the second suggested a trait-like nature. The personal narrative findings of this study were not reported.

What then do we know conceptually about the entity which binds life events, cognition, coping, social support and mood? Firstly, it is at the very least a partly conscious entity which has its locus in CNS functioning, otherwise it would not be linked to cognition and feeling state. It is certainly central to experience and it covaries bivalently with positive and negative affect. It entails an evaluative process in relation to the self and provides a mirror to social prestige (social support) and is a reference point for life events. It has been shown by empirical research to be closely related to illness morbidity. Lastly, sociometer theory (Leary, 2005a) stipulates that there is an affective process, following the evaluation of relational value, which drives behaviours intended to increase prestige. Paying heed to the quotation by Bentall et al. (2011) it is therefore concluded that this locus is likely to be the neural manifestation of self-esteem.

Conclusion

Bipolarity arises in the nexus between the biological template, life events and the social milieu. Research has focused heavily on biological factors, all too often overlooking ultimate contributions to the disorder. This chapter has reviewed the
social and life event underpinnings of bipolarity and located bipolar disorder as a social subgroup with its needs and vulnerabilities, reliant on the flow of Cobb's social information. Though there is a clear relationship between social support, expressed emotion, life events, subsyndromal symptoms and the course of mood disorders, it is difficult to establish the causal direction. Low social support may be both a cause and consequence of mood syndromes, or mood may act as a perceptual filter to the world. Similarly, individuals with mood disorders may respond to life events and generate their own stressors. It seems likely that the micro life events of everyday life, accruing over time, act as an additional significant stressor to bipolar episodes. At this level of analysis, cognitive and behavioural dispositions become more relevant. These variables all constitute an interacting web of factors which produce a vortex to mood relapse – putatively through neural self-esteem.

This analysis has found reasonable support for the macro factors which are operant in the generation of bipolar mood episodes. The literature, however, is relatively agnostic as to what stressors mean to individuals, there being significant shortcomings in longitudinal relapse data. Future research will focus on a unification of both inner (self) and outer factors, linking bipolar disorder to prestige and its variants.
"It is the simple fact that I live in the facial expressions of the other, as I feel him living in mine." (Merleau-Ponty, 1964, p. 146)
Chapter 3: Prestige

“A story is told, that a certain person, incurably affected, fell in love with a girl; and when the physicians could bring him no relief, love cured him. But I think that he was originally in love, and that he was dejected and spiritless from being unsuccessful with the girl, and appeared to the common people to be melancholic. He then did not know that it was love; but when he imparted the love to the girl, he ceased from his dejection, and dispelled his passion and sorrow; and with joy he awoke from his lowness of spirits, and he became restored to understanding, love being his physician.” (Areteaus, 1972)

Introduction

In the quotation from the first century physician Areteaus, the subject’s recovery was determined by seeing himself loved in the eyes of the girl; reciprocated affection predicating love and prestige. Chapters 1 and 2 have hinted at the dynamic between self-esteem and prestige. In order to advance this logic, this chapter turns to an examination of the phenomenological and evolutionary basis of prestige. The section serves as a bridge between sociological and clinical aspects of the prestige construct; thereby the model presented may be adapted for later usage in a bipolar context.

Prestige will be defined and contrasted to dominance; as both phenomena lead to social status. The dual aspects of honour (Cooley, 1922) will be examined in modern day terms and self-esteem will be more closely allied to affect. A basic approach–withdrawal affective system will be presented to subserve the function of prestige and self-esteem; these are part of a reciprocally interacting process, in which the tokens of prestige link self to society. We are taken back two million years to the putative origins of prestige systems with Steven Mithen (2005) and his Hmmmomm communication. An evolutionary model is presented to account for dynamic fluxes in self-esteem and prestige. The chapter will conclude with the notion that prestige is a vital and far reaching aspect of societal function and an eminent locus for illness.
The Prestige Construct

“One's honor, as he feels it, and his honor in the sense of honorable repute, as he conceives it to exist in the minds of others whose opinion he cares for, are two aspects of the same thing.” (Cooley, 1922, pp. 207-208)

Modernly recognised prestige was likely to have arisen in Africa at some point in the past two million years with the appearance of Homo erectus, which had developed technologies for food procurement, coinciding with a hunter gatherer social structure. These societies may have been similar to modern day hunter gatherers, where socialisation and relaxation form a large part of communal life (Lomas, 2009). Dunbar (2003) argues that such social factors led to progressive brain size evolution and that social cognition was dependent on the phenomenon of Theory of Mind (ToM) – allowing individuals to appreciate the minds of others. This fostered the building of self-esteem, derived from the prestige accorded by conspecifics. Further assisted by language, social cognition facilitated coalitions based on grooming partnerships (Dunbar, 2003).

While dominance infers the ability to inflict costs on others, prestige relates to the ability to confer benefits. Dominance has an even deeper phylogeny than prestige, being seen in insects, crustaceans, reptiles and mammals (Buss, 2012). Dominance competitions lead to a dominance hierarchy, which is a property of the group, rather than the individual (Buss, 2012). Status striving is a candidate for the top universal human motive (Buss, 2012). A distinction was made (von Rueden, Gurven, & Kaplan, 2010) between “men who are more likely to win a dyadic physical confrontation” (dominant men) and “men with more community-wide influence” (prestigious men) (p. 2223) and both are seen as pathways to status, leading to greater reproductive fitness (von Rueden et al., 2010). While dominance is positively correlated with testosterone levels, prestige is not (Johnson, Burk, & Kirkpatrick, 2007).

Eminent authors such as Barkow et al. (1975) and Buss (2012) have conflated the notions of prestige and dominance and Barkow et al. (1975, p. 570) boldly declares that "...human social dominance is prestige and self-esteem and at the same time the outcome of agonistic encounters, power-striving and politicking, 'toughness',

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charisma, and many other things”. Barkow’s comments notwithstanding, dominance and prestige are likely phylogenetically and phenomenologically distinct psychosocial processes as reflected in the work of von Rueden et al. (2010).

Cooley (1922), quoted early last century, captures the dual aspects of "honour". Using a precocious Theory of Mind concept, he eloquently highlights the subjective and introjective notions which facilitate our self-value. The American Heritage dictionary defines prestige as "the level of respect at which one is regarded by others; standing" (American-Heritage-Dictionaries-Editors, 2011, p. 1). In his seminal paper “Prestige and Culture: A Biosocial Interpretation”, Barkow et al. (1975) argue for the transformative role of natural selection in shaping the striving for high social rank into a need to maintain self-esteem. Barkow et al. (1975) argue that self-evaluation becomes a covert cognitive process, creating new possibilities for the distortion of perception and invoking prestige-giving membership groups. Introjection of other individuals, living or deceased is a means of maintaining prestige. Barkow sees the self-esteem goal as a biological constant, while prestige strategies are products of sociocultural evolution, transmitted through socialisation. Knudson (Barkow et al., 1975) questions Barkow and colleagues' conceptualisation of self-esteem as a ranking mechanism, citing examples where the individual maintains self-esteem without placing himself higher than others. The notion of self-esteem as intentional object is to be contrasted with the sociometer model of Leary et al. (1995) – in which relational value rather than self-esteem per se – is the ultimate motivational locus.

In the film *The Prestige* two late 19th century magicians are engaged in a battle of one-upmanship with fatal consequences. The three stage process of a magic trick is explained; in *the pledge* something ordinary is displayed and then in *the turn* it magically disappears. Finally, in the part of the magic trick called *the prestige* the object reappears, the audience is amazed and the trick is complete. Social prestige, just like the prestige finale of the magician, involves a sort of magic which unites actor and audience. In the most important part, the prestige, something – the object (the person) – appears magically within the group through the power of social investment.
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

Prestige is the investment of the group in the individual (Price et al., 2007) and leads to social inclusion and status. It may be conceptualised as a topographic surface, with mobile actors spread around the landscape. The surface encompasses peaks and troughs; the peaks representing spaces of high prestige and affect and the troughs spaces of low prestige and affect. The actors are attracted to individuals on the peaks, to enhance their self-esteem and aggregate with socially desirable group members. They communicate via affective display and self-enhancing narratives. Actors in the troughs temporarily lack “magnetism” and console themselves that this is a state of misery from which they may escape through their capacity for bipolar leadership. Some unipolar individuals, who don’t frequent the peaks, may not be as motivated or skilled in exiting the troughs. The landscape represents a dynamic social space, held together by the interaction of prestige, affect, and motivation for prestige leadership. This will be discussed later and results drawn to support these conceptualisations.

**Self-Esteem**

“People cannot function in an interpersonal life without paying attention to how they are perceived and evaluated by others, particularly with regard to their relational value in others eyes.” (Leary, 2005a, p. 97)

The term “self-esteem” is a problematic notion which entails the global evaluation of the self, through which one’s complex self-evaluations are collapsed into one term. The iconic Rosenberg (1965) Self-esteem Scale, for instance, utilises ten statements on a four point Likert scale. These statements include comparisons with others, self-descriptions and global evaluations of the self, with affective, cognitive and behavioural aspects. Because of the complexity of the concepts from which it is derived, traditional self-esteem is a concatenated and poorly defined construct. Affective self-esteem, on the other hand, may be a self-referring term, denoting a favourable regard of the unitary self with its states of positive, neutral and negative valence. What then of the distinction between pure self-esteem and hedonic affect? Hedonic emotion necessarily implies two intentional objects and three mental states: "I feel [good],[neutral],[bad] about myself or a part of myself" and implicitly "I feel [good],[neutral],[bad] about the world or part of the world". Experience shows that the two covary strongly. There can be no other referents, though the constitution of
the world and the self may be selective. Feeling [good], [neutral], [bad] about oneself (self-esteem) is therefore likely inseparable from hedonic or anhedonic affect.

With relation to the relationship between subjective self-esteem and objective prestige, it is argued that there are two intentional objects to feeling [good], [neutral], [bad] – one of which is perceived reality. Why do people feel good about the human world? This state accords with the world feeling [good], [neutral], [bad] about them. It is not common to feel good about another person when that person derogates us. Similarly, valuing from another person is generally associated with a positive affect toward that person. Hence a person-to-world affective estimation implies a reciprocal equal valence imaginal world-to-self estimation. Therefore, when self-esteem is high, world estimation is generally high and perceived prestige is reciprocally high also. It is worth noting that prestige entails both a communal (system) reality and also an internalised state (self-esteem), which is an introject.

Referring to Figure 3, we see that prestige and self-esteem are subject to social approach and withdrawal; withdrawal being exemplified when a respected community entity disparages us and we react with a lowered self-esteem/affect. In this context, the prestige gap metaphorically increases in width. States of approach, on the other hand, are associated with proximity between self and objects; between elevated self-esteem and prestige. It is notable that prestige does not operate as an intangible community parameter, but rather through prestige objects, such as narrative, play, interpersonal feelings and explicit social goal behaviours. These act as tokens which intervene to drive self-esteem and prestige. In this way self-esteem acts on the world, and prestige acts on the self, in a process of externalisation and introjection. These events result in subjective positive hedonic affect or sorrow, depending on the context. It is possible to describe a four part iterative cycle of prestige in which the self confers benefit on the world (display) in the context of the world paying notice to the self. In turn, the world invests in the self (esteem) and the self accordingly invests in itself (self-esteem). Thereby, the self engages with the world through cathexis and display, building on the feedback relationship between all four elements. Described broadly in this way, prestige is about both interpersonal investment and the
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

introjective response to that investment. I will further argue that this introjection of social processes has a neurobiological basis – the *organ* of self-esteem.

![Figure 3. Prestige-self-esteem system with operators indicated](image)

Table 1 indicates the prestige objects, self-esteem and prestige state which are coincident with a number of prestige contexts. The most common of these contexts is interpersonal exchange, notable for language, gesture, vocal prosodic signalling, and energisation, which generate affective fluxes in the participants, elevating self-esteem and affection. In its broadest sense, play is energetic affective engagement between actor and audience. In fact, the PLAY and SEEKING systems as described by Panksepp (1998), may be the evolutionary progenitors of positive affect, with creative objects such as song and dance generating self-esteem and prestige. Prestige may, in addition, be generated by imaginal second or third order ToM. Community objects may be represented solely in the affective mind.
Table 1  Contexts and Outcomes of Prestige Behaviour

<table>
<thead>
<tr>
<th>Context</th>
<th>Prestige object(s)</th>
<th>Self-esteem/prestige “affect”</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social exchange (social engagement)</td>
<td>Reciprocal affective elevation, Narrative</td>
<td>Hedonia/Liking</td>
</tr>
<tr>
<td>Play (social incorporation)</td>
<td>Reciprocal affective elevation and energisation</td>
<td>Hedonia/ Acceptance</td>
</tr>
<tr>
<td>Affection</td>
<td>Act of kindness</td>
<td>Joy/Acceptance-love</td>
</tr>
<tr>
<td>Societal problem</td>
<td>Social goal solution</td>
<td>Hedonia/Acclaim</td>
</tr>
<tr>
<td>Social cohesion need</td>
<td>Creative process$^2$</td>
<td>Hedonia-joy/Acclaim</td>
</tr>
</tbody>
</table>

Negative prestige is fostered by invalidating social events which confer sorrow, which is experienced as an absence or void, while secondary emotions such as anger, sadness, and apprehension may also arise. Sorrow is internalised prestige withdrawal and experienced as anhedonic affect. Negative prestige may be generated by imaginal ToM actions, as seen in some individuals with a neurotic disposition. Both positive and negative prestige, described in this way, represent the action of dynamic variables on the subject’s mental state. Traditional accounts of prestige have, on the other hand, focused on static or structural aspects more akin to social status. Particular persons may possess greater prestige holding and allocating power.

On the cognitive level, prestige is a complex process, which in its essence involves the interplay between actor and audience, influenced by the intervening prestige object. From the perspective of a person in an audience position, he or she is thinking "I think that we believe that s/he is worthy of our valuing". This is a third order Theory of Mind process. For the actor, he or she is required to think "I think that they suppose that I am worthy of their value because of what I do or how I think about them". The process may be fourth order ToM, with the actor thinking to him or

$^2$ Henrich and Gil-White emphasise information transfer as the primary aetiologic agent in the genesis of prestige. Information copiers "ingratiate themselves with their chosen models" to create distributions of deference (Henrich & Gil-White, 2001). It is clear that this is but one factor.
herself "I believe that they suppose that I am thinking that they value my behaviour highly". Such orders of reflexive thinking represent a remarkable, though poorly understood, human characteristic (Leary, 2005a). Such systems are biased towards false positives, in the sense of the anxiety as smoke detector analogy of Nesse and Williams (1994), because "a single failure to realise that one's behaviour has angered or alienated other people may be far worse than many false alarms in which a person needlessly worries that he or she has been evaluated negatively." (Leary, 2005a, p. 89).

The Evolution of Prestige

“The display behaviour…is a form of social solicitation, as it leads on to forms of associative behaviour in which there is a continuing interaction between individuals, such as grooming, play, sexual or mothering behaviour with the displayer…Play which involves grappling the other individuals can also be readily transferred to ropes…or for poking…but suggesting an affinity with pleasant human feelings…It is further suggested that an individual’s social attention in the hedonic mode is polydiadic: i.e. at any moment it relates one individual to another, but frequent changes of attention to other individuals are possible…” Chance and Jolly (1970, p. 176)

Before the advent of social cooperation, rank was the central axis of interpersonal and group relationships and a proxy for reproductive success, food and power (Stevens & Price, 2000). Ritual agonistic behaviour, which signified power and requisite fear, led to hierarchical vertical relationships, later maintained in primate ancestors by the agonic mode, where threat without agonism maintained order (Chance & Jolly, 1970).

Whilst rank remains a given in society, over the last ten million years, a new mode of social organisation has emerged – the hedonic mode (Chance & Jolly, 1970). The hedonic mode describes horizontal relationships between chimpanzees (and humans), where individuals have the freedom to form and dissolve groups under situational leaders. These networks are held together by attachment, touch and reassurance, while behavioural and affective display may be interpersonal or for the benefit of the group, attracting others to oneself. Drama and play are evident as the group grooms, explores, celebrates and cavorts, forming the basis for social
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

generativity. Social prestige arose within human communities and was personally internalized to self-esteem – a psychic representation of social value. Gilbert (1997) has termed its social equivalent social attention holding power (SAHP). Esteemed individuals have the power to influence and be judged “fit”, not only to lead, but to reproduce. Sexual selection favours those who can ‘play’, in its broadest sense.

In *The Singing Neanderthals*, Mithen (2005) speculates on the forms of social interchange used by early hominins and argues that communication was then not symbolic or compositional, but holistic. Following the example of apes and monkeys, communication was manipulative rather than referential and Michael Corballis (1999) has further argued that gesture is likely to have constituted a great deal of early communication. Mithen (2005) terms this *multi-modality* and argues further, that as geladas and gibbons communicate in musical forms, and as infant directed speech may be a form of proto-language, early communication systems were "musical". Building on Dunbar (2003), Mithen (2005) opines that social grooming was likely to have been undertaken with musical vocalisations. Given the human propensity to imitate (Hauser, 1999), Mithen adds that communication forms were mimetic, characterised by intentional iconic gestures, body movements or sounds. He has named this composite form of communication *Hmmm* to encapsulate its holistic, multi-modal, musical, manipulative and mimetic character. Mithen (2005) argues that the display of such behaviour is likely to have aided sexual selection. He links his communication to group-related forms of generativity and personal elevation, providing a rich context for the generation of prestige because of its directed gesture, affective energisation, musicality, dramatisation and group focus.

Selection pressures to communicate complex emotions resulted in more advanced neural development and with the advent of Homo sapiens, language and representation became symbolic. Trade and exchange led to greater pressures to transfer large amounts of information and *Hmmm* became attenuated, being replaced by formal language. Mithen (2005) argues that Infant Directed Speech (IDS) and our musical capacities are remnants of *Hmmm*. Logically, if IDS is an exaggeration of the melodic and rhythmic aspects of normal spoken language (Mithen, 2005), it is therefore a subset of affective and linguistic communication.
generally. As such, it is proposed that a more modern communicative form – linked inherently to our affective systems – has followed Hmmmmm.

Drawing on work with primates, Dunbar (2003) suggests that level 3 Theory of Mind intentionality – required for simple prestige operations – appeared with Homo erectus, while level 4 intentionality was breached with the arrival of anatomically modern humans. As suggested by Mithen (2005), Homo erectus communicated via Hmmmmm – and lacked language (Dunbar, 2003). Forms of prestige were therefore likely to have been comparatively simple. Complex linguistically-enriched communal forms would therefore only come into place with the advent of Homo sapiens with its level 4 intentionality.

Acheulian handaxes appeared in Africa around 1.5 million years ago. They were implements used in butchery, yet they were often plentifully made, both large and beautiful, in a manner which seems to exceed their function. Some researchers have suggested they had social significance – as male emblems of fitness to attract mates (Stringer & Andrews, 2005). Thus these implements may have been the first prestige objects, representing a second level intentionality.

Between 50,000 and 40,000 years ago humankind underwent an unprecedented transition in social and mental functioning – the Upper Palaeolithic Revolution. Artwork flourished in communities, which painted and carved representations of themselves and their natural world; they were both audience and actor. The first bodily ornaments, approximately 50,000 years of age, indicate both a cognisance of the perception of others and the individual's attempts to influence them through ornamentation (Leary & Buttermore, as cited in Leary, 2005a). Statuettes like the many Venuses, suggest that the perception of the other may be transformed by mental workings within the self. Burial sites, such as that of the Grotte des Enfants in Italy, reveal children adorned by hundreds of seashells and pierced animal teeth (Stringer & Andrews, 2005). This indicates a practice of human interpersonal valuing which transcended even death. Guthrie (2005) argues that art is a uniquely related to play and the product of a social species having the flexibility for daydream and fantasy. Certainly, Paleolithic art was likely to have been combined with social
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ceremony (Stringer & Andrews, 2005); play becoming a platform for prestige as mankind witnessed the first full *exteriorisation of the interior*.

Stevens and Price (2000) have argued:

“In hedonic\(^3\) competition, two rivals display, not to each other (as in agonistic competition) but to the group as a whole, presenting their attractive qualities in an effort to elicit group approbation. The group members’ response (either approbation or disapprobation) differentially raises or lowers the self-esteem of those who are displaying, and at the same time confers on them differential prestige...They assume leadership roles and have access to more resources than their less successful competitors. In an environment similar to the Environment of Evolutionary Adaptedness, they tend...to have more wives, sire more children and their children are more likely to survive. Their fitness therefore increases.” (pp. 159-160).

This passage documents the fitness advantages of positive prestige. Baumeister and Leary (1995) have further explored the proximate values of belonging; a state which generates prestige.

**Affective Systems in Prestige**

"There is something in the bonds between humans that is akin to play: smiles and giggles, expanded awareness, delight in one another, and the ability to enjoy imagining another's enjoyment – in short, the ability to love." (Guthrie, 2005, p. 389)

Prestige is a social process whereby self-esteem is reciprocally generated by means of emotional and other systems for the purpose of social incorporation. Panksepp and Harro (2004) have described basic mammalian emotional circuits which predicate the behaviours described in the hedonic, and other, modes. Along with the SEEKING system, LUST, and the homeostatic systems, Panksepp (1998) has titled emotional operating systems CARE, PANIC, RAGE, FEAR and PLAY. These have been linked to neural functioning in lower mammals, which has been extrapolated to primates. Pankseppian PLAY is homologous with human proclivities for play, socialisation and enjoyment (Panksepp & Biven, 2012).

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\(^3\) Hedonic competition is equivalent to prestige competition
Shaver, Wu, and Schwartz (1992) undertook a crosscultural study to examine how people and cultures organise emotions into families. Both American and Chinese cohorts identified positive and negative emotions, agreeing on anger, sadness, fear and happiness. Cluster analysis subdivided happiness into liking, joy and exuberant arousal, suggesting a dimensional character. Happiness recurs as principal emotion in numerous typologies and constitutes an emotion family with a shared countenance, but specific vocal signaling (Ekman, 2007). The homologous PLAY cluster – in the system developed here – is an affect family equivalent to the constructs of positive emotions, the happiness family and the dimension of pleasure. *Enjoyment* and *amusement* are commonly encountered and contiguous emotions. *Excitement* is of greater intensity and, with its superordinate emotions, connotes greater arousal and motor involvement suggestive of concurrent SEEKING activity.

In their earlier analysis Shaver et al. (1992) identified sadness as one of the common emotion families where it was represented by disheartenment, distress, sorrow and loneliness. SORROW (differing from sadness) is an emotion family designated as a state of diminished emotional response, with a notable lack of PLAY and SEEKING emotions. It is characterised by social withdrawal along with scorn of the self and disengagement from the world. It is notable for feelings of hopelessness and disadvantage and has a characteristic expression. While PLAY intends us towards social objects, SORROW draws us away from them. Thus the PLAY-SORROW dichotomy is an example an antagonistic approach and withdrawal system.

The facial externalisation of the affective state of enjoyment is the smile. Keltner (2009) notes that: "in evolution's toolbox of adaptations that promote cooperation, the smile is perhaps the most potent tool... The smile smooths the rough edges of our social life, creating a medium of benevolent exchange." (p. 99). He further opines that "smiles... are the first incentives towards which young children

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4 For the purpose of this thesis the following conventions apply; designated emotions are indicated in italics – sorrow; affect programs capitalised – SORROW. Strategic Motivational Affect Systems are indicated: HEDONIC ENERGETIC SOCIAL ENGAGEMENT (HSE) etc.

5 Sadness is taken to be an experientially "full" emotion which intends the subject towards attachment. Sorrow, on the other hand, is a "void" state, which is in effect the absence of emotion. It intends the subject towards social withdrawal.
move, and that parents hungrily seek.” (Keltner, 2009, p. 111). Mehu, Grammer, and Dunbar (2007) find that the Duchenne smile may be "an important signal for the formation and maintenance of cooperative relationships" (p. 415). Waller and Dunbar (2005) have argued that both the silent bared teeth display and the relaxed open mouth display of the chimpanzee both increase affinitive behaviour, suggesting a common ultimate function – social bonding. The two may have converged in humans from different motivational complexes. de Waal (2009) notes the inherent sociality of humans, based on a shared need for security. This sociality is seen in the instinctive processes of interpersonal synchrony, mood contagion, dance, music, mimicry and empathy. He equates this with social connection and an advanced herd instinct.

Affective signals are important for social cohesion, which is involuntary, adaptive and species-wide and may therefore be termed instinctive. Panksepp (1998) has described SEEKING as the neural system which gives us the impulse to become actively involved with the world. The feeling tone involved is one of psychic energisation and the emotions are of intense interest, engaged curiosity and eager anticipation. Solms (2011, March) has said that the SEEKING system is the core engine of the mammalian mind, responsible for desire and the hunger for pleasurable experiences. SEEKING is involved in both primary process PLAY social neural circuits and play-fighting (Panksepp & Biven, 2012). Rats involved in play make high frequency chirps – a form of ancestral laughter – linked to the dopaminergic rewards of the SEEKING system. Though PLAY and SEEKING appear (structurally) distinct, both systems are interactive during play (Panksepp & Biven, 2012). Therefore, the two are interwoven in the process of play and social engagement and it may be possible to think of them as a functional unit from this perspective. Indeed, Panksepp has based his research on early mammals (rats) and it may be that with neomammalian evolution that the circuits have become more functionally united. For the purposes of this thesis, I will make an assumption of functional unity in these systems for the purpose of hedonic social engagement.

I term the Homo synergism of PLAY and SEEKING Hedonic Energetic Social Engagement (HESE), which, alongside attachment (CARE), may be the ultimate origin of social engagement. HESE is posited as a functional Strategic
Motivational Affect System\(^6\) (SMAS) which intends the individual toward play and group engagement. It operates through the affects of *enjoyment, amusement*, and *excitement* and it links individuals through gesture, narrative and “performing artworks”, generating prestige and amity. Freud (1930) has termed a related notion *Eros*, emphasising communal life-giving characteristics. Prestige is the product of HESE signals, which generate inclusion cognitions and affect in the dyadic and polydiadic partner(s). The expression of HESE generates a positive feedback loop within the dyad or group, where positive affect generates positive affect, internalised to self-esteem. Self-esteem arises as we see ourselves being seen affirmatively in the eyes of others. Prestige is generated in conspecifics; the group is seeing the individual affirmatively. Hedonic affect, as well as self-esteem, is driven positively or negatively by this process. HESE may be construed by some as a social subsystem of the behavioural activation system (BAS), where cues of reward promote increases in positive affect, attention, energy and goal pursuit (Johnson, 2005b).

**The Prestige Model**

“If no one turned round when we entered, answered when we spoke, or minded what we did, but if every person we met “cut us dead,” and acted as if we were nonexisting things, a kind of rage and impotent despair would ere long well up in us, from which the cruelest bodily tortures would be a relief; for these would make us feel that, however bad might be our plight, we had not sunk to such a depth as to be unworthy of attention at all.” (James, 1890, pp. 293-294)

Prestige may be seen as a commodity which is traded within the social economy, binding hunter gatherer societies, but also setting the conditions for social exclusion. The livelihood of band members rests on sharing and conformity, yet burdensome individuals may impede group function. Collective ostracism, ridicule, gossip and shunning (Lomas, 2009) are important to eliminate these individuals and maintain cohesiveness (Williams, 2007a). Individuals unable to generate sufficient prestige are located in a "marginal zone", where their status becomes dubious. In ancestral environments, failure to remain tethered to the tribe led to a precarious state, as resources were bound to the nomadic group. Not only would the individual have no

\(^6\) The Strategic Motivational Affect System (SMAS) concept is described in chapter 4 and adopts terms from Lang, Bradley, and Cuthbert (1992)
access to sustenance, but s/he would lose the ability to reproduce. There were no supermarkets, support groups and charities to ensure life outside of the band. Homicide of the ostracised may have been a risk, as is sometimes seen in contemporary hunter gatherers (Woodburn, as cited in Lomas, 2009). Unless able to join another tribe, the individual’s genetic line would end forthwith. Therefore, prestige was a matter of life and death in the Environment of Evolutionary Adaptedness.

Ostracism threatens at least four fundamental needs: belongingness (prestige), maintenance of self-esteem (the internalisation of prestige), perception of personal control (sense of agency) and recognition of meaningful existence (in the group – prestige) (Williams, 2007a). Williams (2007a) states that: “organisms that were especially good at detecting or anticipating ostracism were probably most likely to be able to do something about it that might prevent the inevitable loss of group membership, protection, and reproductive opportunities.” (p. 429). He further argues that “an ostracism-detection system, therefore, probably coevolved with the widespread use of ostracism. Such a detection system was probably selectively biased to detect any possibility of ostracism, thus leading to an error management system that favored a bias for false alarms over misses.” (p. 429). Williams (2007a) further divides the responses to ostracism into (a) behaviours to re-engage, (b) antisocial and aggressive behaviours (equivalent to rank behaviours), (c) an affectless/stunned state and (d) attempts at flight. Here, we will focus on (a) and (c).

In response to exclusion, humans’ first response is typically distress, which draws attention to the prevailing social circumstance to assess threat and to marshal resources. Following appraisal, a reflective stage may ensue (Williams, 2007a) with parallel compensatory behaviours. Though the research is ambiguous, factors such as self-esteem, loneliness, rejection-sensitivity, narcissism and attachment style may moderate the response to ostracism (Williams, 2007a). As indicated, responses may include pro-social behaviour, which serves to re-engage the individual with his or her community; females, in particular, are likely to exert effort towards such goals (Williams, 2007a). This effort may include affective responsiveness to social cues. Clearly, individuals in an ostracised situation are thinking about what other people are
thinking about them, thus exercising ToM. Social narrative may ensue and the person may engage in generative and creative actions. Twenge, Ciarocco, Baumeister, DeWall, and Bartels (2007) have documented instances of affective restriction following contexts of imaginal social isolation. Allen and Badcock (2003) have suggested that “depressed mood evolved to facilitate a risk-aversive approach to social interaction in situations in which individuals were typically at risk of exclusion from social contexts (i.e. dyadic relationships or groups) that were vital to dealing with adaptive social reproductive challenges” (p. 887). This is consistent with a stealth model of depressed mood, in which individuals socially withdraw with a lowered mood to avoid attention and ostracism, thereby "flying under the radar" (Le Bas, Castle, Newton, & O'Loughlin, 2013).

Some of the tokens which attract prestige are personal effectiveness, directedness, attractiveness and recognition from peers. A person who is perceived as honest is likely to garner prestige, as is someone who displays knowledge, success and skill. Positive affect and gesture increase social interaction and a more energised person is likely to receive positive regard. There are social strategies, such as assertion, which an individual may pursue to increase his or her prestige. Capturing social attention to personal assets may be important, along with inventiveness and goal pursuit. In states of threatened exclusion, individuals may turn to kin, signaling need through distress behaviours to facilitate proximity, support and social re-entry. In a response to relational threat, the sociometer of Leary et al. (1995) has the capacity to launch a wide range of adaptive behaviours.

**Conclusion**

This chapter has served as an examination of contemporary and ancient aspects of prestige, arising in hominins over the past two million years. Increasingly complex Theory of Mind processes led to the ability of individuals to think inside the minds of others, leading to reflexive changes in the way we were able to think and "esteem" about ourselves. In spite of these benefits, however, hominins lived in a state of incipient marginality, a reflection of which we experience today. Prestige imposes an ongoing pressure to be included, whilst ostracism pushes us away from the group.
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Processes of social engagement became crucial to our species and these phenotypes were selected on the basis of prestige fitness.

Social practices such as honour codified prestige, leading to not only a socially focused self-talk, but a society which valued the self and its individual esteem. Hominins became anchored within a homoeostatic process of social approach and withdrawal, predicated by prestige objects such as narrative and social action. Affective systems were inextricably linked to self-esteem – the internal homologue of prestige. Over evolutionary time, we have seen a transition from Mithen’s Hmmm social communication system, which utilised affect, to a system of affect which intends the communication of prestige (HESE). A model has been proposed which links prestige to adaptive behaviours. Authors such as Williams (2007a) have stressed that ostracism detection systems must be finely tuned and thereby prone to false alarm, resulting in the risk of excessive prestige-seeking behaviours. It is envisaged that in some contexts, this propensity to act on ostracism risk may become a self-perpetuating byproduct of evolution. This will be the focus of the next chapter, as we examine affective pathogenesis.
Chapter 4: Affective Pathogenesis

Introduction

“The question as to why discrete emotions are interrelated and frequently occur in dynamically interacting patterns may be equally or more interesting than the question of the structure of emotions.” (Izard, 2007, p. 269)

The previous chapter introduced the notion of prestige and emphasised its relevance to social and personal functioning, while relating it to the subjective and iterative notion of self-esteem. An account has been made of the evolution of these constructs, linking them to affective systems which mediate personal and social homoeostasis. Notions of emotion and motivation have long been considered separately, whilst paying little regard to object relations and the basic tendency toward approach and withdrawal. A comprehensive model for a strategic motivational affect system will be introduced and related to Panksepp's primary emotional systems, therein drawing together Thayer's tense and energetic arousal concepts within a broader account of arousal and basic emotion. Though this model is ambitious in its scope, an attempt is made to limit its derivation to first principles, derived from what is known of emotion, prestige, and affective illness. A tentative phylogeny of arousal and emotion is presented and hedonic energetic social engagement is considered from a triune perspective. This will lead to the proposal that affective illness is a product of evolution, being constituted by pathological excitation and inhibition within HESE.

The Evolution of Emotion

"The theoretical view shared among the investigators is that expressed emotions are founded on motivational circuits in the brain that developed early in evolutionary history to ensure the survival of individuals and their progeny. These circuits react to appetitive and aversive environmental and memorial cues, mediating appetitive and defensive reflexes that tune sensory systems and mobilize the organism for action and underly negative and positive affects". (Lang & Bradley, 2010, p. 437)

Like the Hindu goddess Mahadevi, emotion is both beautiful and terrible, delivering bounty and pain from deep evolutionary time. Emotion moves minds and
bodies and shapes the social world, making clear what is of import to our survival. Emotion arises in the interstice between subject and object as a valent subjective state, expressed through the autonomic, endocrine, and musculoskeletal systems. It invests experience and mediates social interactions. Emotion inspires creativity and social evolution, fuelling narratives, which it bends accordingly. Yet, how did emotion evolve? Does it exist as discreet states, arising in subcortical circuits, or are emotions “point-clouds in an N-dimensional space” (Nesse & Ellsworth, 2009, p. 132)? What does emotion have to do with prestige?

Illuminatory progress has been wrought by scientists of the nineteenth and twentieth centuries. In The Expression of the Emotions in Man and Animals Darwin (1872) demonstrated that human and animal emotions were homologous in their expression. McDougall (1919) elucidated the relationship between emotion and motivation, which he unified under the rubric of instinct. He conceived a sensory pole linked through an affective core to a motor extremity. Ekman and Friesen (1989) designated six cross-cultural basic emotions: happiness, sadness, fear, anger, surprise and disgust. In so doing, they ventured that emotions were biological kinds and not social constructions and Ekman (1999) continued to advocate the view that emotions were adaptations, especially important to interpersonal relationships. Contrary to the James-Lange theory, and in line with Panksepp (1998), he contended that emotions involve specific CNS activity (Ekman, 1999). A comprehensive taxonomy of facial expression in emotion was conceived (Ekman & Friesen, 1978).

Nesse and Ellsworth (2009) have shed light on the evolutionary rationale for emotions: “Emotions are special modes of operation shaped by natural selection. They adjust multiple response parameters in ways that have increased fitness in adaptively challenging situations that recurred over the course of evolution.” (p. 129). They see evolution not as an alternative emotion theory, but “the common foundation for all.....defining what emotions are in terms of how they came to exist.” (Nesse & Ellsworth, 2009, p. 129). Emotion is but one of many adaptations, crafted by selfish genes to facilitate the capacity of survival machines (organisms) to carry forward their genes (Dawkins, 1976).
The turn of the 19th century saw a flurry of psychological thinking and a prominence of the concept of emotion and motivation. Whilst believing that instincts themselves were innate, James (1890) saw them as part of a total action, which could be modified by learning. William McDougall (1915) extended Wundt’s association of instinct and emotion, arguing that instincts were centrally affective – a hormic7 striving toward a particular outcome. For the contemporaries McDougall and Freud, instinct “occupied the core of virtually all behaviour, energizing it and guiding it to certain objects or goals” (Herrnstein, 1998, pp. 86-87). In the modern era, Robert Plutchik (2001) has described the iteration between stimulus, cognition, feeling state and arousal with action impulse and behaviour – emotions being more than just linear processes. Energy and arousal have been central to models of psychic functioning since before the time of Freud (Thornton, 2010). The combination of valence and arousal have been termed a psychological primitive, common to all psychological states (Feldman Barrett & Bliss-Moreau, 2009). Thayer (1989) on the other hand, has divided arousal into the systems of tense arousal and energetic arousal.

Humans have an innate drive to instigate and maintain relationships (Klee, 2010) – this dynamic creating a relational space between subject and object. This space is in turn replete with elements, which are here termed intervening objects. Emotion “falls” between subject and object, linking the two for a moment of time, this connection indicating a motive of approach or withdrawal. Affect products such as facial expression, vocal prosody, and gesture are technologies employed by selfish genes to achieve ends pertinent to survival. Philosophers have long discussed intentionality – the cognitive and objective “aboutness” of emotions (Solomon, 2008). In Figure 4 we see a SMAS, encompassing arousal and its functions. Four arousal types are conceived; tense, sexual, agapic8 and hedonic-energetic. Each has approach and withdrawal orientations leading to contrasting emotions, which serve different motivational imperatives. All are anchored around subject-object relations, which have served to enhance fitness.

7 Hormic: from the Greek word for urge
8 Agape is an ancient Greek term, used to describe love of kin, as opposed to erotic love
Lang et al. (1992) coined the notion of strategic motivational affect, though in subsequent work they appear to advocate for only two distinct systems; one for approach and one for withdrawal (Lang & Bradley, 2010). They are inclined to the circumplex model of emotion as described by Russell (1980), where emotion space is described by the dimensions of pleasantness (valence) and arousal (activation), as supported in a number of studies (Larsen & Diener, 1992).

Contrary to the dimensional view, Panksepp (2010) has emphasised that "primary-process emotional feelings are organized within primitive subcortical regions of the brain that are anatomically, neurochemically, and functionally homologous in all mammals that have been studied." (p. 383). He also notes that neurotransmitters and neuropeptides regulate emotion intensity, creating the possibility that a number of distinct arousal types in subcortical regions may exist (Panksepp, 2010). Panksepp's
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notion of distinct neural circuits underlying SEEKING, RAGE, FEAR, LUST, CARE, GRIEF and PLAY is in accord with the notion here of SMAS’s, albeit without bivalent notions of approach and withdrawal. Translating Panksepp's terms we may represent SEEKING as energetic arousal, RAGE and FEAR as part of tense arousal, LUST as sexual arousal, CARE and GRIEF as a constituent of agapic arousal and PLAY as hedonic arousal, which is taken to be synergistic with SEEKING in social engagement. Therefore, to Panksepp's well delineated experimental types we may add bivalent notions of approach and withdrawal, object relationship, and specific emotion (Table 2). In writings subsequent to the seminal Affective Neuroscience (Panksepp, 1998), Panksepp and Biven (2012) have included human play, socialisation and enjoyment in their conception of play. This meshes with a triune HSE (Figure 5).
Table 2  Strategic Motivational Affect Systems

<table>
<thead>
<tr>
<th>Strategic Motivational Affect System</th>
<th>Arousal type</th>
<th>Orientation</th>
<th>Affect program</th>
<th>Emotion(s)</th>
<th>Strategic Locus</th>
</tr>
</thead>
<tbody>
<tr>
<td>TENSION</td>
<td>Tense</td>
<td>Approach</td>
<td>ANGER/RAGE$^a$</td>
<td>Anger emotions</td>
<td>Dominance</td>
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<tr>
<td></td>
<td></td>
<td>Withdrawal</td>
<td>FEAR$^a$</td>
<td>Fear emotions</td>
<td>Submission</td>
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<tr>
<td>ATTACHMENT</td>
<td>Agapic</td>
<td>Approach</td>
<td>CARE$^a$</td>
<td>Love</td>
<td>Nurturance</td>
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<td></td>
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<td></td>
<td>Joy</td>
<td></td>
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<td></td>
<td></td>
<td>Agapic pride</td>
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<tr>
<td></td>
<td></td>
<td>Withdrawal</td>
<td>GRIEF$^a$</td>
<td>Sadness</td>
<td>Love object recapture</td>
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<td></td>
<td></td>
<td></td>
<td>Anguish</td>
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<tr>
<td>HEDONIC ENERGETIC SOCIAL ENGAGEMENT</td>
<td>Hedonic</td>
<td>Approach</td>
<td>PLAY$^a$</td>
<td>Enjoyment</td>
<td>Social engagement</td>
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<tr>
<td></td>
<td>Energetic</td>
<td></td>
<td>SEEKING$^a$</td>
<td>Amusement</td>
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<td>Excitement</td>
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<td>Withdrawal</td>
<td>SORROW</td>
<td>Sorrow</td>
<td>Social withdrawal</td>
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<td>Despair</td>
<td></td>
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<tr>
<td>LUST$^a$</td>
<td>Sexual</td>
<td>Approach</td>
<td>LUST$^a$</td>
<td>Sexual longing</td>
<td>Intercourse</td>
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<td></td>
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<td></td>
<td>SEXUAL AVERSION</td>
<td>Sexual aversion</td>
<td>Abstinence</td>
</tr>
</tbody>
</table>

Note. $^a$ Panksepp’s emotional operating systems
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There are, however, good reasons to refute the SMAS model, the most particular being that it contravenes the established view that approach and avoidance are largely independent motivational orientations (Ito and Cacioppo as cited in Elliot & Covingtonge, 2001). Electrical brain stimulation reveals a localisation of stimulus to particular behaviour repertoires, such as those described by Panksepp (1998), suggesting a distinct spatial location for approach and withdrawal neural circuits. Lastly, as here described, the SMAS would need to be an exceptionally complex apparatus to coordinate the functions that it does, as current notions of evolutionary neurology favour a modular structure.

Yet there are several levels of conceptualisation that we may apply to this notion, the least grandiose of which would be to see the SMAS as simply a coordinating mechanism linking approach and withdrawal motivations around arousal, therein coordinating social dominance, attachment, and engagement. There are further reasons to retain the SMAS concept. Davidson, as cited in Elliot and Covingtonge (2001) uses a bipolar (reciprocal) construct to explain approach and withdrawal across the cerebral hemispheres. Panksepp (1998), in a similar vein, has united CARE and GRIEF into an Integrative Emotional System for Social Affect. He further recognises the possibility that defensive anger may represent a concurrent mixture of ANGER and FEAR, and the two circuits interdigitate extensively (Panksepp, 1998). As will be discussed later, the neurochemistry of the opposing circuits in ATTACHMENT and HESE are remarkably linear in their orientation; neurotransmitters which are associated with approach are typically linked to the amelioration of withdrawal states (Table 18). Clinically, if it is assumed that affective disorders arise from dysfunctional approach and withdrawal neural circuits, states such as lability and mixed states suggest a cross sectional activation of opposing diatheses. Similarly, the approach and withdrawal tendencies of many psychiatric disorders are linked longitudinally, for instance, the triggering of hypomania from depressive states by antidepressants. In an imaginal context, joy is often closely associated with its antithesis, grief (sadness). For these reasons, it is preferable to adhere to Izard (2007) in concluding that emotions are "dynamically interacting patterns" (p. 269) and retain the SMAS model as an heuristic.
Nesse (2004) has presented his schematic *A Possible Phylogeny of Emotions*, in which he proposes a fundamental progression of emotions across evolutionary time, from arousal to the opposition of approach (*excitement/desire*) and withdrawal (*apprehension/fear*). This resulted in a division of affect into positive and negative, heralding more differentiated personal, reproductive, and social efforts. Specific emotions are seen as partially differentiated from more primal generic states due to selective advantage. These overlapping affects rest on branches which depict evolutionary bifurcations such as status (*pride*) and status threat/loss (*shame*), kin/opportunity (*love*) and kin loss (*grief/sadness*). Elicitors, functions, cognitive and somatic characteristics overlap (Nesse & Ellsworth, 2009) and Nesse and Ellsworth (2009) argue that “different emotions are not defined by different functions or mechanisms, or specific stimuli, or brain modules, or even by particular points in dimensional space. To the extent that there are different emotions, they correspond to different situations that have recurred over the course of evolutionary time.” (p. 132) Thus, this model places emotion within its evolutionary context, without location or specific mechanism. In stressing individual variation, they suggest that a clear taxonomy of emotions may not exist.

In distant time, genes learnt the benefits of confederation and cooperation (Dawkins, 1976), allowing cellular entities to aggregate and form specialised organs. Assisted by pluralism, genetic programs facilitated the pursuit of beneficial objects, while protecting their survival machines from dangerous ones. From the beginning of cellular life, approach and withdrawal variants have undergone neural differentiation and selection. Approach and withdrawal have been described as the two basic patterns applicable to all motivated behaviour (Maier & Schneirla, 1964). It has been speculated that early in evolution the nascent nervous system became subject to two systems of arousal; *energetic arousal* (facilitating movement, rest and sleep), and *tense arousal*, mediating danger orientation (Thayer, 1989). In alternate phylogenetic bifurcations of CNS arousal, strategic motivational affects such as thirst and hunger maintained homeostasis.

I suggest, in the following model, that with vertebrate evolution, tense arousal became differentiated into tense-approach (later to become *anger*) and tense-
withdrawal (later to become fear). These affects facilitated the ability of organisms to oppose competitors and achieve dominance, or else submit in order to survive. Tense arousal, according to this model, therefore formed bivalent poles, instantiating the SMAS TENSION, which differentiated to the ANGER approach emotions of defiance, contempt, and anger. Disgust may have arisen as a derivative of both fear and aversive hunger. With hominin evolution, tense-withdrawal became fully differentiated, expressing the FEAR emotions; surprise, embarrassment, apprehension, fear and freeze-fight-flight and fright, arranged in a gradient of increasing tense arousal. The core of tense arousal remained at the heart of these emotions, just as it does with anger. Ontogenetically, social dominance is one of the earliest and most persistent personality traits (Cummins, 2005). Anger, which induces stratified dominance, promotes resource holding potential (RHP) (Parker, 1974) and ensures that sexual and other resources are allocated to fit individuals. TENSION stabilizes society through agonism, inhibited agonism (Chance & Jolly, 1970) and the creation of a dominance narrative. The gene, in this way, has extended its phenotype (sensu Dawkins & Dennett, 1999) to the domain of social structure. Social rank determines not only subjective state, but long term quality of life, morbidity and survival (Cummins, 2005).

The triune forebrain “has evolved and expanded to its great size, while retaining commonalities of three neural assemblies. These reflect an ancestral relationship to reptiles, early mammals, and late mammals.” (MacLean, 1985b, p. 219). MacLean (1982) has described courtship, challenge, and submissive displays in lizards, reflective of the early emergence of sexual affects, dominance (anger) and submission (fear), within the R complex. He dates play and affiliation with the development of the paleomammalian brain (MacLean, 1985b). The mammalian separation call, an emblem of object loss and an integral part of attachment, appears closely linked to the GRIEF emotions.

Within the limbic system, particular emotions and behaviours are specifically linked (MacLean, 1985a). The evolutionary layering of this tripartite neurophysiology is represented in Figure 5. In the bottommost anatomical layer basic reptilian energetic processes are represented as simple approach and withdrawal, while, with
the advent of ancient mammals (paleomammalian) play speculatively evolved as an homology of energetic arousal. In Homo sapiens and Pan troglodytes hedonic process arose as an homology of play – representing the *hedonic mode* of Chance and Jolly (1970). In this neomammalian mode the object of arousal is prestige (evident in emotional valence), or in some contexts sorrow. MacLean (1982) stipulated that later derivations of the psychencephalon are additive to the earlier levels. Thereby in the *HESE* triune model, hedonic arousal, play and energetic arousal all constitute a parallel yet synergistic system. For these reasons in states of *HESE* arousal one sees energisation, gesture, approach to social objects, playfulness, vocal prosody and positively valenced emotions. In the withdrawal – depressed – state one sees just the opposite.

*Figure 5.* A triune depiction of the hedonic energetic social engagement system.
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

The Leary circle, or interpersonal circumplex, was derived from observations of therapy group process, leading to an empirically derived two dimensional structure of dominance-submissiveness and nurturance-coldness (Pincus, Gurtman, & Ruiz, 1998). Kiesler and Auerbach (2003) cite extensive data establishing the interpersonal importance of control (power, dominance) and affiliation. Oatley, Keltner, and Jenkins (2006) have posited a framework for the interaction of social motivation and emotion, whereby the orthogonal motivational dimensions; attachment, assertion and affiliation map to those of the interpersonal circumplex and to the author’s system. Affiliation designates warmth and affection, which are extended in HESE to encompass play and creativity. Oatley et al. (2006) have further proposed bivalent emotion terms internal to their dimensions, though issue is drawn with their emotion assignment. Nevertheless, the convergence of the circumplex with McDougall (1919), Plutchik (2001) and Oatley et al. (2006) with the author suggests an approximation of a coherent motive structure. Alongside LUST (Panksepp, 1998), 1998) and ATTRACTION (romantic love) (Fisher, Aron, Mashek, Li, & Brown, 2001), three SMAS’s, ATTACHMENT, TENSION and HESE, adroitly compass our interpersonal emotional terrain. Notably, the systems are open, as contrasted to closed instincts. With the latter the behaviour pattern is fixed genetically in every detail; open instincts are “programs with a gap…filled in by experience” (Midgley, 2002, p. 53).

The phenomenological linearity of HESE is reflected in Table 3, which reflects a domain gradient, increasing in arousal from melancholia, through normative HESE, to hypomania. This suggests that the three states arise within the one system with different levels of arousal.
Table 3 The Linearity of HESE at Three Affective Levels in Multiple Domains

<table>
<thead>
<tr>
<th>Locus</th>
<th>Melancholia - withdrawal (low arousal HESE)</th>
<th>Normative HESE</th>
<th>Hypomania - approach (high arousal HESE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agency</td>
<td>Non-agent</td>
<td>Performer or audience</td>
<td>Performer</td>
</tr>
<tr>
<td>Social</td>
<td>Social withdrawal</td>
<td>Collective role</td>
<td>Social consumption/intrusion</td>
</tr>
<tr>
<td>Affective</td>
<td>Melancholia</td>
<td>Enjoyment, amusement</td>
<td>Enjoyment, amusement, excitement</td>
</tr>
<tr>
<td>Energetic</td>
<td>Anergia</td>
<td>Gesture, emphasis</td>
<td>Gesture, animation, energisation, insomnia</td>
</tr>
<tr>
<td>Cognitive</td>
<td>Psychic retardation</td>
<td>Confidence</td>
<td>Grandiosity</td>
</tr>
<tr>
<td>Speech</td>
<td>Poverty</td>
<td>Prosody, emphasis</td>
<td>Dramatisation, pressure</td>
</tr>
<tr>
<td>Playfulness</td>
<td>Absent</td>
<td>Moderate</td>
<td>Exaggerated</td>
</tr>
<tr>
<td>Narrative</td>
<td>Minimal</td>
<td>Expressive</td>
<td>Creative, rapid</td>
</tr>
<tr>
<td>Appetitive</td>
<td>Non-consumptive</td>
<td>Socially consumptive</td>
<td>Socially monopolistic</td>
</tr>
</tbody>
</table>
On the Origin of Mood Disorders

“At the extremes are pathological states such as mania and paranoia—but even these can be conceived as extreme expressions of such basic emotions as sadness, joy and disgust.” (Plutchik, 2001, p. 350)

Though rooted in prehistory, bipolarity is providing new challenges in its conceptualisation. Aetiological theories involve notions of the manic defense (Abraham, 1927), BAS dysregulation (Alloy & Abramson, 2010), goal dysregulation (Johnson, 2005b) and (de)escalation around rank contest (Price et al., 2007).

We monitor the social environment for cues indicative of reduced relational value (Leary, 2005b) – prestige. The Prestige model of spectrum bipolarity hypothesises that instability in prestige and self-esteem underlies dysregulation of the Hedonic Energetic Social Engagement system (HESE), leading to affective syndromes. In HESE, subject and object are linked by affect, cognition, social goals and intervening objects. Art, here deconstructed, is taken to be the everyday product of HESE. Thus humorous narratives, an inflexion in the voice, fashion, a pose, or a line of song are taken to be artworks, which intervene between subject and object (or group) to enhance relational value. Approach HESE intends the individual toward “play” and group engagement and operates through the emotions of enjoyment, amusement and excitement. HESE signals maintain interpersonal cohesion.

The Environment of Evolutionary Adaptedness mandated group inclusion, as “compulsively social” (Foley, 1995, p. 197), hominin groups faced adversity. Selection meant inclusion to the nomadic ingroup, with benefits for survival and reproduction, while unselection (Price, 2009) meant ostracism. The process of inclusion was metered at the group level by prestige – “the extent to which the group is prepared to invest in the individual.” (Price et al., 2007, p. 540). Individuals, in turn, gauged prestige by internalising it to self-esteem, through the function of the sociometer, a biopsychological meter. The sociometer (Leary et al., 1995) monitors relational value and drives approach behaviours to maintain group inclusion. The prestige model proposes that bipolar disorder episode precursors create relational uncertainty, in turn diminishing self-esteem. A positive feedback loop is initiated;
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activation and circadian rhythm changes increase the extent and duration of goal pursuit. Stabilisation usually occurs, though in states of recursive prestige instability, bipolar persons possess an override which leads to either downregulation (depression) or upregulation (hypomania) of the prestige-HESE system.

Those who were unselected in ancestral time were prone to die through ostracism. Melancholia provides an alternative. The ritualisation of depression conveys submission and promotes survival “like a person asleep or a hibernating animal” (Price, 2009, p. 1034). Melancholia (Figure 6) in its psychomotor retardation, avolition, unadornment, object withdrawal, reduced food demands and absent libido represents a withdrawal from objects and an abrogation of intrasexual competition. It provides for an invisibility which moderates ostracism; the message being "I am dormant" or "Look, I can die all by myself!" The depressed person “wears” the anti-ornaments of prestige – the stigmata of melancholia, as kin are able to maintain the life of the individual. Kindling and sensitisation (Post & Weiss, 1998) may lead to the process becoming semi-autonomous. After recovery, natural selection leads to transmission of the melancholic adaptation.

Whilst in melancholia social disinvestment predominates, in hypomania there is a frenzy of social engagement aimed at raising relational value. Affective upregulation triggers a tonically excited approach HESE arousal system, with affect, intervening objects and goals directed at prestige objects. The faulty prestige override makes the individual fail to recognise that social prestige has stabilised. Increased efforts induce the personal set of performer. Artworks include; affect display-induction, linguistic expression, sociality, gifts, creativity and status. This interactive state encapsulates the behavioural ornaments of intersexual selection, described in Dutton (2009). Hypomania is a state of excess marketing (handicap), which indicates a voluminous capacity to be the perfect partner and parent (fitness). Ethologically, prospective partners are attracted to “profligate waste”, this being an indicator of sufficient fitness to carry the handicap (Zahavi, 1975). Thus hypomania leads to “visible” social inclusion and advantageous intersexual selection. Adaptive genes are transmitted and the hypomanic disposition toward prestige “override” is perpetuated.
(Hypo)mania has an aboutness to it, aptly described by Leader (2013):

“If we look at the different forms of flight of ideas, don’t they all have one thing in common? Don’t they all involve a recognition of the addressee, the one who is being spoken to? Even if the manic subject moves from one topic to the next with an apparent disregard of theme or content, they are still talking to someone. When Behrman characterizes mania with the formula ‘What would you like to see me do next?’ the key is in the ‘you’. There is an unquenchable thirst for an addressee. Manic subjects, unlike others, will not talk to themselves.”

Figure 6 depicts the interrelationship between precursors to affective illness, HESE, affective ornaments, self role, syndrome and adaptation. Prestige may be lowered by a lack of social support, negative life events and high expressed emotion and ostracism is threatened. The sociometer, in this model, detects reduced relational value and lowers self-esteem, leading to adaptive activation, cognitive strategies (including ToM), social goal pursuit and hedonic affective display. Persons with affective disorders are, however, affected by cognitive and behavioural aberrations, being prone to dysfunctional cognition and social reward sensitivity. Goal pursuit is abnormal and they are prone to perfectionism (Mansell & Pedley, 2008) These factors, along with dichotomous self-concepts, make them prone to aberrant up-or down-regulation of affect. Tonic excitation of affect leads to approach toward objects and the prototypal hypomorphic ornaments of linguistic expression, risk taking and energisation. The self is portrayed as performer, as hypomania results in social approach and visible social inclusion. Tonic inhibition of HESE leads to social withdrawal and melancholia. Paralleling the social risk hypothesis of depression of Allen and Badcock (2003), depressed persons may enter a stealth (J. M. Le Bas et al., 2013) mode in which ostracism may be avoided. As previously indicated, because mild to moderate depression may predicate survival in the face of a selection pressure, this may be carried by natural selection into the next generation.
Figure 6. A model for the pathogenesis of melancholia and hypomania
Mania and psychotic depression may eventuate when the tense arousal system is secondarily affected, as it is possible that mutually excited arousal systems may generate more complex pathogenic states. Mania is characterized by motoric agitation, irritability, dyscontrol, and psychosis, which may alternate with hypomanic jocularity. It is conceived here to represent a composite of Panksepp’s PLAY/SEEKING (approach HESE) and RAGE (approach TENSION). Psychotic depression, with its morbid anxiety, sorrow, ruminations, and delusional beliefs, is proposed to arise from the confluence of SORROW (withdrawal HESE) and FEAR (withdrawal TENSION). Within the tense arousal system, composites of contempt and fear may give rise to paranoia. Simple prestige connotes that the community freely confers respect, standing and distinction to the individual. It is noteworthy that paranoia, common in mania, entails the antithesis of prestige. Paranoid persons believe that the community observes malignly, schemes maliciously and holds malevolent intent. Perhaps the prestige system is liable to not only instability, but also to functional inversion? Mixed states may arise when more than one SMAS is pathologically involved, as may occur in bipolar I disorder. It is postulated, that in addition to HESE arousal leading to alternating elevated mood and depression, tense arousal may add anger and agitation to the mix. States of agapic joy (CARE) may fluctuate with GRIEF (sadness-distress) – Panksepp’s Integrative Emotional System for Social Affect – resulting in lability. Such a conglomerate is sometimes seen clinically and cannot be the result of a single aberrant neural circuit.

Panksepp (2010) has suggested that all of the affective networks may be influenced by depression and he suggests that sustained overactivity of GRIEF, via a "protest gateway", may mediate the painful quality of depressive affect. According to this view, persistent depressive dysphoria results from reduced activity of the SEEKING system, making the model contingent on a disturbance of reward. Consistent with the view held here, and consonant with the work of John Bowlby (1969), social relationships may be a key trigger to a depressive episode. Intrinsic to the Panksepp (2010) model – and a position taken in this thesis – is that neural circuits may be co-morbidly activated. Panksepp also invokes FEAR as a possible contributor to anxious forms of depression. With respect to therapy, he suggests that approaches which promote the hedonic activity of social CARE and PLAY may assist recovery.
Conclusion

"In summary, there are identifiable EEG and ERP characteristics that correlate with the states and symptoms of depression and mania that may differentiate the two poles of the illness: withdrawal or negative valence-related right-hemispheric dominance for the depressed phase and approach or positive valence-related left-hemispheric dominance for the manic phase, each related to changes in network properties."

(Outhred, Kemp, & Malhi, 2014)

From the perspective of biological life centred on genes, survival and replication are the ultimate purpose. After Dawkins (1976), bodies are vehicles for selfish genes, being organised through evolutionary devices such as emotion to promote pursuance of the gene passengers. In the Pleistocene, prestige selection was a matter of life and death and determined the adaptive behaviours such as mood change, while byproducts of these adaptations resulted in more severe affective disorders. This chapter has built a model of strategic motivational affect systems – anchored within a phylogeny of emotion and arousal – to argue that emotion and prestige form a cohesive structure. The chapter has integrated extant knowledge of arousal and emotion systems, by adapting models developed by authors such as Panksepp and Thayer. Affect and object relations matter deeply to both individuals and society.
Narrative – Ruth

Ruth is a 44-year-old married mother of two late adolescent children, who works as a part-time legal secretary and beautician. She was the product of a normal gestation and was a placid, healthy and plump baby. She was picked on at school for being overweight. She described long holidays as a child to Inverloch and reminisces about feeling carefree. Ruth's maternal grandmother had been treated with ECT and hospitalisation. There was also a family history of eating disorder.

Ruth complained of depression since her teens – a feeling which left her enervated and despairing. As a girl she would resort to making herself vomit when confronted with things which were out of her control. In her 20s she started to notice unusual periods where she felt energised and terrific in herself. Ruth's elevated moods lasted several days and occurred every two weeks or so. She described becoming racy and jittery with fast speech and increased shopping and spending. She used the phone more and pursued her goals with alacrity and a sense of playfulness. Her depressions were occurring at roughly the same frequency and were notable for agitation, mental slowing and a sense of being "locked-up". She ruminated and became angry with herself at these times, due to her severe fatigue and lack of motivation. Her pattern of mood swings became entrenched in her 30s, with the fatigue and low mood becoming long-lasting. Around this time she was diagnosed with chronic fatigue syndrome and myalgia. Muscular tension, along with neck pain and worry were prominent aspects of her condition and alcohol became a notable relief from these symptoms.

It was particularly difficult for Ruth to be with people in a social context; this leaving her on guard and concerned that "they may not get (her)". In discussing this, she quickly curtailed conversation and said that basically she wanted to be left alone. She would say "I come first or else I have no energy to look after the family…I have to pull the line". Ruth particularly complained of low energy with "fuzzy" thinking and feelings of being slow and spaced out. She complained of little "spring" in her life and said that she was just managing to cope. "The dark might smother me" she said; "I have a choking sensation, I have to block it out… I have to put myself behind a screen". She described creating a mental image of the Great Wall of China, behind
which she could hide. "I can be myself and not display myself to others". Behind this barrier she could visualise herself not being touched by people; feeling detached and in a safe space. Often, however, there was a "dead feeling in (her) gut".

Common triggers to the need to seek such solace were feeling that she made the wrong impression in front of arrogant or critical people. Commonly, however, just socialising with women from a sports club would lead to this form of isolation. Her movements would tend to become slower and this was associated with insecure evaluations of herself. When she is required to, Ruth is able to perform for society on the "outside", despite a shrinking feeling inside. This involves her part-time work, though this results in her becoming deflated and aversive of contact. Despite an overseas trip, she still found herself being "behind a glass wall".

Ruth's daughter Bethany, with whom she identifies strongly, presented in a similar fashion to Ruth during her teens. She would have periods of withdrawal and suicidal thinking, interspersed with times she was affectionate and "in everyone's face". Both Ruth and her daughter are diagnosed with Bipolar II disorder and have been placed on similar treatments.
Chapter 5: Spectrum Bipolarity

“Lastly, we include here 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 over without sharp boundary into the domain of personal predisposition. In the course of the years I have become more and more convinced that all the above-mentioned states only represent manifestations of a single morbid process.” (Kraepelin, 1921, p. 1)

"In perhaps the most notable example, the bipolar clinic in Harvard's teaching hospital, clinicians are taught to ask 'how much bipolarity might this patient have?' as against the categorical 'does this patient have bipolar disorder, or not?'" (Phelps, 2008, p. 23)

Introduction

The matter considered in this thesis is the overlapping landscapes of affect and societal process, representing Naomi’s responses to social engagement, and Ruth’s envelopment in states of depression which may mitigate against ostracism. In building a model of this terrain it is pertinent to examine what is known of the extremes of affect seen in the bipolar spectrum. This chapter lays the groundwork for the prestige spectrum. It does so by presenting a review of the literature on bipolar disorder from the time of Aretaeus to the clinical constructs of Kraepelin, leading to modern day conceptualisations. It will be seen that the history of the spectrum entails revision, reversal and rediscovery. For this reason it is important to note not only clinical archetypes, but also their context.

The bipolar spectrum describes a prevalent group of conditions which are poorly understood. Its nosology involves a tension between lumping and splitting and several models are discussed. These range from a disaggregated category paradigm to a fully dimensional model. An intermediate nodal-dimensional form is considered along with its evidential support. The elucidation of these conditions will, however, involve not only a description of their clinical phenotypes and familial inheritance as presented here, but an understanding of their proximate and distal basis. Lastly, the prestige spectrum will be described, drawing on the notion that mood disorders fall along clinical and genetic gradients, in which “affective building blocks” are situated.
These building blocks may aggregate toward the severe end of the spectrum, culminating in the complexity of bipolar I disorder.

**History**

Bipolar disorder was first described by first century physician Aretaeus (1972) of Cappadocia. He recognized the common aetiology of mania and melancholia and also their biological causation. Of course the illness existed long before this; its geographic dispersal suggests an origin in African Homo sapiens, before migrations north and east 50 000 to 100 000 years ago. King Saul (1079 BC – 1007 BC) arguably suffered with bipolar disorder (Stein, 2011). Plato (427–347 BC) recognised varieties of mood excess, while Aristotle (384–322 BC) may have recognised alternations of melancholy and mania due to perturbations of “black bile” (as cited in Pies, 2007).

Shorter (2008) has written a comprehensive review of the modern history of bipolar spectrum disorders. He cites descriptions of bipolar-like illnesses dating back to the 18th century. Carl Friedrich Fleming (1844) is credited with the description of "dysthymia mutabilis", an alternation of depression and what would now be called low-level mania (Shorter, 2008). Griesinger (1845) heralded Falret’s (1851) description of la folie circulaire – continuous cycles of depression, mania and normal interval (as cited in Angst & Marneros, 2001). The syndrome now termed bipolar disorder was further defined by German psychiatrists, including Ludwig Kirn (1878) (Shorter, 2008). By the end of the century, Kraepelin (1896) had split the functional psychoses into dementia praecox and manic depressive insanity. Kraepelin took a broad view of the latter, incorporating what we now know as bipolar illness, cyclothymia, affective temperaments, and unipolar depression. Affective disorders were defined by the pressure of intense emotion, recovery and recurrence (Kraepelin, 1921).

Following Kleist (1911), in 1957 Leonhard (1995) separated modern-day bipolar affective disorder from "pure depression". This was refined by Angst and Perris in 1966, who proposed a nosological division between unipolar and bipolar illnesses (Angst & Marneros, 2001). By 1980, DSM-III had separated unipolar depression (major depression) from bipolar manic-depression (bipolar disorder). The
bipolar/unipolar dichotomy had continued to dominate diagnostic standards and clinical practice (Ghaemi & Baldessarini, 2007) and had now reached its apogee.

Aretaeus had pre-empted the modern categorical nosology of elevated mood states by some 2000 years, describing two syndromes equivalent to Dunner and colleagues’ conceptualisation:

"And they with whose madness joy is associated, laugh, play, dance night and day, and sometimes go openly to the market crowned, as if victors in some contest of skill; this form is inoffensive to those around. Others have madness attended with anger; and these sometimes rend their clothes and kill their keepers, and lay violent hands upon themselves. This miserable form of disease is not unattended with danger to those around." (Aretaeus, 1972)

Subsequently, Dunner, Fleiss, and Fieve (1976) envisaged depressions with hypomania (bipolar II disorder includes episodes of elevated and energised mood) and depressions with full blown mania (bipolar I disorder). Classifications such as those of Klerman (1981) (Table 4) and Akiskal (2008) (Table 5) began to take on a neo-Kraepelinian form (Shorter, 2008). Frank (2011) argues that the focus on reliability led to a reification of diagnostic categories as research has found both evidence for distinct categories and for the bipolar spectrum. Taylor and Abrams as cited in Shorter (2008) applied a rigourous method to the classification of affective illness in 1980 and suggested that there had been premature closure around classification by illness polarity. Subsequently, Taylor and Fink found that family studies, psychopathology, laboratory tests and treatment response did not support the notion of bipolar disorder as separate (Taylor & Fink, 2006). Darwin's observation that case variations are distributed continuously does not invalidate categories, but rather limits them to certain distributions where case features do not overlap substantially (Ghaemi & Baldessarini, 2007). The validity of the categorical classification was again open to question, as diagnostic systems yawed around notions of dimension and singular disorder.
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

Demographics

The National Comorbidity Survey (Merikangas et al., 2007) examined the lifetime and 12 month prevalence of bipolar spectrum conditions in a US population. It found that the disorders were more common than expected when a broader spectrum was considered. Lifetime prevalence for bipolar I disorder was 1.0%, whilst that for bipolar II disorder was 1.1%. An additional subthreshold category, in which affective symptoms do not meet diagnostic criteria, had a lifetime prevalence of 2.4%. The latter had moderate to severe clinical severity and role impairment. Of relevance to the exploration of the role of tense arousal in bipolar disorder, spectrum illnesses were particularly associated with anxiety (Merikangas et al., 2007).

An international study of bipolar spectrum prevalence found considerable variation in lifetime prevalence, ranging from 0.1% in India to 4.4% for the United States (Merikangas et al., 2011). One must wonder, however, at the basis of this wide intercontinental variation. The notion of a spectrum gradient is further supported by Merikangas’s finding that the severity of affective symptoms and suicidality increases monotonically from subthreshold states to bipolar I disorder. Role impairment, however, was comparable across bipolar subtypes (Merikangas et al., 2011). Up to 20% of adolescents, furthermore, may have a "wide bipolar trait" as measured on the Mood Disorder Questionnaire, wherein some degree of hypomania occurs, but usually causes little impairment (Goodwin, 2010). With a prevalence of 16.5% (Anonymous, 2011), were unipolar depression included in the spectrum construct, the lifetime prevalence of bipolar spectrum conditions would climb to around 20%.

The Bipolar Spectrum

“Socrates: The second principle is that of division into species according to the natural formation, where the joint is, not breaking any part as a bad carver might.” (Plato, 360 BCE)

A spectrum is a system in which components are contiguously distributed according to rank. This is best exemplified by the spread of the wavelengths of light, as induced by refraction through a prism. Various attempts have been made to arrange the mood disorders into such a hierarchy. Devin Henry traces realism back to Plato's
Phaedrus, in which Socrates employs the metaphor of a butcher to describe both *collection* and *division*. In collection, scattered things are seen and drawn “into a single form”. In division, each thing is cut “along its natural joints.” (Henry, 2011, p. 198). Clearly, categorisation was an issue for Plato, no less than for contemporary psychiatric nosologists, who grapple to collect bipolar phenomena together, whilst dividing natural kinds at their borders.

Kelsoe (2003) notes that whilst it is clear that wavelength is the essence of the photic spectrum, it remains less clear as to what construct underlies the bipolar spectrum. Because of non-bipolar constituents, it is not “bipolar-ness” and because unipolar probands may have a similar severity to bipolar individuals, it is not severity (though see Merikangas et al. (2011). The bipolar spectrum is neither strictly quantitative nor qualitative, but a mix of both. Kelsoe posits that a solution may be that the bipolar spectrum is a “surface in a multidimensional space”, which allows for a quantitative relationship between qualitatively different aspects of illness.

Further problems in psychiatric nosology include the clinical overlap between unipolar and bipolar disorders. Several symptom-based studies have failed to identify a zone of rarity between unipolar and bipolar conditions (Benazzi, 2003; Hantouche, Angst, & Akiskal, 2003; Parker et al., 2012). Epidemiological and clinical research has found a continuous distribution of both depressive and (hypo)manic symptoms and syndromes ranging from normal to morbid (Angst, 2007). The BRIDGE study used a novel “bipolarity specifier” and found that 47% of putatatively unipolar patients in fact were likely to have bipolar disorder (Angst et al., 2011).

Counterintuitively, depressive affect and cognition are situated prominently within factor analyses of mania (Cassidy, Murry, Forest, & Carroll, 1998). A lifelong follow-up of hospitalised patients indicated a likelihood of diagnostic change from recurrent depression to bipolar disorder of 1.25% per year (Angst, 2007). Major depression is common to both bipolar and unipolar illness, while mixed affective episodes are relatively frequent also (Akiskal & Akiskal, 2007). Subthreshold conditions do not fit squarely into diagnostic categories. It is common to find that amongst monozygotic twins, in addition to bipolar-bipolar concordance, there will be siblings with other psychoses, affective personalities or suicide (Kelsoe, 2003). Only around 15% of the
population report no depressive and hypomanic symptoms (Angst, 2007). Lastly, psychopharmacological agents often show a nonspecific effect across a range of mood and temperamental disorders. Clearly, there are problems around the lumping and splitting of affective conditions, which prompt alternative conceptualisations.

Angst (2007) notes that a dimensional concept for affective disorders (cyclothymic temperament – cycloid "psychopathy" – manic depressive disorder) was noted by Kretschmer (1921) and Bleuler (1922). The term "spectrum" received use in psychiatry in 1968 with relation to the schizophrenia spectrum. The principal forms of the spectrum models are presented below:

**Disaggregated Spectrum Categories.**

A cyclothymic-bipolar spectrum was first proposed by Akiskal, Djenderedjian, Rosenthal, and Khani (1977), while Klerman (1981) suggested a mania spectrum. In Table 4 Klerman disaggregates affective conditions into six types based on clinical, treatment and familial features. The refinement by Dunner et al. (1976) of bipolar disorder into bipolar I and II is recognised. Grof, as cited in Ghaemi, Ko, and Goodwin (2002) had suggested that classic type I bipolar illness may differ in many respects from less typical types of bipolar illness, especially in being more lithium-responsive. The affective temperament of cyclothymic personality (cyclothymia) is designated bipolar III, whilst according to Ghaemi and Baldessarini (2007) this is included in the extended bipolar II grouping. Klerman includes a category for hypomania or mania precipitated by antidepressant drugs (category IV), whilst Akiskal and Mallya (1987) term patients with recurrent major depression, and a requisite family history of bipolarity, pseudounipolar or bipolar III. As with group IV of Klerman (1981), these people are prone to hypomanic episodes only upon challenge with antidepressants (Akiskal & Mallya, 1987). Klerman (1987) recognises unipolar mania (category VI) and a group of persons with depression and a familial history of bipolar disorder (category V).

Akiskal (2008) calls for a unification of affective syndromes into the spectrum, saying that the breadth of bipolarity is not represented in the DSM. He draws on documented brief hypomanic episode duration and the prevalence of
cyclothymic depression to argue for an enlarged bipolar II construct. His model is represented in Table 5. Alongside the major categories he includes intermediate forms, suggesting that his bipolar spectrum types are categorical phenotypes, suitable for genetic investigation (Akiskal, 2008). He has also promoted the cause of temperaments: "In brief, temperament as a construct appears proximal to the chain of the charged emotional atmosphere and the resultant personal adversity which define the psychosocial context of affective disorders." (Akiskal, 2008, p. 3). The hyperthymic temperament entails intermittent subsyndromal or hypomanic features with infrequent intervening euthymia. Such patients sleep briefly, use denial excessively, and are notable for an exuberant or irritable demeanour. They may be vigorous and full of plans, overtalkative, warm, stimulus-seeking and extroverted (Akiskal & Mallya, 1987). In those with depression occurring in the context of hyperthymic personality, Akiskal (2008) opines that their depressions are in fact pseudo-unipolar. The subaffective dysthymic temperament entails low-grade depression, habitual hypersomnia and depressive personality traits (Akiskal & Mallya, 1987). The cyclothymic temperament shows intermittent short cycles of elevated mood and depression with infrequent euthymia, in which hypomanic symptoms alternate with subsyndromal depression (Akiskal & Mallya, 1987). Paris, Gunderson, and Weinberg (2007) reviewed the putative relationship between bipolar conditions and borderline personality disorder, where rapid and usually triggered fluctuations of mood occur. They concluded that the current separation of borderline personality from bipolar I was justified, but conceded possible shared phenotypes between bipolar II and borderline disorders.

**Nodal-Dimensional.**

Ghaemi, Ko and Goodwin’s model of "bipolar spectrum disorder", on the other hand, bridges the gulf between bipolar I disorder and major depressive disorder (Figure 7). In subsequent writings, Ghaemi and Baldessarini (2007) concur with Benazzi (2007), saying that "extreme clinical archetypes in mood spectrum may indeed allow for separate, categorically conceived mood disorders, whereas intermediate variants may best be described dimensionally." (p. 66). Their 2007 model includes an extended "bipolar spectrum disorders" group which bridges bipolar
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II and cyclothymia. Manic-depressive illness is construed as an overarching construct comprising the manic, depressive, and intermediate forms of disorder. This is compatible with Phelps, Angst, Katzow, and Sadler (2008), where the “nodes along a continuum” are “analogous to the visual spectrum.” (p. 186). They conceive a revised DSM system, which may conceivably “incorporate a spectrum perspective without abandoning its categorical approach.” (p. 186). This could be achieved by adding nodes such as bipolar III (Phelps et al., 2008). Ghaemi et al. (2002) also set out non-manic "clues" suggestive of bipolarity. These include a postpartum onset and atypical depressive symptoms, to name just a few. These build on the "subtle indicators" which Akiskal and Mallya (1987) documented.
### Table 4  
Klerman's Primary Bipolar Types (Klerman, 1981)

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bipolar I</td>
<td>Mania and depression</td>
</tr>
<tr>
<td>Bipolar II</td>
<td>Hypomania and depression</td>
</tr>
<tr>
<td>Bipolar III</td>
<td>Cyclothymic disorder</td>
</tr>
<tr>
<td>Bipolar IV</td>
<td>Hypomania or mania precipitated by antidepressant drugs</td>
</tr>
<tr>
<td>Bipolar V</td>
<td>Depressed patients with a family history of bipolar illness</td>
</tr>
<tr>
<td>Bipolar VI</td>
<td>Mania without depression [unipolar mania]</td>
</tr>
</tbody>
</table>

### Table 5  
The Evolving Spectrum of Bipolar Disorders (Akiskal, 2008)

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bipolar</td>
<td>Schizobipolar disorder</td>
</tr>
<tr>
<td>Bipolar I</td>
<td>Manic-depressive illness</td>
</tr>
<tr>
<td>Bipolar I_</td>
<td>Depression with protracted hypomania</td>
</tr>
<tr>
<td>Bipolar II</td>
<td>Depression with spontaneous discrete hypomanic episodes</td>
</tr>
<tr>
<td>Bipolar II_</td>
<td>Depression superimposed on cyclothymic temperament</td>
</tr>
<tr>
<td>Bipolar III</td>
<td>Repeated depression plus hypomania occurring solely in association with antidepressant or other somatic treatment</td>
</tr>
<tr>
<td>Bipolar III_</td>
<td>Repeated hypomania occurring in the context of substance and/or alcohol (ab)use</td>
</tr>
<tr>
<td>Bipolar IV</td>
<td>Depression superimposed on a hyperthymic temperament</td>
</tr>
</tbody>
</table>
Full Dimensional Model.

The spectrum of Phelps et al. (2008) refers to the full unpunctuated continuum between unipolar and bipolar disorders inclusive. In a fully dimensional spectrum, varying *degrees* of bipolarity are represented. Such a model describes a point on this continuum where there is a bipolar spectrum disorder without mania or hypomania (Phelps, 2008). Such a model addresses the problematic phenomena of categorisation where the data appears dimensional. Boundary phenomena are no longer an issue and the shared genetic diathesis is accounted for. In a practical application of this model, the Bipolar Clinic at Harvard utilises a Bipolarity Index, wherein clinical, demographic and treatment variables may be summed to a score up to 100. This scale has not yet been validated or systemically weighted (Phelps, 2008). The logical extension of this approach is the diagnosis of "bipolarity" without the nuances inherent in categorical approaches. This would have major implications for clinical practice and communication.

Orthogonal Models.

Orthogonal arousal systems may be used to map affective syndromes. Angst (2007) combines the dimensions of affectivity and severity to locate the various disorders. Phelps (2008) on the other hand, has utilised the axes of mania and
depression to map mixed states, where high-mania and high-depression connote such conditions. Whilst this model has utility for mixed states, it makes the *a priori* assumption that mania and depression are orthogonally divergent. This contravenes the bipolar spectrum’s tenet of a continuous dimension.

**Summation of Models.**

"Instead, risk of bipolar disorder seems to be associated with dozens or even hundreds of genetic polymorphisms, each of which increases risk slightly—either as an additive effect or in interaction with as-yet-undetermined environmental factors. This suggests that spectrum presentations will be more common than the most extreme presentations. The same pattern holds for environmental risk factors such as low birth weight, substance exposure, low fish consumption, abuse, trauma, and familial high expressed emotion [33]. No single environmental mold presses out copies of bipolar disorder. Instead, all the factors confer risk for other conditions as well as for bipolar disorder. Different doses of risk and also interactions between environmental and biological-constitutional effects will generate a continuum of expressions that shade along a bipolar spectrum.” (Youngstrom, Van Meter, & Algorta, 2010, p. 483)

The application of the bipolar concept has led to wide consternation when applied to youths (Youngstrom et al., 2010), with particular respect to detrimental pharmacotherapy. The expanded boundaries of bipolarity lead to encroachment into the domains of unipolar illness, anxious-depression and axis II cluster B conditions (Akiskal, 2008). On the other hand, according to both Phelps (2008) and Akiskal and Akiskal (2007), if the full cluster of hypomanic symptoms cannot be elucidated (except in extreme and obvious bipolar cases) the diagnosis will erroneously default to unipolar depression and antidepressants are likely to be misprescribed. Conversely, the possibility of overdiagnosis of bipolar disorder exists (Phelps et al., 2008) and Healy (2006) warns of the potential for creating an "epidemic".

Paris (2009) opines that the wider notion of bipolarity, should meet broader criteria for validity and in doing so he is setting a standard which is not yet universally applied to psychiatric disorders. Similarly, Patten (2006) singles out extended bipolarity for lack of biological markers, which are of course lacking for psychiatric disorders in general. In his critique of the bipolar spectrum, Paris (2009) focuses on the potential inclusion of adult impulsive disorders, such as substance abuse, bulimia
and cluster B personality disorders. He views these as lying on the bipolar spectrum and putatively benefiting from comparable treatment. In so doing, he justifies his criticism by focusing on arguably the most spurious addition to the spectrum concept. Seeking to split off the intermediate bipolar spectrum from the categorical disorders, he also notes that subthreshold symptoms may be solely due to a different phenomenon or a normal variation and may not necessarily be linked to mood disorders. These are arguably erroneous criticisms, which fail to appreciate the modest claims made for the adoption of the bipolar spectrum concept.

Benazzi (2007) contends that by focusing on the mood spectrum’s extremes (bipolar I and major depressive disorder), a categorical model is supported. Midway disorders, on the other hand, (bipolar II and major depressive disorder with bipolar signs) support a continuity/spectrum approach. His literature review therefore supports both a categorical and a dimensional model of mood disorders. Psychometric validators (e.g. unimodal versus bimodal distribution, correlational and factor analysis) lend support to the continuity of affective disorders. Phelps (2008) states that “patients presenting with depression should be presumed (author's italics) to be bipolar unless a Bipolarity Index (or equivalent analysis including not just manic features but also family history, course of illness and response to treatment) is nearly or completely negative. It would be wiser, he says, to use the bipolar spectrum model alongside the categorical models such as the DSM. This opinion is given weight by the BRIDGE study (Angst, Azorin, et al., 2011), which found a high proportion of purportedly unipolar persons to have a more bipolar demographic, phenomenology and course.

The concept of a "bipolar spectrum" remains controversial for a number of reasons, including the lack of widely accepted definitions (Youngstrom et al., 2010). Due to its severity if untreated, undetected bipolar disorder leads to greater human costs than major depression (Angst, 2007). On the other hand, increasing bipolar diagnosis may have consequences for pharmaceutical marketing and side-effect burden. The introduction of dimensionality to bipolar disorders makes sense, though requires conceptual and empirical rigour. The alternative may be a broad and scientifically unworkable notion akin to the old concepts of "schizophrenia" and
modern constructs like “major depressive disorder” (Ghaemi & Baldessarini, 2007), where diagnostic terms harbour a range of heterogenous conditions. There are logistic consequences to a further overhaul in psychiatric nosology, which may increase chaos rather than diminishing it. "One can use two lenses, the categorical and spectrum perspective, simultaneously, asking which seems to bring a particular patient into focus more clearly.” (Phelps, 2008, p. 32).

The Research-Domain-Criteria-(RDoC) (2012) is a project which seeks to "develop, for research purposes, new ways of classifying mental disorders based on dimensions of observable behaviour and neurobiological measures." (p. 1). RDoC claims to be agnostic about current disorder categories, being built on fundamental units of analysis in the system. The project allows for variance which incorporates subthreshold psychopathology. It is clear that this endeavour meshes with the bipolar spectrum program.

Genetics

"The other inward, inbred course of melancholy is our temperature, in whole or part, which we receive from our parents… such as the temperature of the father is, such is the son’s, and look what disease the father had when he begot him, his son will have after him, and is as all well inherited of his infirmities as of his lands." (As at 1621 Burton, 2001, p. 211)

With respect to the understanding of bipolar disorder, psychiatry is hampered by a number of factors. Firstly, we do not understand the pathophysiology of the condition. To a large extent we lack biological candidates to reverse engineer to genetic loci. Diagnostic systems such as DSM-5 are based on clinical consensus, rather than biometrics. The bipolar spectrum is, furthermore, heterogenous and complex.

Kelsoe (2003) has written an excellent account of spectrum genetics – Arguments for the genetic basis of the bipolar spectrum. Probands with bipolar disorder have an increased familial incidence of both bipolar disorder and unipolar major depression, the latter being more prevalent given its higher base rate Smoller and Finn (2003). The illness risk for first degree relatives of bipolar probands
approximates 9% (Smoller & Finn, 2003). The approximate rate for unipolar disorder in first degree relatives is roughly 10%, relative to the population prevalence of these conditions (1% for bipolar disorder and 5% for MDD respectively). About 30% of dizygotic twins are concordant for bipolar disorder, whereas in monozygotic pairs the proportion climbs to 70%. It can be seen that there is a large genetic influence on bipolar disorder expression (Kelsoe, 2003).

Evidence is lent to the quantitative spectrum model by the fact that severe disorders are less prevalent than milder disorders and this supports the polygenic multiple threshold model (Figure 8). There is mixed support for a relationship between illness severity and genetic loading, however (Kelsoe, 2003). It has been found that there is an association between bipolarity and affective temperaments in relatives of bipolar probands. This applies also to affective phenomena such as depressive cognition and neuroticism, along with traits such as rigidity and compulsivity (Kelsoe, 2003). Kelsoe (2003) argues that there may be a complicated overlap between the multiple major loci and the polygenic models of bipolar inheritance. He terms this a mixed model.

Bipolar I and II disorders may aggregate separately within families, suggesting at least some genetic distinction (Andreasen et al., 1987). Coryell, as cited in Kelsoe (2003) investigated a cohort of families of bipolar individuals and compared respective rates of bipolar I and bipolar II illness. First degree relatives of bipolar I probands had a roughly equal representation of bipolar I and bipolar II diagnoses. First degree relatives of bipolar II probands, however, had overwhelmingly more bipolar II individuals than bipolar I (Coryell, as cited in Kelsoe, 2003). This suggests that a more common genotype predisposes preferentially to bipolar II illness (Kelsoe, 2003). It raises the possibility that bipolar I disorder may be a phylogenetic step further from bipolar II, with both shared and unique genes. On the other hand, Edvardsen et al. (2008) undertook a twin study which found that the genetic risks for bipolar I, bipolar II and cyclothymia were also shared, constituting "one entity". Their bipolar spectrum category had no shared family environment effects. According to the polygenic model, bipolar trait alleles are principally transmitted by persons without bipolar disorder. Such genes may have a selective advantage at the population level.
The picture of bipolar disorder as arising from defective genes leading to a deleterious phenotype may well be simplistic (Kelsoe, 2003).

![Diagram of Multiple threshold model of affective illness](image)

*Figure 8.* Multiple threshold model of affective illness. With an increase load of polygenes the risk of each illness passes a threshold. The pink area represents affective genes in the non-ill general population at a threshold which may confer advantage. (Modified with permission from Kelsoe (2003))

It is common to find, that amongst monozygotic twins, in addition to bipolar-bipolar concordance, there will be other twins with other psychoses, affective personalities or suicide (Kelsoe, 2003). The pure quantitative spectrum notion does not account for all of the diagnostic variants which exist in the families of bipolar individuals. This includes dysthymia, unipolar depression, schizoaffective disorder, and schizophrenia (Kelsoe, 2003). Kelsoe suggests a spectrum which includes all of these syndromes:

Schizophrenia-Schizoaffective -Bipolar I-Bipolar II-Unipolar Dep- Unipolar Dep Single-Cyclothymia-Dysthymia- Affective personality

Such a spectrum incorporates a psychotic dimension on the left overlapping with an affective dimension on the right, making clinical sense (Kelsoe, 2003). Please note that this model does not accord with the notion that affective and psychotic disturbance are orthogonal dimensions.

Several chromosomal regions have been implicated in bipolar disorder by linkage studies, although results have been inconsistent. An overlap with
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schizophrenia and schizoaffective disorder has been noted (Barnett & Smoller, 2009). Because of the apparent polygenic nature of the condition, genomewide association studies have become the principal method of investigation (Barnett & Smoller, 2009). Genomewide association analysis has implicated specific genes for the condition and these include DGKH, CACNA1C and ANK3 (Barnett & Smoller, 2009) and NCAN (Cichon et al., 2011). Twin studies have found that the hereditability of bipolar disorder is around 80% – 85% (Barnett & Smoller, 2009; Mitchell, 2011). It is widely held that individual genes predisposing to bipolar disorder have small effect sizes and that gene-gene and gene-environmental contributions operate (Barnett & Smoller, 2009).

Tsankova, Renthal, Kumar, and Nestler (2007) and Rutten and Mill (2009) have reviewed the evidence for epigenetic influences in psychiatric illnesses. Tsankova et al. (2007) note that "…most psychiatric disorders are not due to mutations in a single gene; rather they involve molecular disturbances entailing multiple genes and signals that control their expression." (p. 355). Rutten and Mill (2009) opine that the evidence is consistent "with the concept that the biologic underpinnings of these disorders are epigenetic in form rather than DNA sequence based." (p. 1045). Epigenetic processes involve DNA methylation and changes in chromatin structure (Rutten & Mill, 2009). The stage is set to begin an investigation of the ways in which environmental vectors interact with the gene to lead to epigenetic changes in gene expression leading to conditions such as bipolar disorder (Rutten & Mill, 2009). Given that this process is potentially reversible, consequences for clinical practice and prevention are implicit (Rutten & Mill, 2009).

Conclusion

The bipolar spectrum is a powerful and evolving model for affective disorders. The literature reviewed both unifies, and brings into relief, theoretical, clinical, epidemiological and genetically pertinent aspects. As such, we have a richer and more nuanced account of the landscape of affect. This allows for a foundation on which aetiological models may be built. Platonic notions of collection inspired Kraepelin to unify affective disorders into the manic depressive insanity concept, whilst evidence for division led to our current categorical types. The bipolar spectrum represents an
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attempt to unite these approaches according to the extant evidence. "Red" may be seen as red, and "blue" as blue, whilst acknowledging that the photic spectrum is constituted by dimensional radiation. For this, and evidential reasons, the nodal-dimensional models of Ghaemi, Phelps and Benazzi are given credence.

The notion of bipolarity continues to be expanded and refined. Future models will incorporate the clinical and genetic evidence for continuity with schizophrenic and schizoaffective disorders as suggested by Kelsoe (2003) and Barnett and Smoller (2009). Epigenetic research promises a scientific rapprochement of genetic and environmental vectors. The convergence of basic and clinical research will, in time, make the dispositional substrate of the bipolar dimension more fully known.
Chapter 6: Research Design

Introduction

The chapters preceding have sought to examine aspects of the bipolar spectrum and with respect to self-esteem have found normal scores for explicit measures, whilst implicit measures reflect low values (Winters & Neale, 1985) – reliant on external contingencies (Blairy et al., 2004; Pardoen et al., 1993) – and being therefore unstable (Knowles et al., 2007). I suggest that self-esteem is the biopsychosocial nidus which unifies the interaction of life events, cognition, response style, social support, and mood state. There is a gap in the understanding of the understanding of bipolar disorder which led to the methodology used here; a gap which was evident in dealing with affectively ill patients. Early in the preliminary research, an attempt was made to formulate emotions within a logical structure, one which would facilitate a conceptualisation of affective disturbance. This coincided with reading *Evolutionary Psychiatry* by Stevens and Price (2000). Their aforementioned quotation about prestige and its relation to self-esteem was intriguing, yet the mechanism appeared unclear until I started to contemplate what hedonic engagement was about. It presented itself as a consequence of self-esteem and the precursor of prestige in moment-to-moment interpersonal interactions. Seeing as approach and withdrawal hedonic energetic social engagement appeared a logical substrate for bipolar disorder, a connection between emotion, bipolarity and prestige appeared evident. The aim was therefore to investigate the relationship between prestige and bipolar disorders, as mediated by emotion. This was initially undertaken through the clinical interview and its evident mental state, leading to the belief that – in hypomania and melancholia – one was seeing prestige *ornaments*, sensu Zahavi (1975); emblems which predicated social engagement and disconnection. Naomi’s account of her illness was illustrative of the “sociology” of bipolar symptoms. Similar exploration of the accounts of bipolar depression suggested that it was not principally about simple loss, but about loss of prestige. Buss (2012) had noted that evolution sometimes produced adaptational "byproducts" and it seemed that some bipolar spectrum sequelae could well fall into this category. In this chapter the design of the research and the methods of analysis will be described.
Method

Several preliminary clinical scales pertinent to prestige were developed in a private practice context and the results were compared to those of friends and family; the impressionistic findings were compatible with the provisional theories as they developed. The context at hand for fully fledged research was a hospital and community mental health service which did not have a strong history of research. The option of an experience sampling method, employed longitudinally, was attractive, though the theoretical domain lacked for empirical support for any theory. Furthermore, without a sound theoretical construct, it was unclear to what extent idiographic methods may be generalisable. An experimental method was not chosen, as the various scales had not been previously tested and randomisation to prestige status was not an option. As such, it was decided to adopt a relatively simple and unobtrusive method of data collection, one which could involve all of the clinical staff. Therefore, a case-control method was selected, which fitted with a largely exploratory approach, in which a small number of confirmatory hypotheses may be applied. There was little direct research or literature to refer to and therefore the outcome of this research needed to be preliminary (USC-Libraries, 2012). The designation of the seven bipolar spectrum groups would allow for a comparative method, which was encompassed within the Structural Model. Correlational linkage between the various measures of mood and prestige were assayed within the Dynamic Model.

Research Question

The principal question was formulated as:

*Can the concept of prestige be provisionally applied to explain the biopsychosocial pathogenesis of bipolar spectrum conditions?*

Research Propositions

Structural Model

The research at hand indicated that the bipolar disorders were based on temperament and shaped by life events and psychology in the context of social
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support. A review of the history of the bipolar spectrum suggested that a nodal-dimensional approach may provide a useful model. Preliminary scale analysis indicated that prestige approach motivation may constitute the dimension of bipolarity. Accordingly, affective disorders were mooted to fall into a spectrum of nodes, which have an evolutionary origin pertaining to prestige maintenance. These groups were differentiated, not only on clinical grounds, but on genetic and psychometric (MDQ) bases also. The proposed structural model is depicted in Table 6.

<table>
<thead>
<tr>
<th>Node</th>
<th>Clinical grouping</th>
<th>Descriptor</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1</td>
<td>Bipolar I</td>
<td>Bipolar with a history of hospitalisation or psychosis</td>
</tr>
<tr>
<td>S2</td>
<td>Bipolar II</td>
<td>Bipolar with no hospitalisation or psychosis</td>
</tr>
<tr>
<td>S3</td>
<td>Pseudounipolar</td>
<td>Unipolar depression with a positive bipolar family history</td>
</tr>
<tr>
<td>S4</td>
<td>Subthreshold</td>
<td>MDQ score 3-6 or residual mood scale hypomania</td>
</tr>
<tr>
<td>S5</td>
<td>Simplex</td>
<td>A family history of bipolar disorder with no documented illness</td>
</tr>
<tr>
<td>S6</td>
<td>Unipolar</td>
<td>Unipolar depression with no bipolar family history</td>
</tr>
<tr>
<td>S7</td>
<td>Control</td>
<td>Nil affective illness, nil bipolar subthreshold symptoms, nil bipolar family history</td>
</tr>
</tbody>
</table>

Note. MDQ = Mood Disorder Questionnaire

This model will be evaluated in the Results and elaborated on in the Discussion. It is proposed that meaningful differences in prestige and affective variables exist by reason of their spectrum location and that a gradient of prestige approach motivation occurs from the S1 to S7 node.
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Dynamic Model

The Prestige Model of Spectrum Bipolarity draws on the concept that individuals garner prestige by the investment of the group (Price et al., 2007). The sociometer, as described by Leary et al. (1995), determines the social inclusion significance of events and relational value is internalised to self-esteem (Figure 1) as part of a both a normative and pathological process. In bipolar spectrum conditions, self-esteem is unstable, through the aegis of inherited and developmental factors. Events, which are relevant to a person’s perceived social inclusion, perturb neural self-esteem and generate tension (the tension term being added post hoc). Depending on context and disposition, tension may generate approach and withdrawal states. In the former the individual interacts with objects in order to drive up prestige approach motivation and subsequently prestige. Thereby the self-esteem sociometer (Leary et al., 1995) is normalised and relational value re-established. In withdrawal states the onus is on a holding pattern of prestige within a reduced range (prestige withdrawal) to escape social risk and avoid ostracism (refer Allen & Badcock, 2003). Adaptive versions of bipolarity developed to maintain prestige-self-esteem homeostasis in a challenging ancestral social environment, where marginality was incipient. Evolution has seen the origin of aberrant byproducts such as psychosis, mania, inanition and suicidality, which have been “carried along” with the adaptations (after Buss, 2012). I propose that the model (Figure 9) can be shown to have construct validity by means of correlational and path model testing.
Figure 9. Dynamic model of relationships between prestige, self-esteem, tension and affective variables. The model posits that prestige-related events impact self-esteem and prestige, affecting tension. Tension in turn drives prestige approach motivation and withdrawal symptoms, which have effects on prestige.
Specific Hypotheses

Structural Model
1. The measure of bipolar I (S1) prestige approach motivation (MSPaM) is significantly greater than controls (S7)
2. As for 1. for 2a. bipolar II (S2), 2b. pseudounipolar (S3), 2c. subthreshold (S4)
3. The presence of a bipolar family history is reflected in a higher prestige approach motivation in the simplex (S5) group than the controls (S7)
4. Unipolar illness (S6) has a lower prestige approach motivation (MSPaM) than bipolar illness (S1,S2)
5. The pseudounipolar (S3) group will have a higher prestige (SIPS) than the unipolar group (S6)

Dynamic Model & Various
6. Dimensional bipolarity, as measured by the sum of the Mood Disorder Questionnaire (MDQ), correlates significantly with prestige approach motivation (MSPaM) in the total cohort
7. Prestige approach motivation (MSPaM) will correlate with income, education and work status in the control (S7) group
8. Hypomania (ASRM) is associated with an elevation in prestige (nlogSIPS)
9. Depression (PHQ-9) is associated with a reduction in prestige (nlogSIPS)
10. Prestige approach motivation (MSPaM) covaries with bipolar (S1) illness severity (hospitalisation index)

Sample
The Measurement of Prestige Factors (MOPF) research was an observational case control study of adults 18 to 65 years of age diagnosed with bipolar disorder or depression, combined with a general community group of subjects. It was conducted over twelve months in an area mental health service, where participants had a diagnosis of major depression or bipolar disorder, as noted by consultant psychiatrists. Health service-seeking community participants were sought at a nearby general
practice over consecutive days. Participants completed either an online or paper survey, which were visually identical. Sporadic single item missing data was managed through the scale items being averaged to a scale total, though where more than 40% of the fields were missing the item was deleted. Cases with completely missing MDQ or spectrum values, or missing multiple scales, were deleted and 228 of 241 total surveys were found valid. Cases were allocated to one of seven nodes within a bipolar spectrum (Table 6) – reflecting a bipolar *enriched* sample.

**Measures**

*Diagnosis*: persons may elect “no psychiatric disorder”, “bipolar disorder”, “depression”, or “another psychiatric condition” (which is specified by text entry). Algorithms allocate persons to a final spectrum category, depending on stated diagnosis, Mood Disorder Questionnaire (MDQ) score, bipolar family history, mood elevation score and current state depression (to detect undiagnosed depressive cases). The distinction between bipolar I and II was made on the basis of hospitalisation or reported psychosis and is therefore an approximation. See Diagnostic Algorithm, Spectrum Calculator (hierarchical) in the Appendix.

The *Mood Disorder Questionnaire* (MDQ) (Hirschfeld et al., 2000) was used in screening for bipolar disorder and as a dimensional measure of lifetime bipolarity. It has a sensitivity of 58.0% and a specificity of 93.0% for diagnosis in a primary care depression population (Hirschfeld, Cass, Holt, & Carlson, 2005). Its range is 0-13 and the (hypo)manic symptom cut-off is ≥ 7 when accompanied by symptom coexistence and severity criteria. MacIntyre (2013) has used the MDQ as a valid dimensional measure of bipolarity and in particular has applied the measure in correlation studies.

The *PHQ-9* (Spitzer, Kroenke, & Williams, 1999) is a nine item self administered scale – the range being 0-27 and the moderate depression (or greater) cut-off score being ≥ 10 – which utilises DSM-III-R and DSM-IV criteria for major depression. It has a sensitivity of 88% and a specificity of 88% (Kroenke, Spitzer, & Williams, 2001).
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

The *Altman Self Rating Mania Scale* (ASRM) (Altman, Hedeker, Peterson, & Davids, 1997) is a five item scale each with five Likert points – the score range being 0-20 with the (hypo)manic cut-off being $\geq 6$ – and was used to assess current state mood elevation. It has a sensitivity of 87.3% and a specificity of 85.5% for mania or hypomania (Altman et al., 1997).

The *SISE* (Robins, Hendin, & Trzesniewski, 2001) measure is a five point single item Likert scale with a range 1-5; it was used to assess global self-esteem. It has a high convergent validity with the Rosenberg Self Esteem scale in adults (Robins et al., 2001).

A 7 point Likert TENSION scale – designed for the study – measured subjective tense arousal (construct from Thayer, 1989). Its range is 1-7, where 7 represents strong agreement with feeling tense. Despite the fact that the arousal (SMAS) system is deemed bivalent in extremis, this scale is unidimensional. States of anger and fear have a common archaic dimension as seen in the fight, flight and freeze response.

The 20 item, 7 point Likert, Prestige Approach Motivation Scale (MSPaM) (Appendix) measured the motivated drive to improve social investment (prestige) through the pursuit of social strategy, attractiveness, reputation, social information, possessions, creativity and goal achievement. It has a range of 20-140. The scale has a Cronbach’s alpha of 0.93 and its convergent validity was tested by correlation with the CSW.av (Contingencies of Self-worth) scale – which measures the average extent to which self-worth is contingent on approval, appearance, and competition – conceptually related to the pursuit of prestige. This found a correlation of $r_p = 0.58$ (N = 228) – therefore, a moderate positive correlation between these scales and their constructs was supported.

The three item *Social Inclusion and Prestige scale* (SIPS) captured data about community prestige. It is a three item, five point numerical scale which asks participants to judge the extent to which the community invests in them; for instance: “I receive the following number of birthday wishes each year”. Each item had 5
categories comprising of a range of numerical values. The mid-point of each selected range was used as the numerical response for that item then the three item responses were summed. The measure was log transformed due to a heavily right-skewed distribution \(\text{nlogSIPS}\), giving a possible range of 4.5 to 108, where higher scores represent higher prestige. The scale has a Cronbach’s Alpha of 0.76.

The author’s \textit{MOPF Social Inclusion Sensitivity (MSIS)} scale measures responsiveness to social events which centre on social inclusion and exclusion; for instance: “I feel valued when people seek my company” or “When people don’t value me I often feel despair”. It is a five item scale with a seven point Likert format and a range of 5 to 35 and a Cronbach’s alpha of 0.80.

Following ethological notions inspired by Maier and Schneirla (1964), cases were allocated to one of two groups for the purposes of the dynamic model testing; those with a euthymic or (hypo)manic mood category were deemed \textit{Approach}, while those remaining cases with a syndromal (PHQ-9 \(\geq 10\)) or mild depressed mood (PHQ-9 5-10) were deemed \textit{Withdrawal}. The cases with both categorical mood elevation and depression (manic-mixed) \((n = 9)\) were excluded from this analysis.

\textit{Mood category} (MOODcat) was used to reflect the individual’s cross-sectional mood state. It draws on the ASRM and the PHQ-9; persons scoring below both of the scale-designated thresholds were deemed to be euthymic. Responses reflecting a suprathreshold ASRM (6 or greater) (Altman et al., 1997) mood elevation – with a subthreshold PHQ-9 – were considered (hypo)manic. Individuals who scored 10 or greater on the PHQ-9 (Spitzer et al., 1999) – with a subthreshold ASRM – were deemed syndromally depressed. If responses were suprathreshold on both the ASRM and PHQ-9 scales then the mood category was assigned as “mixed”, which in DSM-5 (American-Psychiatric-Association, 2013) terms would be conceived of as mania with mixed features. A PHQ-9 score 5 or more, and less than 10 – with subthreshold ASRM – indicated “mild depression” (Spitzer et al., 1999).

The \textit{Contingencies of Self-Worth Scale (CSW)} (Crocker, Luhtanen, Cooper, & Bouvrette, 2003) mensurates the extent to which self-worth is contingent on prestige
variables such as competition and attractiveness. It is a long scale and for the purposes of this research, only three of the seven domains were included; the external contingencies of appearance, competition and approval from others (with J. Crocker’s consent). The researcher’s short-form obtains the average of these three subscales, which are in turn an average of the five individual item scores which they incorporate. The average ranges from 1 to 7. As of 2010 it had not been tested in bipolar populations.

Perceived Childhood Relational Trauma was measured as a binary “yes/no” response to the statement “I suffered abuse or neglect as a child:”. It is a broad response which is open to interpretation and retrospective bias, as well as the effects of mood. The two childhood traumas are concatenated in order to attempt to capture a wide range of relational difficulties, where the subjective element is given precedence.

All scales may be found at http://tinyurl.com/3rqwfa8.

Ethics

The invitation was dispensed as part of a routine consultation by the clinician (care coordinator, mental health doctor and general practitioner) and informed consent was obtained. It was noted that it is a completely voluntary activity for the consumer, but one that will help mental health research. With the exception of the author, dispensing clinical staff were not involved in the design or analysis of the survey. It was emphasised that if subjects felt any distress from doing the questionnaire, they should feel free to approach the clinician, the researchers or the relevant Ethics committees, as described in the Plain Language Statement (PLS). It was indicated that the questionnaire formed part of research undertaken into the causes of mental conditions, as part of the principal researcher’s PhD. Participants were encouraged to read the PLS and to consider opening the link to the questionnaire or complete the paper survey. An invitation to ask any questions of the clinician or via the research email was made. Comprehension of the information provided was assessed.

The PLS was sited at the introduction of the paper and web versions of the survey, as are contact details for the researcher. Subjects then followed questions and
indicated responses, moving through the 15 minute survey. At completion they were thanked for their contribution and advice was given on the best steps to take if distress ensues. The contact details of the researchers were again given. For persons without the possibility of access to a computer, a paper version of the survey was available. The study was approved by the Human Research and Ethics Committees of the University of Melbourne and Peninsula Health. Verbal informed consent was obtained prior to each survey and data analysis was undertaken anonymously.

Statistical Management

Data was analysed using Minitab. A two tailed alpha of .05 was selected as the criterion for significance.

Data Collection

Data was collected by means of a web link, or through the completion of a paper version of the survey. Data was entered to SurveyMonkey via a secure password protected portal. SurveyMonkey offers high security with data protection, including SSL technology. If the survey was undertaken via the web, the IP address was not stored or downloaded. No names were collected. Cookies were stored on web users’ computers. Survey responses were kept in strict confidence and used in compliance with legal requirements. The author remains the controller and owner of the data collected. Web data were downloaded to the author’s computer and stored in Excel and Minitab databases. The anonymous data were shared with members of the research team only as needed for purposes of analysis. The data will be stored for fifteen years. Research findings may be obtained by participants through emailing the author.

Data Manipulation

Sporadic single item missing data were managed through the scale items being averaged to a scale total, though where more than 40% of the fields were missing the item was deleted. If cases had missing MDQ or PHQ-9 scales then the case was deleted, as these items weighed heavily in categorisation. Cases with no Spectrum diagnosis were deleted from the cohort. Several scales were found to be non-normally
distributed on inspection of their probability plot, these being the ASRM, the PHQ-9 and the SIP scale – in several regressions these were natural log transformed. The SIP scale was modified by means of a natural log transformation (nlogSIPS). Boxplots for the scales were obtained for each Spectrum node and one MSPaM, two MDQ and two PHQ-9 univariate scale outliers were identified and reassigned to two deviations from the mean, as described by Field (2009). The data were analysed using Minitab and a two tailed alpha of .05 was selected as the criterion for significance.

**Interim Analysis**

This was undertaken at two points in data collection – once thirty persons with bipolar disorder were enrolled and again in October 2012, prior to the study finishing at year’s end. The data were not cleaned at the first instance and rough hypothesis tests were run. The October analysis allowed for a mock run of data cleaning and a closer review of hypotheses.

**Outcome Analysis**

Data was analysed separately for the Structural Model and the Dynamic Model. A demographic analysis was undertaken. Thence, with respect to the *Structural Model*, analysis of hypotheses 1 and 2 was by means of ANCOVA with a single factor SPECTRUM and the covariates of ASRM and PHQ-9. The difference in MSPaM between the nodes was ascertained in post hoc tests. A T-test for MSPaM was undertaken on the combined S5 and S7 cohort (hypothesis 3) as well as the combined S3 and S6 group, these pairs differing only on the presence of a bipolar family history. Any difference in prestige (nlogSIPS) between the pseudounipolar (S3) and unipolar (S6) nodes (hypothesis 5) was investigated by means of a T-test on the extended depressive cohort. The correlation of the MDQ with MSPaM (hypothesis 6) was tested as part of the Dynamic model correlational analysis.

The overall method is consistent with that advanced in the HREC research protocol, where T-tests as well as ANOVAs were advocated for comparing illness groups for MSPaM. A number of proposed research protocol statistical analyses were subsequently found to be flawed in design, due to the author’s inadequate knowledge of statistics at the time, though it was possible to achieve their intentions via different
statistical means. Various metrics established whether or not there was an association between MSPaM and age, sex, illness duration and bipolar I illness severity. An MSPaM factor analysis was undertaken. A T-test sought to establish any relationship between MSPaM and the presence of psychosis in the combined bipolar group.

Following ethological notions inspired by Maier and Schneirla (1964), cases were allocated to one of two groups; those with a euthymic or (hypo)manic mood category were deemed *Approach*, while those remaining cases with a syndromal (PHQ-9 ≥ 10) or mild depressed mood (PHQ-9 5-10) were deemed *Withdrawal*. The cases with both categorical mood elevation and depression (manic-mixed) \( n = 9 \) were excluded from this dynamic model analysis as their polarity was mixed. Contour plots detailing the relationships between prestige, MSPaM, depression (PHQ-9) (Figure 35) and mood elevation (ASRM) (Figure 36) were drawn.

Hypothesis 7, that prestige approach motivation (MSPaM) would correlate with social outcome, was tested by Spearman correlation in the control (S7) group. Because the work field was not ordinal it was excluded from analysis. Hypotheses 8 and 9, relating to mood and prestige, were tested for the total spectrum cohort by means of an ANOVA of MOODcat against the nlogSIPS measure. Hypothesis 10 was analysed by means of a Pearson correlation. A number of exploratory analyses will also be presented.

The measures were all treated as continuous and produced acceptable residuals in simple and multiple regressions. The data was subject to path analyses (Figure 10), which entail estimation of the strength of relationship between three variables in a putative causal chain, such as Independent Variable (IV) – Mediating Variable (MV) – Dependent Variable (DV). In a valid path the IV predicts the DV (path C). With the addition of the MV it can be shown that this entity explains the relationship between the IV and DV (path C’), where the C’ relationship strength, compared to path C, falls. In partial mediation, the C’ path remains statistically significant, whilst in full mediation the path becomes non-significant with the addition of the MV. Further criteria are discussed by Preacher and Leonardelli (2013); the path between the IV-MV (path A) should be significant, as should the relationship between
the MV and DV (path B). The mediation effect can be further tested by the application of the Sobel (1982) test.

Figure 10. Schematic of path relationships in mediation analysis.

Conclusions

The Prestige Model of Spectrum Bipolarity represents a bold attempt to examine affective disorder – one which, on the face of it, is likely to be falsified. It draws on a number of hypotheses, based around structural and dynamic models, augmented by exploratory methods. Its correlational claims are testable through extant and new instruments, assaying mood and prestige variables.
Chapter 7: Results

Demographics

At the conclusion of the study 228 usable responses from 241 surveys were received with an estimated return rate of over 60%. Table 6 documents the criteria which were used to assign individuals to spectrum nodes, while Table 7 shows demographic data from the survey. The allocation criteria will be discussed further in the Discussion. It can be seen that individuals with bipolar I (S1) and unipolar (S6) disorders were marginally more commonly female, though overall there was no significant overrepresentation by sex. The age for S1-S4 was somewhat lower than those for S5-S7, suggesting an effect of recruitment from general practice for the latter group. The bipolar I (S1) node had a longer duration of illness than the unipolar (S6) patients with a mean difference of 6.48 years; 95% CI [0.81, 12.16], p = .026, d = 0.56. The bipolar I (S1) node had an average of 7.12 lifetime hospitalisations compared to the unipolar (S6) mean of 0.12. Compared to controls (S7), bipolar I (S1) individuals were more likely to be single, $\chi^2(1, n = 101) = 19.64, p < .001$, Cramer’s V = 0.45 and earn less than $50,000 per annum, $\chi^2(1, n = 101) = 7.43, p = .006$, Cramer’s V = 0.28. Bipolar I (S1) individuals were slightly more likely to be unemployed than controls (S7); $\chi^2(1, n = 101) = 3.91, p = .048$, Cramer’s V = 0.20. Of note, there was no significant difference in educational levels across the spectrum, suggesting that the aforementioned indicators of social disadvantage may represent illness “drift”.

For the 53 bipolar I (S1) participants 52 (98%) arose from the mental health population – where clinical diagnosis was applied for case selection – and 46 (87%) of the S1 cohort self-indicated a bipolar diagnosis. 48 (91%) of the S1 group endorsed a suprathreshold MDQ score (indicating likely bipolar disorder) – including 1 subject with a self-nominated “no psychiatric disorder” and 6 (all arising in the mental health population) persons who indicated a diagnosis of “depression”. Forty six of 51 (90%) of S1 responses indicated they had been hospitalised, while 31 (61%) said they had been told they were psychotic when most ill. Control subjects – on the other hand –
indicated “no psychiatric disorder”, scored below 3 on the MDQ and were non-cases on the ASRM and the PHQ-9 and had no bipolar family history.

The cross-tabulation of spectrum node by mood state is depicted in Table 61 (Appendix). This found that a large number of S1, S2 and S3 individuals were depressed, while S4 was finely balanced between euthymia and (hypo)mania. Depressive states were common in the unipolar (S6) node. Though largely euthymic, nine of forty seven controls had mild depression.

Table 7  Demographic Features of the Seven Spectrum Nodes

<table>
<thead>
<tr>
<th></th>
<th>S1</th>
<th>S2</th>
<th>S3</th>
<th>S4</th>
<th>S5</th>
<th>S6</th>
<th>S7</th>
<th>Stat.</th>
<th>P value</th>
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<tbody>
<tr>
<td>N</td>
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<td>24</td>
<td>17</td>
<td>32</td>
<td>17</td>
<td>37</td>
<td>48</td>
<td>37.88(\alpha)</td>
<td>&lt; .001</td>
</tr>
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<td>Gender</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>19</td>
<td>12</td>
<td>7</td>
<td>13</td>
<td>7</td>
<td>11</td>
<td>22</td>
<td>3.66(\beta)</td>
<td>.720</td>
</tr>
<tr>
<td>Female</td>
<td>31</td>
<td>11</td>
<td>10</td>
<td>19</td>
<td>10</td>
<td>25</td>
<td>25</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, median range</td>
<td>36-45</td>
<td>36-45</td>
<td>26-35</td>
<td>36-45</td>
<td>46-55</td>
<td>46-55</td>
<td>46-55</td>
<td>56.07(\gamma)</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Years illness, mean (SD)</td>
<td>17.9 (11.6)</td>
<td>18.4 (13.5)</td>
<td>12.6 (12.6)</td>
<td>35.7(^a) (28.9)</td>
<td>11.4 (-)</td>
<td>11.5 (-)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N hospitalisation, mean (SD)</td>
<td>7.12 (13.5)</td>
<td>0 (0)</td>
<td>0.36 (0.63)</td>
<td>0.12 (0.60)</td>
<td>0 (0.63)</td>
<td>0 (0)</td>
<td>130.54(\gamma)</td>
<td>&lt; .001</td>
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<tr>
<td>Marital status</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married/defacto</td>
<td>17</td>
<td>11</td>
<td>8</td>
<td>18</td>
<td>12</td>
<td>17</td>
<td>37</td>
<td>22.92(\alpha)</td>
<td>.001</td>
</tr>
<tr>
<td>No relationship</td>
<td>33</td>
<td>12</td>
<td>9</td>
<td>14</td>
<td>5</td>
<td>19</td>
<td>10</td>
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<td></td>
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<td>Income</td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>&lt; $50000</td>
<td>36</td>
<td>13</td>
<td>12</td>
<td>13</td>
<td>8</td>
<td>21</td>
<td>20</td>
<td>12.58(\alpha)</td>
<td>.050</td>
</tr>
<tr>
<td>&gt; $50000</td>
<td>14</td>
<td>9</td>
<td>5</td>
<td>19</td>
<td>8</td>
<td>15</td>
<td>25</td>
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<td></td>
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<tr>
<td>Employment</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gainful</td>
<td>23</td>
<td>15</td>
<td>12</td>
<td>29</td>
<td>15</td>
<td>22</td>
<td>31</td>
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<td>8</td>
<td>5</td>
<td>3</td>
<td>2</td>
<td>13</td>
<td>16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Part high school</td>
<td>21</td>
<td>11</td>
<td>8</td>
<td>5</td>
<td>7</td>
<td>14</td>
<td>14</td>
<td>13.41(\alpha)</td>
<td>.340</td>
</tr>
<tr>
<td>High school/tech.</td>
<td>20</td>
<td>8</td>
<td>7</td>
<td>15</td>
<td>7</td>
<td>13</td>
<td>23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>University/postgrad</td>
<td>8</td>
<td>4</td>
<td>2</td>
<td>12</td>
<td>3</td>
<td>8</td>
<td>9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Collector</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>General practice</td>
<td>1</td>
<td>9</td>
<td>8</td>
<td>32</td>
<td>17</td>
<td>21</td>
<td>48</td>
<td>145.23(\gamma)</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Mental hlth service</td>
<td>52</td>
<td>15</td>
<td>9</td>
<td>0</td>
<td>0</td>
<td>16</td>
<td>0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note.  \(\alpha\) chi-square statistic, \(\beta\) F statistic, \(\gamma\) Kruskal-Wallis test
The S4 years of illness mean is based on only 3 individuals with other psychiatric conditions concurrently
Structural Model

The standardised mean scores of MSPaM and the Mood Disorder Questionnaire (MDQ) were plotted against Spectrum (Figure 11) to create a comparison of prestige approach motivation and a measure of dimensional bipolarity. This found that both plots were negatively sloped and roughly parallel. The Pearson correlation of MDQ and MSPaM was 0.47 (N = 228) in the complete spectrum cohort. While no statistical conclusions can be drawn from this result, it relates prestige approach motivation to dimensional bipolarity – supporting metrics determined elsewhere. It should be noted that MDQ scores were used in the spectrum calculator algorithm to allocate cases and that the slope of MDQ is therefore determined somewhat *a priori*.

*Figure 11.* Standardised plot of mood disorder questionnaire (MDQstd) and prestige approach motivation (MSPaMstd) by spectrum.
**ANCOVA on Prestige Approach Motivation (MSPaM)**

An ANCOVA on prestige approach motivation (MSPaM), with Spectrum as the principle factor and depression (PHQ-9) and mood elevation (ASRM) as covariates was run in the spectrum cohort. As is the case for all the highlighted results, the residuals of the ANCOVA were normally distributed and their variance constant. The descriptive and inferential statistics are shown in Table 8, Table 9 and Figure 12. SPECTRUM contributed moderately to MSPaM ($F(6,214) = 4.95$, $p < .001$), as did depression (PHQ-9) ($F(1, 214) = 4.80$, $p = .029$). Mood elevation (ASRM) contributed more significantly; ($F(1, 214) = 7.08$, $p = .008$). The predictors together accounted for 26.92% ($r^2_{adj} = 24.18\%$) of the variability in MSPaM. Therefore, after controlling for mood variables, bipolar I (S1) subjects were found to have a significantly higher mean MSPaM than controls (S7), with a large Cohen’s d of 1.05 – confirming hypothesis 1. The bipolar II (S2) node scored significantly higher than controls, with a large Cohen’s d of 0.84 – confirming hypothesis 2a. With an $n$ of only 17, the pseudounipolar (S3) node had a score 16.98 points higher than controls – of marginal statistical significance ($p = .051$) – but with a large Cohen’s d (0.86) – supporting hypothesis 2b. The other spectrum nodes did not have significantly higher MSPaM scores than controls and hypothesis 2c (regards S4>S7) was therefore rejected.

Hypothesis 4 argued that MSPaM for the unipolar (S6) node would be lower than that of the S1 (bipolar I) and S2 (bipolar II) nodes. The S1 node scored 15.37 points higher than the S6 node – 95% CI [3.41, 27.34], $p = .003$, $d = 0.80$, while the S2 node scored 10.93 points higher than S6 – 95% CI [-3.86, 25.73], $p = .300$, $d = 0.58$. Thus hypothesis 4 is supported for the bipolar I but not for bipolar II node.

The descriptive statistics for all of the scales used are found in Table 19 and Table 20 (Appendix).
## Table 8  Descriptive Statistics for Prestige Approach Motivation (MSPaM) – Spectrum Cohort (raw scores)

<table>
<thead>
<tr>
<th>Spectrum</th>
<th>n</th>
<th>Mean</th>
<th>Standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1</td>
<td>53</td>
<td>90.05</td>
<td>16.22</td>
</tr>
<tr>
<td>S2</td>
<td>24</td>
<td>84.72</td>
<td>19.82</td>
</tr>
<tr>
<td>S3</td>
<td>17</td>
<td>85.65</td>
<td>15.37</td>
</tr>
<tr>
<td>S4</td>
<td>32</td>
<td>78.40</td>
<td>16.37</td>
</tr>
<tr>
<td>S5</td>
<td>16</td>
<td>78.77</td>
<td>18.01</td>
</tr>
<tr>
<td>S6</td>
<td>37</td>
<td>72.88</td>
<td>19.15</td>
</tr>
<tr>
<td>S7</td>
<td>48</td>
<td>61.83</td>
<td>23.97</td>
</tr>
</tbody>
</table>

## Table 9  Inferential Statistics for ANCOVA Prestige Approach Motivation (MSPaM)

<table>
<thead>
<tr>
<th>Spectrum</th>
<th>Estimate</th>
<th>95% Confidence Interval</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1 minus S7</td>
<td>21.17</td>
<td>[8.16, 34.18]</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>S2 minus S7</td>
<td>16.73</td>
<td>[0.92, 32.54]</td>
<td>.030</td>
</tr>
<tr>
<td>S3 minus S7</td>
<td>16.98</td>
<td>[-0.03, 34.00]</td>
<td>.051</td>
</tr>
<tr>
<td>S4 minus S7</td>
<td>11.90</td>
<td>[-1.39, 25.19]</td>
<td>.112</td>
</tr>
<tr>
<td>S5 minus S7</td>
<td>13.08</td>
<td>[-3.12, 29.29]</td>
<td>.202</td>
</tr>
<tr>
<td>S6 minus S7</td>
<td>5.80</td>
<td>[-7.15, 18.75]</td>
<td>.836</td>
</tr>
</tbody>
</table>

Note. Depression (PHQ-9) and mood elevation (ASRM) are controlled for.
MSPaM Factor Analysis

The full MSPaM scale has a Cronbach’s Alpha of 0.93. An exploratory factor analysis was undertaken to examine the underlying structure of the scale and to determine whether there was a preliminary basis for the creation of subscales. Principal components analysis yielded 4 components with an eigenvalue greater than 1 and these components accounted for 62% of the variance with component 1 accounting for 42% of the variance. Varimax rotation yielded 3 components with more than two items loading 0.6 or higher. Inspection of the items loading on to each component suggested that each component was coherent and meaningful in the light of the theoretical considerations guiding this study. I labeled these components: NEGESTAFF – negative esteem affect (7 items), POSESTAFF – positive esteem affect (5 items) and POSESTACT – positive esteem action (5 items). The fourth component contained insufficient items with a high loading to warrant further consideration. Internal consistency of the 3 components was tested and in each case
Cronbach's alpha scores were consistent with adequate reliability for research purposes: The “negative esteem affect” scale yielded alpha of 0.87; the “positive esteem affect” scale yielded alpha of 0.82, while the “positive esteem action” scale yielded an alpha of 0.83. A correlation matrix for these sub scales yielded correlations in the range 0.62 to 0.68, indicating non-redundancy. The Guttman Split-Half Coefficient was 0.87. These findings suggest that, the full scale has sufficient internal consistency to warrant use of a total scale score and that there are three meaningful and psychometrically adequate subscales. The factor structure of the MSPaM was sound and the inter-item correlations were high in Table 59 (Appendix). It is also evident that the self-esteem (SISE) variable correlates with NEGESTAFF (negative esteem affect) – but not with the other factors – negative esteem being logically linked with low self-esteem. This, along with the 0.25 correlation of POSESTACT (positive esteem action) with ASRM mood elevation, supports the convergent validity of the MSPaM.

Figure 13 depicts the three MSPaM factors with reference to Spectrum node – Negative Esteem Affect (NEGESTAFF) had the largest gradient. It correlated more strongly with depression (PHQ-9) score ($r_p = 0.50$, $N = 228$) than POSTESTACT ($r_p = 0.18$, $N = 228$) and POSESTAFF ($r_p = 0.24$, $N = 228$). In binary logistic regression (Table 10) it was the only significant predictor of categorical bipolar disorder. It predicted dimensional bipolarity (MDQ), when depression and mood elevation were accounted for (Table 11). All predictors together accounted for 37.85% ($r^2_{adj} = 36.42\%$) of the variance in MDQ.
Figure 13. The MSPaM factors as depicted by spectrum node. NEGESTAFF = negative esteem affect, POSESTACT = positive esteem action, POSESTAFF = positive esteem affect. NEGESTAFF showed the greatest differential between bipolar I (S1) and controls (S7).
Table 10  Binary Logistic Regression into Bipolar Disorder (BD) diagnosis

<table>
<thead>
<tr>
<th>Predictor</th>
<th>OR</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>NEGESTAFF</td>
<td>1.13</td>
<td>[1.07, 1.19]</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>POSESTACT</td>
<td>0.99</td>
<td>[0.93, 1.06]</td>
<td>.793</td>
</tr>
<tr>
<td>POSESTAFF</td>
<td>0.99</td>
<td>[0.92, 1.06]</td>
<td>.769</td>
</tr>
</tbody>
</table>

Note. NEGESTAFF = MSPaM factors Negative Esteem Affect; POSESTACT = Positive Esteem Action; POSESTAFF = Positive Esteem Affect. Even strong continuous predictors are associated with modest-looking OR’s.

Table 11  Coefficients for Multiple Regression Analysis into Mood Disorder Questionnaire (MDQ)

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Coefficient</th>
<th>Standard Error</th>
<th>T Statistic</th>
<th>DF</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>0.67</td>
<td>0.88</td>
<td>0.77</td>
<td>217</td>
<td>.444</td>
</tr>
<tr>
<td>NEGESTAFF</td>
<td>0.13</td>
<td>0.04</td>
<td>3.38</td>
<td>217</td>
<td>.001</td>
</tr>
<tr>
<td>POSESTACT</td>
<td>0.14</td>
<td>0.05</td>
<td>2.78</td>
<td>217</td>
<td>.006</td>
</tr>
<tr>
<td>POSESTAFF</td>
<td>-0.10</td>
<td>0.05</td>
<td>-2.08</td>
<td>217</td>
<td>.039</td>
</tr>
<tr>
<td>Depression (PHQ-9)</td>
<td>0.16</td>
<td>0.03</td>
<td>4.88</td>
<td>217</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mood elevation (ASRM)</td>
<td>0.21</td>
<td>0.08</td>
<td>2.78</td>
<td>217</td>
<td>.006</td>
</tr>
</tbody>
</table>

Note. NEGESTAFF = MSPaM factors Negative Esteem Affect; POSESTACT = Positive Esteem Action; POSESTAFF = Positive Esteem Affect.
The Determinants of Prestige Approach Motivation (MSPaM)

A multiple regression analysis (Table 12) was undertaken on the spectrum cohort to investigate the measurable contribution of variables to MSPaM. SPECTRUM was a significant factor in the prediction of MSPaM score; in particular, membership of the S1 (bipolar I) node predicted an elevated MSPaM score compared to the control group (S7), while the unipolar (S6) node was significantly associated with a lowered MSPaM compared to the Control (S7) group. Tension, mood elevation (ASRM), MOPF Social Inclusion Sensitivity (MSIS) and Contingencies of Self-Worth (CSW.av) (adapted from Crocker et al., 2003) were significantly related to increases in MSPaM. The Contingencies of Self-worth scale measures the extent to which self-worth is contingent on appearance, competition and approval. MSIS – which measures the sensitivity of a person to social inclusion – had the largest positive impact on prestige approach motivation, such that an increase in ten units of MSIS corresponded to an increase in 16.6 units of MSPaM. The predictors accounted for 61.43% of the variability ($r^2_{adj} = 59.29\%$) in MSPaM.
### Table 12  Coefficients for Multiple Regression Analysis of MSPaM

<table>
<thead>
<tr>
<th>Predictor/Factor Level</th>
<th>Factor Reference Level</th>
<th>Coefficient</th>
<th>Standard Error</th>
<th>T Statistic</th>
<th>DF</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONSTANT</td>
<td></td>
<td>8.33</td>
<td>5.34</td>
<td>1.56</td>
<td>198</td>
<td>.120</td>
</tr>
<tr>
<td>SPECTRUM</td>
<td>S7</td>
<td></td>
<td></td>
<td></td>
<td>198</td>
<td>.005*</td>
</tr>
<tr>
<td>S1</td>
<td></td>
<td>5.60</td>
<td>2.27</td>
<td>2.47</td>
<td>198</td>
<td>.014</td>
</tr>
<tr>
<td>S2</td>
<td></td>
<td>4.02</td>
<td>2.80</td>
<td>1.43</td>
<td>198</td>
<td>.154</td>
</tr>
<tr>
<td>S3</td>
<td></td>
<td>2.25</td>
<td>3.21</td>
<td>0.70</td>
<td>198</td>
<td>.484</td>
</tr>
<tr>
<td>S4</td>
<td></td>
<td>-1.36</td>
<td>2.58</td>
<td>-0.53</td>
<td>198</td>
<td>.599</td>
</tr>
<tr>
<td>S5</td>
<td></td>
<td>1.69</td>
<td>3.23</td>
<td>0.52</td>
<td>198</td>
<td>.602</td>
</tr>
<tr>
<td>S6</td>
<td></td>
<td>-6.36</td>
<td>2.24</td>
<td>-2.84</td>
<td>198</td>
<td>.005</td>
</tr>
<tr>
<td>TENSION</td>
<td></td>
<td>2.68</td>
<td>0.69</td>
<td>3.89</td>
<td>198</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>ASRM</td>
<td></td>
<td>0.88</td>
<td>0.35</td>
<td>2.54</td>
<td>198</td>
<td>.012</td>
</tr>
<tr>
<td>PHQ9</td>
<td></td>
<td>-0.27</td>
<td>0.18</td>
<td>-1.48</td>
<td>198</td>
<td>.142</td>
</tr>
<tr>
<td>CSW.av</td>
<td></td>
<td>4.49</td>
<td>1.19</td>
<td>3.77</td>
<td>198</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>MSIS</td>
<td></td>
<td>1.66</td>
<td>0.23</td>
<td>7.20</td>
<td>198</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

*Note.* *Overall* P value for SPECTRUM, ASRM = Altman Self Rating Mania Scale; PHQ9 = Patient Health Questionnaire 9 (for depressive symptoms), CSW.av = Contingencies of Self-worth (external domains averaged); MSIS = MOPF Social Inclusion Sensitivity Scale.
Prestige Approach Motivation (MSPaM) Associations

There was no association between MSPaM and age in the spectrum cohort; $r_p = -0.063, N = 228$. MSPaM was not significantly associated with sex; the M-F difference in means following a T-test being 3.73, 95% CI [-2.16, 9.62], $t(176) = 1.25, p = 0.21$. In the combined bipolar I and II cohort, there was no association between illness duration and MSPaM score; $r_p = 0.074, n = 77$. It was hypothesised that prestige approach motivation (MSPaM) covaried with bipolar I (S1) illness severity (the ratio of $n$ hospitalisations over years of illness) in a “dose-response” fashion, yet a Pearson test found no linear correlation between the two variables, $r_p = 0.081, n = 51$. Thus, hypothesis 10 was refuted. The mean MSPaM for those with a psychosis history in the bipolar (S1 & S2) cohort was only 3.6 points higher than for those without, and a T-test of this showed that this was a non-significant effect; $t(70) = -0.90, 95\% \text{ CI } [-4.37, \text{ 11.48}], p = 0.37$.

The convergent validity of the MSPaM scale was tested by correlation with the CSW.av scale – which measures the average extent to which self-worth is contingent on approval, appearance, and competition – conceptually related to the pursuit of prestige. This found a correlation of $r_p = 0.58 (N = 228)$ – therefore, convergence between these scales and their constructs is supported. The abbreviated CSW.av scale was compared to its constituent subscales of approval, appearance, and competition and the range of correlations was 0.78 to 0.85.

Dynamic Model

The prestige dynamic model is depicted in Figure 9 and portrays a proposed feedback loop, linking input prestige with affective variables and ultimately linking back to prestige output. Correlations involving the dynamic stem involved the whole sample (minus the manic-mixed group), while those correlations involving only the approach and withdrawal segments were confined to those cohorts. The dynamics of Figure 9, reflected in Table 13, showed a positive correlation of prestige (nlogSIPS) and self-esteem (SISE). There was a moderate negative relationship between self-esteem and tension. Tension had a positive relationship with prestige approach motivation (MSPaM), which in turn correlated moderately with mood elevation in the
Approach group. There was a weak positive association of mood elevation (ASRM) with prestige in the approach group. TENSION had a strong positive relationship with depression (PHQ-9), while in the categorically depressed group depressive symptoms did not covary with prestige (nlogSIPS). On running an ANOVA of mood category into prestige (nlogSIPS) the mean prestige rating for categorically depressed persons (PHQ-9 ≥ 10) was 0.52 points lower than for euthymics, 95% CI [0.18, 0.86], $p < .001$, $d = 0.69$.

The overall Pearson correlation of depressive (PHQ-9) score with MSPaM was 0.34 ($N = 228$), though there was notably no correlation for the unipolar (S6) node ($r_p = 0.01$, $n = 37$). The otherwise significant $p$ values indicate that these relationships are unlikely to be due to chance and correlational support for the dynamic model is therefore obtained. The significant correlation of MDQ and MSPaM supported hypothesis 6. Figure 14 reveals the inverse relationship of depression and self-esteem across the bipolar spectrum.

Because the SIP scale is novel, there was no comparator to utilise. I contend that the SIPS range of items has content validity and that the relationship of prestige (nlogSIPS) to depression (PHQ-9) for the withdrawal group (Table 13) is consistent with predictions. Prestige (nlogSIPS) further correlates with mood elevation (ASRM) – $r_p = 0.25$ ($n = 113$) in the subset of individuals with a euthymic or (hypo)manic mood (approach group). Furthermore there is a correlation between prestige and self-esteem (0.35, $N = 228$), as predicted by Stevens and Price (2000).
Table 13  Correlations Between Variables in the Dynamic Model

<table>
<thead>
<tr>
<th></th>
<th>nlogSIPS</th>
<th>SISE</th>
<th>TENSION</th>
<th>MSPaM</th>
<th>MDQ</th>
<th>ASRM</th>
</tr>
</thead>
<tbody>
<tr>
<td>SISE</td>
<td></td>
<td>0.35***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TENSION</td>
<td></td>
<td>-0.25***</td>
<td>-0.47***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MSPaM</td>
<td></td>
<td>-0.07</td>
<td>-0.24***</td>
<td>0.49***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MDQ</td>
<td></td>
<td>-0.13</td>
<td>-0.31***</td>
<td>0.18</td>
<td>0.44***</td>
<td></td>
</tr>
<tr>
<td>ASRM</td>
<td></td>
<td>0.25*a</td>
<td>0.04</td>
<td>0.11</td>
<td>0.42*a</td>
<td>0.39***</td>
</tr>
<tr>
<td>PHQ-9</td>
<td></td>
<td>-0.08w</td>
<td>-0.52***</td>
<td>0.70***</td>
<td>0.40***</td>
<td>0.48***</td>
</tr>
</tbody>
</table>

Note. Refer to Glossary for terms. *Approach group correlations; wWithdrawal group correlations. Note that in the total cohort the Pearson correlation of nlogSIPS and PHQ-9 was -0.32*** All results are Spearman correlations; *p < .05, ***p < .001, referring to total cohort unless specified. Note that the correlation of MSPaM and MDQ in the total cohort is 0.47.

Figure 14. Self-esteem (SISE) score compared with depression (PHQ-9) score by Spectrum node. It can be seen that for all nodes depression and self-esteem have an opposed relationship. PsUni = pseudounipolar, SubTh = subthreshold, Simplex = bipolar fhx (+).
Path Analysis in the Dynamic Model

A series of simple and multiple regression analyses were undertaken to test path relationships in the dynamic model. Table 21 indicated that SISE was a full mediator variable when modelling TENSION using nlogSIPS; once SISE was introduced into the model, the coefficient of nlogSIPS on TENSION is reduced to -0.21 from -0.57, with a Sobel z-value of -3.96 ($p < .001$) (Figure 16).

TENSION was a full mediator when modelling prestige approach motivation using self-esteem (SISE) (Appendix Table 22) with a Sobel test statistic of -5.01 ($p < .001$). TENSION was found to be a moderate-sized partial mediator when modelling depression (nlogPHQ-9) using self-esteem (SISE) (Table 23) – Sobel z-value -6.14 ($p < .001$). The natural log of PHQ-9 was used for this calculation due to non-constant variance in the residuals in the PHQ-9 regression. A significant relationship between self-esteem (SISE) and depression (nlogPHQ-9) score persisted despite the addition of tension as predicted by the model. Was depression score a full mediator when modelling prestige using tension, as the model predicted? A mediation analysis (Appendix Table 24) found this to be the case and the Sobel statistic was -3.14 ($p = .002$).

Examination of the full cohort did not find a statistically significant mediation effect for MSPaM when modelling prestige (nlogSIPS) using tension (Sobel z-value 0.28, $p = .780$). In the subset of (hypo)manic cases (Appendix Table 27, Figure 18) ($n = 23$), the $p$ value for the C component (tension into prestige) was only .212, indicating that tension had a non-significant effect on prestige. The Sobel z-value was non-significant at 0.93 ($p = .355$). It was, however, found that mood elevation (ASRM) significantly moderated the relationships of both TENSION (Appendix Table 25) and MSPaM (Table 26) with prestige (nlogSIPS) in the Approach cohort. Figure 15 shows that at an ASRM level of 10 – indicating moderate (hypo)mania – there was a positive relationship between tension and prestige as suggested by the dynamic model, while with an ASRM of 0 the reverse pattern was observed.
Figure 15. Fitted lines for prestige (nlogSIPS) value against TENSION at three levels of mood elevation (ASRM). This indicates a positive correlation of prestige and TENSION at an ASRM level of 10, while with an ASRM of 0 the reverse pattern is observed. (approach cohort).
Figure 16. Mediation model for self-esteem (SISE) modelling TENSION using prestige (nlogSIPS). Numbers indicated are standardised coefficients. *p = .143, ***p < .001.
Figure 17. Mediation models for TENSION, modelling MSPaM and depression (nlogPHQ-9) using self-esteem (SISE). Numbers indicated are standardised coefficients. * $p = .639$, ** $p < .001$; +++ indicates partial mediation.
Figure 18. Mediation models for MSPaM and depression (nlogPHQ-9), modelling prestige using TENSION. The right half applies to the total cohort, whereas the left half to those (hypo)manic. Numbers indicated are standardised coefficients. \( a \) \( p \) = .690 with addition of nlogPHQ-9 into regression, \( b \) \( p \) = .844 with addition of MSPaM, \( * p < .05, ** p < .01, *** p < .001 \).

**Sociometer Sensitivity**

The sociometer is a theoretical entity which measures relational value; it outputs through self-esteem to behaviours which seek to re-establish relational value. MOPF Social Inclusion Sensitivity (MSIS) and CSW.av conceptually mensurate relational sensitivity and respectively drive affect or self-worth – they are sociometer sensitive. It can be seen (Table 14) that the sociometer sensitivity variables (MSIS and CSW.av) do not correlate with prestige (nlogSIPS). Note that SISE self-esteem correlates negatively with CSW.av – to contexts where self-esteem is contingent on relational events. The table further reveals that these sociometer variables relate strongly to the dynamic model variables of tension, PHQ-9 and particularly MSPaM. There is a strong positive linear correlation between MSIS and CSW.av. The dynamic
model predicates that sociometer sensitivity is most marked for those with bipolarity – hence CSW.av ($r_p = 0.41$, $N = 228$) and MSIS ($r_p = 0.38$, $N = 228$) correlated positively with the MDQ – a result which parallels that for S1 in Figure 23.

The Behavioural Approach System (BAS) Reward Responsiveness subscale (Carver & White, 1994) seeks to measure the appetitive motivation toward desired objects such as success, gain, opportunity and winning, while the MSIS scale measures affective responsiveness to social inclusion, gain and loss. Is there any convergence between these scales? In order to evaluate this, the scale totals were subject to a correlation test – this found that $r_p = 0.41$, $N = 228$. This lends support to a convergence between these “reward” scales and their constructs.
### Table 14 Intercorrelations Between Sociometer and Dynamic Model Variables

<table>
<thead>
<tr>
<th></th>
<th>nlogSIPS</th>
<th>SISE</th>
<th>MSIS</th>
<th>CSW.av</th>
<th>TENSION</th>
<th>MSPaM</th>
</tr>
</thead>
<tbody>
<tr>
<td>SISE</td>
<td></td>
<td>0.35***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MSIS</td>
<td>0.05</td>
<td>-0.14*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CSW.av</td>
<td>-0.13</td>
<td>-0.45***</td>
<td>0.55***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TENSION</td>
<td>-0.25***</td>
<td>-0.47***</td>
<td>0.39***</td>
<td>0.42***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MSPaM</td>
<td>-0.10</td>
<td>-0.24***</td>
<td>0.70***</td>
<td>0.58***</td>
<td>0.47***</td>
<td></td>
</tr>
<tr>
<td>PHQ-9</td>
<td>-0.32***</td>
<td>-0.52***</td>
<td>0.29***</td>
<td>0.37***</td>
<td>0.66***</td>
<td>0.34***</td>
</tr>
</tbody>
</table>

Note. nlogSIPS = prestige; SISE = global self-esteem; MSIS = MOPF social inclusion sensitivity; CSW.av = average CSW score; TENSION = tension; MSPaM = Prestige Approach Motivation and PHQ-9 = depressive score. Pearson correlations, except where marked *S* Spearman correlations; *p* < .05, ***p* < .001.

To investigate possible mediation and moderation effects for the sociometer sensitivity variables – MSIS and CSW.av – on the dynamic model (Figure 9) a series of simple and multiple regression analyses were undertaken. The measures were all treated as continuous and produced acceptable residuals in simple and multiple regressions. Such treatment of Likert scales is supported by the statistician Norman (2010).

Appendix Table 28 documents the relationships between prestige (nlogSIPS) and self-esteem (SISE) with reference to the variables MSIS and CSW.av – neither of which was found to be a significant mediator at the .05 level of significance. CSW.av was, however, a significant predictor of self-esteem in its own right. CSW.av was found to be a partial mediator when modelling tension using self-esteem, with a significant Sobel z-value (Appendix Table 29). Both MSIS and CSW.av were partial mediators when modelling MSPaM using tension (TENSION) (Appendix Table 30, Table 31) and MSIS was also a moderator of this relationship, as shown by the significant interaction term. Neither MSIS nor CSW.av were mediators when modelling depression (nlogPHQ-9) using tension, though CSW.av was found to be a
moderator – revealed in the significant interaction term in Table 32 (Appendix) – this is depicted graphically in Figure 19. I have therefore demonstrated that sociometer (prestige) sensitivity has a pervasive influence on the function of the dynamic model stem and branches, through direct, mediating and moderating effects.

Figure 19. Depression (nlogPHQ-9) fitted line by TENSION at three levels of contingencies of self-worth (average) – CSW.av. The fits show that at high levels of the contingencies of self-worth scale (CSW.av = 6) there is a significantly stronger relationship between tension and depression than for low CSW.av.
Path Analysis for Bipolarity in the Dynamic Model

I have demonstrated that sociometer (prestige) sensitivity is implicit to the function of the dynamic model. I have also noted a correlation between MDQ bipolarity and the sociometer sensitivity variables. However, is the dynamic model directly affected by dimensional bipolarity? To investigate this possibility, simple and multiple regressions for MDQ as a potential mediating variable were undertaken. It can be seen, from the fall in coefficients and the positive Sobel tests, that dimensional bipolarity – as reflected in the MDQ – exerts a consistent role as a partial mediator through the complete dynamic model stem and branches (Appendix Table 33, Table 34, Table 35, and Table 36).

Sociometer Dynamics

Prestige and self-esteem were mapped by means of a line plot, comparing them against mood category (Figure 20), where it can be seen that they parallel each other; where prestige is high self-esteem tends to be high and the converse applies. The sociometer ratio is conceived as the ratio of self-esteem to prestige. With reference to prestige (as a social “given”) how much self-esteem is evident at each mood state? In euthymia the ratio is close to one, while in (hypo)mania self-esteem is lower than expected, as the SISE cannot register high self-esteem. In depression self-esteem level is disproportionately low relative to prestige. ANOVA found that there was no significant difference in sociometer ratio between the groups compared with Euthymics, except for the Depressive group versus Euthymics, where the 95% CI for the difference was [0.08, 0.50], \( p = .001 \) and \( d = 0.65 \). Therefore, following from the dynamic model, the given level of social inclusion/prestige in depression appears to be having a larger effect on self-esteem than in the other groups.

Figure 21 shows prestige and depression level by mood state. The Depressed group shows a disproportionate increase in depression relative to prestige level, when compared to Mild Depression where there is a proportionately equivalent prestige score. Figure 22 shows prestige and prestige approach motivation by mood state. While prestige and prestige approach motivation correspond in (hypo)mania, when prestige is low in the “Mixed” and Depressed states, the MSPaM is increased.
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

There is a peak for the MSPaM/Prestige ratio in the “Mixed” (manic) group and an elevated value for both depressive groups. An ANOVA (Appendix Table 37, Table 38) confirmed this; the “Mixed” (manic) and both depressed groups have ratios significantly greater than the Euthymic group. The Cohen’s $d$ for the “Mixed” group, relative to the Euthymic group was 1.71. The “Mixed” group had a higher MSPaM/Prestige ratio than the (Hypo)manic group by 22.65 points, the 95%CI being [9.21, 36.09], $p < .001$, $d = 1.83$.

A similar analysis was undertaken for spectrum versus sociometer ratio. When depression was added as a covariate, there was no significant difference in sociometer ratio between spectrum groups. The sociometer ratio was found to correlate with the contingencies of self-worth average (CSW.av) score at a Pearson score of -0.36 ($N = 228$). Therefore, the more self-esteem is contingent on prestige as measured by CSW.av, the lower is the sociometer ratio – indicating convergent validity.

![Figure 20](image.png)

*Figure 20.* Prestige (nlogSIPS) and self-esteem (SISE) level by mood state. It can be seen that the two trend together, but that in depression the self-esteem is disproportionately lowered relative to prestige.
Figure 21. Standardised line plot of prestige and depression scores across the five mood states. The figure shows that high depression scores are associated with low prestige in the depressed and manic (mixed) cohorts. The level of depression in these groups is disproportionate to prestige level when compared to the mild depression group, where prestige is roughly equivalent.
**The Prestige Model of Spectrum Bipolarity**

![Graph showing standardized data for mood states](image)

**Figure 22.** Standardised line plots of prestige and prestige approach motivation (MSPaM) by mood state. Prestige correlates roughly with prestige approach motivation in (hypo)mania, while prestige approach motivation is increased divergently to prestige in the two depressed states and the manic (mixed) category.

**Genetics and Development**

Does having a bipolar family history impact on MSPaM in those with no evidence of bipolarity (and no subthreshold symptoms)? This was explored in the extended “normal” respondents (S5 and S7), where a T-test (with no assumption of equal variance) found that the group with a familial bipolar history had a mean MSPaM of 78.8 while the non-familial group scored 62.6, the 95% CI for the difference being [4.45, 27.98], \( t(36) = 2.80, p = 0.008, d = 0.75 \). However, assay of MDQ status in the S5 group found that 11 of 17 of the cases had scores at, or greater than 3, all being non-MDQ cases due to absent symptom co-occurrence of severity criteria. In this node MDQ and MSPaM did not correlate \( (r_p = 0.11, p = .675, n = 17) \). Nevertheless, a T-test (with no assumption of equal variance) revealed that the S5 node had a significantly higher mood elevation score than the control (S7) node – the
95% confidence interval for the 2.5 point difference being [0.81, 4.15], \( t(18) = 3.11, p = .006, d = 1.00 \). Given the uncertainty of the S5-S7 MSPaM result, multiple regression (Table 63) was utilised to examine predictor variables for prestige approach motivation with a focus on family history. This found that family history did indeed predict MSPaM to a significant degree.

The extended normal group was also subject to a T-test which examined the difference in prestige (nlogSIPS) between those with a bipolar family history and those without. The mean nlogSIPS of the family history group was 0.245 higher than the non-family history group [-0.17, 0.66], \( t(34) = 1.20, p = .240 \). Therefore prestige was not higher in extended normals with a bipolar family history. A T-test (with no assumption of equal variance) for Social Inclusion Sensitivity (MSIS) was run on the pooled extended normal cohort (S5&S7). It was found that those with a positive bipolar family history had a mean MSIS 3.67 points higher than those without a bipolar family history – 95%CI [1.30, 6.03], \( t = 3.12, p = .003, d = 0.80 \). As noted, a bipolar family history was associated with an increased perception of childhood relational trauma (PCRT) \( \chi^2 (1, n = 181) = 20.82, DF = 1, p < .001 \) (Appendix Table 39).

A T-test was undertaken to investigate the relationship between perceived childhood relational trauma (PCRT) and depression score in the 228 case cohort. This found that persons with positive PCRT had a mean depression score of 12.34, while those not reporting PCRT had a mean score of 7.55 – the 95% CI of the difference [2.25, 7.34], \( t(189) = 3.72, p < .001 \) and \( d = 0.59 \). PCRT was associated with an increase in MSPaM ratings by 11.42 points – the T-test 95% CI for the difference being [5.17, 17.67], \( t(112) = 3.62, p < .001, d = 0.56 \). There was an association between PCRT and social inclusion sensitivity (MSIS) such that the mean score for the subjects claiming childhood relational trauma was 26.44 and that for those claiming no PCRT was 23.54, the difference in means having a 95% CI of [1.33, 4.48], \( t(122) = 3.65, p < .001 \) and \( d = 0.56 \). Table 40 (Appendix) shows a moderate overrepresentation of PCRT in individuals with the diagnosis bipolar disorder – but not in the depression or null diagnosis groups – with a \( \chi^2 \) statistic of (2, \( N = 189 \)) = 38.34, \( p < .001 \).
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

The issue of the contribution of PCRT to dimensional bipolarity was examined by a multiple regression of PCRT and depression score (PHQ-9) into Mood Disorder Questionnaire (MDQ) (Table 15). It can be seen that, when controlling for depression, childhood relational trauma had a major impact on dimensional bipolarity. PCRT and PHQ-9 together accounted for 25.45% ($r^2_{adj} = 24.66\%$) of the variability in MDQ with PCRT alone estimated to account for 12.33% of the explained variance.

Hypothesis 7 indicates that prestige approach motivation (MSPaM) will correlate with higher educational (7a) and financial (7b) advance in the control group (S7) on the premise that it is an adaptive trait. The Spearman coefficient for the relationship of MSPaM with education level was a weak 0.127, $n = 46$, $p = 0.400$, and the null hypothesis for 7a was therefore retained. A coefficient coefficient was computed to assess the relationship of MSPaM with income and no relationship between the variables was found, $r_s = 0.213$, $n = 45$, $p = .160$ and hypothesis 7b was rejected. It is conceivable that such a relationship may only become significant in a population based survey.
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

Table 15  Coefficients for Multiple Regression Analysis into MDQ

<table>
<thead>
<tr>
<th>Predictor/ Factor Level</th>
<th>Coefficient</th>
<th>Standard Error</th>
<th>T Statistic</th>
<th>DF</th>
<th>P value</th>
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<tr>
<td>Constant</td>
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<td>0.40</td>
<td>9.99</td>
<td>188</td>
<td>&lt;.001</td>
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<tr>
<td>PCRT (-)ve</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(+)ve</td>
<td>2.36</td>
<td>0.61</td>
<td>3.88</td>
<td>188</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PHQ-9</td>
<td>0.19</td>
<td>0.03</td>
<td>5.75</td>
<td>188</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Note. PCRT = Perceived Childhood Relational Trauma, PHQ-9 = Patient Health Questionnaire-9 (depression score). The table indicates that childhood relational trauma predicts dimensional bipolarity (MDQ) when controlling for depression level.

Hedonic Energetic Social Engagement (HESE)

Self-Esteem

Self-esteem is posited as the key element of the sociometer, which drives the putative dynamic model. A Chi-square analysis was undertaken on the total cohort to investigate the relationship between self-esteem and the major mood states (Appendix Table 42). This found that \( \chi^2(8, n = 171) = 57.5, p < .001 \), Cramer’s V = 0.41, indicating a moderate relationship between mood category and self-esteem. Examination of the depression cohort found that almost half of the column was located in SISE 1, which is the lowest self-esteem score. SISE categories 2 and 3 also were also overrepresented in the depression cohort. Though the (hypo)mania group count was lower than expected in 1 and 2 (low) self-esteem sectors (though higher than expected for SISE 3, 4 and 5 – higher self-esteem), – this pattern did not appear significantly different to those with a euthymic mood. Thus, there was a concordance between depression and low self-esteem, though the self-esteem attribution of (hypo)manic participants did not differ from euthymics, when subject to simple explicit SISE measurement.

It can be seen from the line plot Figure 23 of prestige (nlogSIPS) juxtaposed with self-esteem (SISE) and the Contingencies of Self-Worth (CSW.av) scales that significant relationships existed within the prestige-self-esteem spectrum. Prestige was low in the unipolar and bipolar nodes and – except for the pseudounipolar group
– prestige and self-esteem roughly paralleled each other. It is notable that pseudounipolar subjects had a higher prestige than unipolar subjects, while their self-esteem was comparable to the unipolar node. The contingencies of self-worth values (CSW.av) presented as a mirror image to self-esteem – where self-esteem was high, contingent self-worth was low – self-esteem in this context being non-contingent on appearance, approval, and competition. The bipolar I (S1) group had a significantly higher contingent self-worth score (CSW.av) than controls (S7) – a T-test found that S1 had a CSW.av score of 4.73, while the S7 score was 3.68, the 95% CI for the difference being [0.65, 1.44], $t(97) = 5.26$, $p < .001$, $d = 1.06$. 
The MOPF Social Inclusion Sensitivity (MSIS) scale asked individuals to what extent events such as people seeking their company, or being valued or excluded, affects their hedonic state. The scale measures a form fruste of \textit{HESE}; individuals reacting intensely to prestige contexts with elevated or lowered mood are ipso facto showing emotional expressivity (\textit{HESE}). MSIS was assayed in the five mood states in Appendix Table 43 and Table 44, where it can be seen that the (hypo)manic, depressed and, especially manic (mixed) groups have significantly higher MSIS values than the euthymic group.
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

Figure 24 shows the relationship between spectrum nodes and MSIS, indicating that the control (S7) group has a lower social inclusion sensitivity than the other clinical and subclinical groups. To test this apparent association – looking for trait rather than mood-related relationships – an ANCOVA of MSIS score by SPECTRUM was run, with depression (PHQ9) and mood elevation (ASRM) as covariates (Appendix Table 45 and Table 46). SPECTRUM, PHQ9 and ASRM together accounted for 19.52% ($r^2_{adj} = 16.41\%$) of the variance in MSIS, with the individual contributions being for SPECTRUM $F(6, 207) = 3.03, p = .007$, depression (PHQ9) $F(1, 207) = 2.78, p = .10$, and mood elevation (ASRM) $F(1, 207) = 4.77, p = .03$. Thus, SPECTRUM and mood elevation (ASRM)) in the spectrum analysis were significantly related to MSIS. With respect to the individual nodes it was found that the bipolar I (S1) and subthreshold (S4) nodes had significantly higher MSIS than the controls (S7) with moderate to large $d$ values. Whilst failing NHST – due to $p$ values at or below .10 – the S2 and S3 groups had low $n$’s and substantive $d$ values, indicating a trend toward significance in these nodes. The Pearson correlation of dimensional MDQ bipolarity with MSIS was 0.38 ($N = 228$). Together, these results strongly suggest that social inclusion sensitivity is a core component of bipolarity.
Figure 24. Confidence intervals for social inclusion sensitivity (MSIS) by spectrum node. The figure indicates that the control node (S7) has an MSIS score lower than all of the other nodes, though this is qualified by ANCOVA statistics which control for mood variables.

Stealth (Prestige Withdrawal)

Figure 25 depicts the relationship between depression score (PHQ-9) and MSPaM for the spectrum cohort. As depression scores increased in the subthreshold depression range, MSPaM increased – though the unipolar (S6) node shows no such correlation ($r_p = 0.01, n = 37$). At a depression (PHQ-9) score of around 10, MSPaM plateaued. A mirror response occurred for prestige (nlogSIPS) score (Figure 26) – prestige fell as subthreshold depression scores increased, but reached a plateau at clinical levels of depression. There was a positive correlation between depression (PHQ-9) scores and MSPaM in the combined (S1&2) bipolar group ($r_p = 0.25, n = 77$), in the subthreshold (S4) node ($r_p = 0.31, n = 32$) and in the control (S7) group ($r_p = 0.39, n = 48$). In the control (S7) group the correlation was 0.41 ($n = 38$) for the euthymic persons and only 0.23 ($n = 9$) for those with mild depression.
To test whether MSPaM differs between the pseudounipolar (S3) and the unipolar (S6) conditions, a T-test (with no assumption of equal variance) was run, which found a mean score for the family history positive group 13.26 points higher than for those without a family history, 95% CI [3.08, 23.43], $t(40) = 2.63, p = 0.012$, $d = 0.75$ – indicating a medium to large effect. The presence of a bipolar family history (S3) mediated higher MSPaM in persons with a history of depression, though does this correlate with an increase in prestige (hypothesis 5)? A T-test (with no assumption of equal variance) found that the depression group with familial bipolarity (S3) had a mean nlogSIPS of 3.54 while the non-familial group scored 3.02, the 95% CI for the difference being [0.06, 0.98], $t(29) = 2.30, p = 0.029$, $d = 0.69$ – supporting hypothesis 5. However, the Pearson correlation of nlogSIPS and MSPaM in the pseudounipolar (S3) group was only -0.01, $n = 17$ – suggesting that the higher prestige of the pseudounipolar group may be a trait rather than being dynamically related to MSPaM.
Figure 25. Scatterplot of MSPaM versus depression (PHQ-9) score in spectrum cohort (jitter applied) – Lowess smoother indicated. From PHQ-9 scores of 10 and above (corresponding to depressive caseness) there is an asymptote at which MSPaM scores do not rise further.
Figure 26. Relationship of prestige and depression in the spectrum cohort with jitter and Lowess smoother applied. In non-clinical levels of depression (PHQ-9<10) as prestige falls depression increases, while with depressive cases an asymptote is reached and prestige levels remain stable despite increasing depression.

Prestige Enhancement (Hypomania)

Figure 27 includes the plot of mood elevation (ASRM) against spectrum node – revealing an elevation in mood in the subthreshold (S4) node – this is in part an artifact of the transfer of nine categorically mood elevated participants from the control (S7) node to S4. These (hypo)manic S4 participants also tended to have higher MDQ scores (with a much larger variance) – suggestive of subclinical bipolarity when compared to euthymic S4 persons. The mean MDQ scores between the (hypo)manic and euthymic S4 groups differed by 3.12 points – the 95% CI [0.62, 5.62] \( t(13) = 2.70, p = .018, d = 1.05. \)

There was no direct relationship between TENSION and mood elevation (Figure 27) and this was confirmed by the correlation coefficient \( r_p = -0.05, N = 228. \) Even more curious was the apparent elevation of mood in the simplex (S5) node,
where participants were deemed normal, but for the presence of a positive bipolar family history.

**Mood State**

An ANOVA (Appendix Table 47, Table 48) was undertaken to investigate the relationship between prestige (nlogSIPS) and mood state. This found that the mean prestige (nlogSIPS) score for categorical mood elevation – (hypo)mania – was 3.98, while that of the euthymic group was 3.42, the 95% CI for the difference being [0.06, 1.06], \( p = .019 \), \( d = 0.74 \). The categorically depressed group had a mean prestige of 2.90 and the 95% CI for the difference from the euthymic being [-0.86, -0.18], \( p < .001 \), \( d = 0.69 \). Figure 20 graphically depicts this finding which confirmed hypotheses 8 and 9 – that prestige is increased in (hypo)mania and reduced in depression. The “mixed” (manic) and mild depression groups had prestige levels equivalent to those with a depressive syndrome. Figure 28 shows the relationship between prestige (nlogSIPS) and MSPaM by mood state panels. As prestige approach motivation (MSPaM) increased for individuals with (hypo)mania, prestige (nlogSIPS) showed a commensurate increase with a Pearson correlation of 0.51 (\( n = 23 \)). There was no relationship between these variables for the remaining mood states.
Figure 27. Line plots of mean mood elevation, depression score and TENSION by spectrum node.
Figure 28. Prestige (nlogSIPS) versus prestige approach motivation (MSPaM) for five mood states. Only the (hypo)manic category shows a correlation ($r_p = 0.51$) between the two variables.

A one-way independent-measures ANOVA (Appendix Table 49, Table 50) revealed that mood category was significantly related to prestige approach motivation (MSPaM), $F(4, 219) = 8.51$, $p < .001$, $r^2 = 13.45\%$. Analysis revealed that the (hypo)manic group had a weakly positive and non-significantly higher MSPaM score than the euthymic group; estimate = 11.59 points, 95% CI [-1.22, 24.41], $p = .097$, $d = 0.58$. The depressed group, on the other hand, was 16.33 points higher than the euthymic group, 95% CI [7.39, 25.27], $p < .001$, $d = 0.82$ – indicating a significant and large between groups difference. Interestingly, with an $n$ of only 9, the manic (mixed) group had a significantly higher prestige approach motivation than the euthymic group by a total of 26.34 points, and this was statistically significant, 95% CI [7.17, 45.51], $p = .002$, $d = 1.32$. Though the manic (mixed) ($n = 9$) group had an MSPaM 14.75 points higher than the (hypo)manic ($n = 23$) the difference was not significant with a 95%CI of [-6.80, 36.29] and a $p$ of .330, $d = 0.74$. 
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

Tension

A one way ANOVA (Figure 29) was utilised to test differences in TENSION between mood states. This found that there was no difference in the mean TENSION between euthymia and (hypo)mania 95% CI [-1.12, 0.85], \( p = .996 \); though the (hypo)manic group had a broader interquartile range – indicative of individuals with both increased and decreased tension. The depression group was on average 2.68 points higher on TENSION than the euthymic group 95% CI [2.00, 3.35] \( p < .001 \) \( d = 1.67 \), while the mild depression group mean was 1.73 points higher than the euthymic mean 95% CI [0.92, 2.54] \( p < .001 \). The “mixed” (manic) group had a mean TENSION score 2.61 points higher than the euthymic 95% CI [1.09, 4.14] \( p < .001 \). Mood state explained 40.85% \( (\hat{r}^2_{adj} 39.75\%) \) of the variance in TENSION. When comparison was made between the “mixed” (manic) and (hypo)manic groups the manic group had a TENSION level 2.75 points higher than the (hypo)manic 95% CI [1.05, 4.45], \( p < .001 \), \( d = 1.83 \).
Figure 29. Boxplot of TENSION versus mood state. There was no difference in the mean TENSION between euthymia and (hypo)mania, though the interquartile range for (hypo)mania was broader. The depression group was higher on TENSION than the euthymic. The “mixed” (mania) group had a mean TENSION score 2.61 points higher than the euthymic 95% CI [1.09, 4.14] p < .001.

Tension level was analysed by spectrum node in the combined (hypo)manic and “mixed” (manic) groups by means of a dotplot (Figure 30). This found that the bipolar I (S1) and bipolar II (S2) nodes accounted for the majority of those with a tension level greater than 5 (the third quartile for TENSION in the control S7) group. This was consonant with the notion that high tension equates with mania, rather than hypomania (which may be expected in the subthreshold S4).
Figure 30. Dotplot of TENSION by spectrum for all cases with a categorically elevated ASRM. This includes the (hypo)manic and manic (mixed) mood states. Scores above 5 exceed the third quartile for the control group – where it can be seen that 6 of 8 cases arise in the S1 and S2 cohorts with 2 from S4.
23 individuals were rated as (hypo)manic and their MDQ scores were analysed by means of a dotplot (Figure 31). This found that the majority (13/23) had suprathreshold scores, while 7 had subthreshold scores. Three rated as completely negative for the MDQ.

The (hypo)manic group was investigated for the relationship between mood states and tension. Figure 32 reveals that there was a neglible change in mood elevation (ASRM) as TENSION rose ($r_p = 0.28$, $p = .21$, $n = 23$), while there was a borderline significant increase in depression (PHQ-9) scores as TENSION rose ($r_p = 0.42$, $p = .05$, $n = 23$) – indicative of possible emergent manic (mixed) states due to tension. This was confirmed in the broader group of all subjects with a categorically elevated mood ((hypo)manic + manic (mixed)), where the correlation of TENSION with depression (PHQ-9) score was 0.62 ($p < .001$, $n = 31$).
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

Figure 32. Mood elevation (ASRMtot) and depression (PHQ-9tot) scores versus TENSION in the (hypo)manic cohort. As TENSION increases there is a trend for mood elevation to fall, while there is a borderline significant increase in depression score (jitter applied).

In Figure 33 the y axis is prestige approach motivation (MSPaM), while the x axis represents depression score (PHQ9) and the z axis designates TENSION. Clinical disorders – as located by the mean values for MSPaM and PHQ9 scales using the mood categories – are superimposed on the xy plane. In this instance it can be seen that the TENSION z axis runs as expected at approximately -30°. As TENSION increases clinical disorder severity increases, while MSPaM and depression scores increase in tandem.
Figure 33. Prestige approach motivation (MSPaM) versus prestige withdrawal (depression – PHQ-9 score) with TENSION represented by contour plot. The severity of disorder increases with increasing tension. Precise mood state loci are located at the centre of the text boxes.

A scatterplot (Figure 34) examined the relationship between prestige (nlogSIPS) and TENSION in the combined mood elevation – (hypo)mania and manic (“mixed”) – groups. This found that there was a minor correlation between the two variables for (hypo)manic subjects, though there was a significant fall in prestige as tension increased in the manic subjects. Note that it is deemed that the “mixed” cohort is in likelihood manic (mixed) – an issue which is explored in the Discussion.
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**Figure 34.** Scatterplot of prestige (nlogSIPS) against TENSION in the combined mood elevation groups – (hypo)mania and manic (mixed) with Lowess smoother and jitter applied. The (hypo)mania group is indicated in black, while the “mixed” (mania) group is in red.

**Systemic Aspects**

Figure 35 shows the relationship between MSPaM and prestige with a third variable – depression (PHQ-9) – indicated by contour. High depression (purple) – which is largely bipolar – was centred in the low prestige/ high MSPaM quadrant. A multiple regression of prestige (nlogSIPS) and MSPaM into the natural logarithm of depression (nlogPHQ-9) found that both prestige ($F(1, 193) = 20.55, p < .001$) and MSPaM ($F(1, 193) = 25.86, p < .001$) predicted the natural log depression score. The latter was chosen because of inconstant residuals in the untransformed PHQ-9. The variance explained by this model was $r^2 = 21.08\%$ ($r^2_{adj} = 20.26$). The scatterplot for the unipolar group was diffusely spread and not clustered by quadrant.
Figure 35. Contour plot of MSPaM versus prestige (nlogSIPS) with level of depression (PHQ-9) represented in colour contour. The upper left quadrant may be seen as a depressive basin of attraction.
Figure 36 indicates the contour relationship of prestige (nlogSIPS) and MSPaM with mood elevation (ASRM) indicated in contours, where high mood elevation was centred in the high prestige/high MSPaM quadrant. Both prestige ($F(1, 150) = 5.74, p = .018$) and MSPaM ($F(1, 150) = 5.94, p = .016$) – the residuals for ASRM being of inconstant variance – significantly predicted the natural log of mood elevation (nlogASRM). The model explained 6.73 % of the variance in nlogASRM ($r^2_{adj} = 5.48\%$).

![Figure 36: Contour plot of MSPaM versus prestige (nlogSIPS) with mood elevation (ASRM) represented in colour contour. The upper right quadrant may be considered a basin of attraction for mood elevation.](image_url)
Bipolar Causation

Utilising binary logistic regression, a family history of bipolar disorder predicted a high MSPaM (using a binary “high/low” value) in the spectrum cohort – (OR 1.98, 95% CI [1.12, 3.52], p = .019). When combined in a multiple regression (Appendix Table 64) both bipolar family history and perceived childhood relational trauma (PCRT) predicted MSPaM. Similarly, family history predicted bipolar disorder – (OR 4.55, 95% CI [2.49, 8.34], p < .001). Bipolar family history also strongly predicted the experience of abuse or neglect in childhood (PCRT) – (OR 4.65, 95% CI [2.35, 9.23], p < .001).

Binary logistic regression was then used to analyse the relationship of potential causal variables to a diagnosis of bipolar disorder (BD I and II) – these included a family history of bipolar disorder, perceived childhood relational trauma (PCRT) and MSPaM (binary low/high variable). Table 52 (Appendix) shows that all three variables significantly predicted bipolar disorder; when accounting for each other. Familial bipolar history also significantly predicted PCRT (Appendix Table 53). When PCRT was added to the regression of bipolar family history into BD (Appendix, Table 54, Table 55) the family history OR (whilst remaining significant) fell from 4.55 to 3.29 – indicative of a modest partial mediation effect for perceived childhood relational trauma (PCRT) when modelling bipolar disorder diagnosis using bipolar family history.

The pairwise odds ratio for MSPaM (high:low) for individuals – contingent on family history – was examined in Table 16. This found that MSPaM predicted BD (OR 6.8), but only in the absence of bipolar family history; where family history was positive the odds for MSPaM were non-significant. Table 17 depicts the odds ratios for bipolar family history (+/-) and found a strong prediction of BD where MSPaM is low (OR 11.33) and a much more modest – though significant – OR when MSPaM is high (2.54). See Figure 37.

In the former examination of binary variables, analysis was on categorical BD, yet the prestige model construes bipolarity in a nodal-dimensional manner. Dimensionality may be approximated by the lifetime tally of (hypo)manic symptoms
as measured by the Mood Disorder Questionnaire (MDQ). Therefore, a multiple regression analysis (Appendix Table 56) into the MDQ was undertaken to investigate the impact of bipolar family history, PCRT and MSPaM on dimensional bipolarity. The regression model found persistent predictive effects for bipolar family history, MSPaM and PCRT. The interaction term between bipolar family history and MSPaM was significant in a new regression – \( p = .005 \) (Appendix Table 57) and the explanation of variance increased commensurately from an \( r^2_{adj} \) of 31.24% to 33.89% when the interaction term was added. These results indicate that the interplay of bipolar family history and MSPaM continues to be significant using a more broadly representative measure of bipolarity. Lastly, a binary logistic regression (Appendix Table 58) found that there was no facilitatory interaction between PCRT and MSPaM into BD diagnosis (interaction term \( p = .978 \)).

Table 16  Binary Logistic Regression on Bipolar Disorder (BD) diagnosis

<table>
<thead>
<tr>
<th>Reference category</th>
<th>Category</th>
<th>OR</th>
<th>95% CI</th>
<th>P value</th>
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</thead>
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<td>Low MSPaM</td>
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<td>[2.56, 18.05]</td>
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<tr>
<td>High MSPaM</td>
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<td></td>
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<td></td>
</tr>
</tbody>
</table>

*Note. OR = odds ratio; CI = confidence interval; MSPaM = prestige approach motivation. Family history BD x MSPaM overall \( p = .031 \).*

Table 17  Binary Logistic Regression on Bipolar Disorder (BD) Diagnosis

<table>
<thead>
<tr>
<th>Reference category</th>
<th>Category</th>
<th>OR</th>
<th>95% CI</th>
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<tr>
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<td>Family history (+)</td>
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<td>Low MSPaM</td>
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</tbody>
</table>

*Note. OR = odds ratio; CI = confidence interval; MSPaM = prestige approach motivation. Family history BD x MSPaM overall \( p = .031 \).*
Figure 37. The relationship of bipolar family history with perceived childhood relational trauma (PCRT), prestige approach motivation (MSPaM) and bipolar disorder. PCRT is a partial mediator, while MSPaM is a moderator of the relationship between bipolar family history and bipolar disorder.
Narrative – Ailsa

Ailsa is a 41 year old, overweight, married mother of a 16 year old son, working in a call centre. She came to treatment after referral by her general practitioner with trouble sleeping, suicidal ideas, low mood and anxiousness. She was taking an antidepressant and a sedative, after failing to get better with numerous treatments. She was dependent on a medication for headache. The referral letter noted self-depreciation in the wake of dealing with family matters and traumatic early years. Her presentation coincided with a sense that her mothering of her increasingly independent son was faltering.

Ailsa describes an almost lifelong experience of distress and self criticism. Her feelings of worth had become linked to her weight, and in her teens she had restricted food to the extent that "not even a pea" passed her lips. She felt that she was in control when she weighed less and hated her current girth.

In her childhood, Ailsa’s Danish father, who unpredictably erupted into violent moods, had not touched her or shown love. Her mother criticised her constantly and she lived on “egg shells”. She was terrified of being alone and would miss school to follow her mother around, carrying tablets with her for solace. She was locked away in the house and told to hide when people visited. At home she was known as "the Australian" and at school "the Danish girl”. She felt that she never fitted in anywhere; she changed identities to match her circumstances to the extent that even she never knew the real Ailsa. She said that she had only learnt how to cope in life through books and television programs. She felt that people had never known her real self, which was “ugly”, despite her beauty. She left school in year 10 and socialised with a group of wayward youths, eventually taking up a bottle of spirits per day and sleeping around.

At age 15 she had had a psychotic breakdown, characterised by auditory hallucinations, which lasted six months. She was raped at the age of 19 and continued to experience nightmares thereafter. In her 20s she was prescribed an antidepressant and noted a significant elevation of mood along with less sleep, overactivity, racing thoughts and non-stop talking. Her interest in sex was increased and she had an affair
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– her “motor running”. She felt that she had become a central player in her life, pursuing goals and experiencing more humour. She said that milder versions of this episode had started around the age of 15. By the age of 28 she was using amphetamines to lose weight and again developed fast and elevated feelings. These high feelings recurred throughout her life, occurring again at age 43 after she was prescribed steroid medication for asthma. At other times she felt depleted and lacking in energy, spending much of her time in bed sleeping. These phases can go on for six months or more. By this time she had ceased cutting her wrists in order to deal with her pain.

Her mother’s grandmother suffered with manic depression and her sometimes dramatic mother was known to drink and get depressed. Ailsa grew up in an environment of emotional austerity, where attack was the order of the day. She became a tense and fearful young woman with an unstable sense of self – the latter fuelled by her carriage of bipolar genes. Looking out to her social world, she generally found no valuing, only indifference or ridicule. This confirmed her sense that she did not belong. She became acutely sensitive to the esteem she was held in and would often retreat into herself, hiding away from scrutiny. When prompted by a substance, or an opportunity, she might advance her prestige, becoming the life of the party. When her mood was steady, she scored 2 on the Rosenberg Self-esteem Scale – the normal range being 15-25.

Over the years of struggle, Ailsa has stopped gambling and been able to curb her drinking. She has returned to work and shed twenty kilograms Her son is an upstanding, if overly relaxed, student and she has remained in a long-term relationship, despite feeling undervalued. "I pulled myself together" she said. Rather than seeing herself as totally damaged, she was able to acknowledge that some things do change with time.
Chapter 8: Discussion

“Other traits valuable to leaders are high motility (rapid and energetic reactions), tonus shown in gesture and ring of the voice (cf. p. 219), erect, aggressive carriage, tenacity, face-to-face mode of address, and the reinforcement of energy flowing from a fairly high emotional level.” (Allport, 1924, p. 422)

Introduction

The problem of the bipolar spectrum has been amply described, though its solution requires current and future endeavour. It is pertinent at this juncture to review the aim of this thesis:

“Therefore, a literature review on extant psychosocial factors involved in bipolar disorder will be undertaken and a dynamic model of bipolar spectrum pathogenesis, centered on the notions of group investment – prestige and self-esteem – will be developed. A schema of affectivity based on Panksepp (1998) and Mithen (2005) – Hedonic Energetic Social Engagement (HESE) – will support this enterprise. This will lead to a bipolar spectrum model based on clinical and genetic parameters, illustrating a gradient of bipolarity. This model will be evaluated for its structural and dynamic properties.”

It is the task of the Discussion to elaborate, integrate, and conclude on these themes – thereby succeeding or failing in the eyes of the examiner. It seems a daunting – but not an impossible – task, if we keep as our target the origins of bipolarity on the distal margins of time. This thesis is developing a model of bipolar spectrum pathogenesis based on the notion of social investment (prestige) and, in order to achieve this, a model for affectivity – Hedonic Energetic Social Engagement (HESE) – has been adopted. I have postulated that the bipolar mood syndromes are extremis states of the HESE and TENSION systems, occurring in the context of prestige/self-esteem disturbance and that they have arisen due to group inclusion pressures in evolutionary time. Measures of prestige and prestige approach motivation have been developed and these have been compared in the spectrum groups. Psychological mood paths have been supported in a dynamic analysis. The model will increasingly adopt biological elements to complement its psychosocial emphasis, with the aim of approximating an ultimate and proximate theorem. Implications for
psychiatry will be discussed and it will be concluded that prestige is a valuable concept which has forged – not only affective illness – but aspects of the fabric of society itself.

**Prestige**

**Structural Model**

Hominin groups walked the African savannah for some six million years after the great split from ancestral chimpanzees. Scant little is known of the social forms of these ancient societies, though based on neocortex ratio they lived in groups ranging in size from 60 (Australopithecines) to 150 (Homo sapiens) (Dunbar, 2003). Their distant forbears had lived in dominance hierarchies, though it is likely that prestige-hedonic processes (Chance & Jolly, 1970) became the prime binding force in the common ancestor to chimps and humans (Stevens & Price, 2000). Later hominins such as Homo sapiens lived for some 150 000 years contemporaneously with species including Homo neanderthalensis and Homo erectus, until extinction left our ancestors alone. But, just what were ancient human societies like? Extrapolating backward from current hunter gatherers – and forward from earlier genera – these groups clung together, surviving internal and external threats through the power of their minds – some of which became disordered.

Psychogenesis is the “development of a medical disorder or illness resulting from psychological rather than physiological factors” (American-Heritage-Dictionaries-Editors, 2011) – that is involving a (social) mind iterating with the body to produce disease. This implies a pathogenic sequence of events which are best described in psychological terms and which lead to a diseased somatic state. Psychoanalytic theorizing regarding bipolar disorders has particularly focused on the difficulties these patients have in dealing with the experience of loss (Klein, 1935). Situations of relational slight, neglect or disappointment are considered further precipitants of either a melancholic or manic episode (Freud, 2006). Self-esteem is lower on implicit measures in people with bipolar disorder (Winters & Neale, 1985) and is commonly unstable (Knowles et al., 2007) and rejection sensitivity is a feature of the psychology of bipolar atypical depression (Thase, 2009). Ailsa, in the narrative,
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amply portrayed this. There is evidence that life events may trigger bipolar depressive episodes (Miklowitz & Johnson, 2009) and there is a correlation between low social support and the course of bipolar disorder (Alloy et al., 2005; Cohen et al., 2004; Depp & Meeks, 2002; Johnson et al., 1999; Pratchett, 2010).

Clearly, psychosocial research into the generation of bipolar spectrum conditions is relatively nascent with regard to both proximate and distal causation; how are these disorders mediated and how did they appear in our ancestors? Syndrome boundaries remain debated – a biological substrate eludes researchers – and many patients continue to suffer a significant illness burden. For these reasons, it is important to develop testable models and to provide targets for intervention. Given that the bipolar disorders are related to how the self “esteems” itself in the context of life events and social support, it is pertinent in this thesis to investigate the role by which responses to the investment of the ancestral and modern group – prestige – may play in affective aetiogenesis. The quotation from Allport (above) conveys the hedonic and energetic behaviour of individuals who have become prestige leaders.

The structural model proposed that bipolarity could be divided into seven spectrum nodes (Table 6) and that these would display meaningful differences with respect to prestige function. A number of hypotheses were suggested with respect to this model and reported in the results. The dynamic model pitted the self in relation to prestige events and divided essential responses into approach and withdrawal. This dichotomy was extrapolated to encompass hypomanic and depressive reactions; the evolutionary benefit of these psychogenetic “postures” will be investigated. Secondary investigations have drawn on the title of this thesis and investigated affect on the prestige landscape by means of contour plots. Similarly, the relationships between MSPaM, bipolar family history, perceived childhood relational trauma and bipolar disorder have been investigated through regression analyses. Prestige is a rich topic of enquiry – I propose that it is the basis of the bipolar diathesis.

The prestige spectrum (Table 6) has been presented in the Research Design and its mensuration delineated in the Diagnostic Algorithm and Spectrum Calculator (hierarchical) (Appendix). Interrogations of the data have attested to the consistency
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of the calculators – as for example demonstrated in Table 41 (Appendix) – revealing equivalence reliability. The demographics of the sample (Table 7) have yielded results which we might expect from clinical consideration of the populations included. Similarly, depression scores (Appendix Figure 41) – are sited where logic would dictate. These examples reflect parallel and internal reliability.

The prestige model of spectrum bipolarity attributes both a clinical and a genetic gradient to the nodal bipolar spectrum. This is consistent with platonic notions – the phenomenon is collected into a spectrum and then divided at the spectrum nodes. As such it is a nodal-dimensional model. The bipolar spectrum typically does not include a normative category, though, as previously noted, Phelps (2008) has conceived a spectrum location with no hypomania or mania. For the purposes of this model, spectrum node 7 (S7) includes those without a history of mood or other psychiatric disorders, without bipolar (MDQ) subthreshold features and without a family history of bipolar disorder. Designating the node in this way seeks to “extract all known bipolarity” from the null position and provide a reference group.

In Western psychiatry pure unipolar melancholia is becoming rarer, being encroached upon by the bipolar spectrum, as attested to by authorities such as Angst (2007). Authors such as Ghaemi et al. (2002) have included various forms of unipolar depression in their bipolar spectra, whilst researchers such as Akiskal (2008) and Klerman (1987) have not. It is argued that severe unipolar depression is an evolutionary byproduct of the adaptive strategy to withdraw from social scrutiny when social inclusion (prestige) is threatened. The ancestors of those with depression undertook this to a greater degree and duration than normals and they entered a phase of psychomotor “stealth”. Unipolar depression constitutes Spectrum category 6 (S6). Further nodes up the bipolar "ladder" may represent a phylogeny of adaptations, as hominins developed ever more complex, and at times maladaptive, means of responding to prestige challenge.

Johnson, Murray, et al. (2012) have reviewed the evidence on the association of bipolar disorder to adaptive characteristics and found that creativity and extrinsic ambition to achieve were overrepresented in those with milder forms of bipolarity,
especially in those with simply a positive family history. Persons with a family history of bipolar disorder – without a history of disorder themselves – may therefore show an increased ability to deal with social contexts and have a capacity for increased prestige. In the prestige model of spectrum bipolarity nosology, the category described is known as Spectrum node 5 (S5) – the simplex node. The “extended normal” cohort therefore comprised such individuals (members of the simplex (S5) group) and controls (S7). It was found that the subgroup with a bipolar family history had a significantly elevated MSPaM with a between groups Cohen’s $d$ of 0.75. As noted above, the collapse of all “normals” together should serve to control for factors other than the possession of a bipolar family history. However, the spectrum calculator allocated 11 cases to S5 with subthreshold and suprathreshold MDQ scores (who were non-MDQ cases by merit of absent severity or co-occurrence criteria). On the face of it – if bipolarity indeed covaries with MSPaM – this raises a serious problem in elucidating the effect of familial bipolarity (in isolation) on prestige approach motivation. Further, the finding of mood elevation in S5 cast doubt on how balanced this cohort was. For these reasons multiple regression offered a method for making a decision on the particular effect of family history across the total cohort. As Table 63 shows, family history – controlling for bipolarity, mood and MSIS – made a significant contribution to MSPaM, suggesting that genetic and/or development-specific factors were at play. Lastly, Table 64 clarifies that both family history and perceived childhood relational trauma made a significant contribution to MSPaM in this cohort, together explaining 9.99% of the variance.

Tijssen et al. (2010) and Murray et al. (2007) have provided evidence that hypomania may not always be a sign of morbid psychopathology. Rather, their work suggests that it is a relatively common developmental variant, which may not be associated with depression or the need for treatment. Such persons may demonstrate a self-limiting, relatively mild, elevation of mood without mania – so called unipolar hypomania, as exemplified in the findings of the National Comorbidity Survey Replication (Angst et al., 2010). Merikangas et al. (2011) found a worldwide lifetime prevalence of 1.4% for subthreshold bipolarity. Hypomania, entailing energisation and hedonic affect, is associated with elevated prestige (Le Bas, Castle, & Newton, 2013). It may arise as an aggregation of hypomania genes with the simplex
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adaptation. In the prestige model of spectrum bipolarity this node is termed Spectrum node 4 (S4) – the subthreshold node.

Klerman (1987) and Akiskal and Mallya (1987) recognise persons with depressive disorders and a family history of bipolar disorder as warranting a category in their spectra. In Spectrum node 6, depressive cases with a family history were explicitly excluded, while in the prestige model bipolar family history positive depression cases are allocated to Spectrum node 3. It is postulated that Spectrum 3 (S3) cases have acquired not only the unipolar adaptation, but also the simplex. As such, the condition makes them prone to not only prestige withdrawal, but also motivated re-entry to the group. As such, they may turn out to have briefer episodes of depression, as is seen with bipolar depression. This node was associated with an increase in prestige relative to the S6 unipolars. Taking into account the marginal $p$ value alongside a significant $d$ value, this node had an increased propensity to prestige leadership motivation compared with controls.

Dunner et al. (1976) conceived depressions with hypomania as bipolar II disorder, separating these out from depressions with mania (bipolar I disorder). Coryell et al. (1989) have suggested that this condition has a somewhat different inheritance from bipolar I disorder. Clearly, people with bipolar II disorder are subject to both hypomania and depression, suggesting the accretion of the unipolar and hypomanic adaptations, possibly in addition to simplex genes. The evolutionary function of this disorder is poorly known, but may involve the capacity for both prestige withdrawal and re-entry prestige modes, along with adaptive bipolar functions as described for Spectrum node 5. This category is known as Spectrum node 2 (S2). This category was associated with an increase in MSPaM relative to controls.

Mania is the defining characteristic of bipolar I disorder, being described as “a distinct period of abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased goal-directed activity or energy… sufficiently severe to cause marked impairment in social or occupational functioning or to necessitate hospitalization…or there are psychotic features.” (American-Psychiatric-Association, 2013). Mania is generally characterised by irritability and
sometimes aggression, as well as psychosis. What then makes this condition different from its bipolar II counterpart? Is it adaptive? Certainly, its features are the most socially disruptive of all the spectrum groups. It is suggested that bipolar I persons harbor all of the anomalies seen in bipolar II disorder with the addition of a propensity to dominance (Stevens & Price, 2000) indicative of a disturbance in tense arousal. In evolutionary time this may have served to forestall exclusion from the tribal group by means of agonistic leadership and psychosis has become the byproduct of this dominance phenotype. This study found that psychosis is a dimension unrelated to MSPaM. This node is termed Spectrum node 1 (S1) and it was associated with an increase in MSPaM relative to controls, despite evidence of sociodemographic and prestige disadvantage. Table 6 depicts the descriptions listed thus far. The prestige model thus sees the nodes on the bipolar spectrum as arising through the accretion of monopolar adaptations – affective “building blocks” – complicated in some instances by morbid “byproduct” development.

Prestige approach motivation signifies the extent to which an individual is motivated to achieve the status or accord of others – one of the centripedal vectors to group inclusion. Prestige maintenance is the intentional object of approach and withdrawal affect as seen in hypomania and melancholia. Like the affective temperaments which Evans et al. (2005) tested, I hypothesised that the motivation to group inclusion was a core feature in bipolarity and sought to demonstrate a “dose-response” relationship in the bipolar spectrum. Like Evans et al., my grouping included a genetic dimension, in the belief that affective temperaments should be polygenically determined according to threshold effects (Kelsoe, 2003).

The prestige spectrum model, as the diagnoses of its participants, is based on current diagnostic modes such as DSM-5 American-Psychiatric-Association (2013) and – just as it is subject to validity challenges – the model will be open to criticism. The dominant diagnostic modes delineate the upper limits of the model’s validity. Klerman (1981) situated the pseudounipolar (S3) node in his spectrum and the model is similar to that of Ghaemi et al. (2002) (Figure 7), who constructed their spectrum by opposed depressive and bipolar I poles: with bipolar II and the “bipolar spectrum disorder” groups located in between. The relative locations of the spectrum groups is
open to question and the spectrum models presented have all followed clinical
guesswork – based on uncertain decision rules – though it is likely that disorder
“puzzling”, however – alongside psychosis – it presents as one of only two categorical differentiators available to the MOPF project. The control (S7) node was given
particular attention to extract persons with evidence of soft bipolarity and create a sound reference group. No previous authors have included a normative category so
denuded of bipolarity as here.

Based on severity, disability and bipolarity – on a notional polygenic gradient – there is content validity in allocating nodes bipolar I (S1), II (S2) before bipolar familial depressions (S3). Persons limited to subthreshold symptoms (S4) clearly have still less “bipolarity” and those undiagnosed – with bipolar family histories alone (S5) – have a similar or lower “dose” of the construct. The inclusion of unipolar disorder (S6) between the simplex (S5) and control nodes (S7) is consistent with Ghaemi et al. (2002), but may be arbitrary. As is the case with radiation, a spectrum should have a full range of values and research mandates an unpolluted reference group.

The prestige model is subject to errors of measurement; for example, a stated family history of bipolar disorder is subject to having a valid knowledge of family diseases and a motivation to answer correctly. This in turn will affect the validity of inclusion to the various categories – eg S3 or S6. There may have been bias in case selection at both the mental health service or general practice levels. Reputable scales were used in the survey (eg PHQ-9, ASRM) and new scales were designed with reliability and validity in mind; three of which had acceptable internal consistency. The bipolar spectrum as described captures all common mood syndromes and other relevant states – eg subthreshold and pure family history. The line plots of TENSION and mood – and self-esteem and prestige – (Figure 23, Figure 21) have face validity.
Being based on clinical history it has convergent validity with the nosology of mood disorders seen in clinical practice.

We come now to discussing the nature of prestige approach motivation itself – of what is it “made”? The concept was derived from the narratives of a number of patients with bipolar disorder who had intimated that prestige was an important thing for them. I had asked them to recount particular cognitions which appear to be recurrent in their lives – particularly those that were linked to mood changes. Once they understood the hunch that I had, it was possible to assemble a list of cognitions with a common theme to do with the striving for inclusion, leadership, and prestige. The list was shortened by reference to the correlation of the individual items with the total scale. Given the high Chronbach’s alpha (0.93), the final MSPaM scale probably could have been shortened. I then sought input from a more extended group of patients, friends, and family as to their approximation of this construct. The early results were encouraging and prompted the development of further scales centring on the notion of prestige. MSPaM is, therefore, a concept based on the iteration of guesswork, phenomenology and field testing, which relates to the individual’s disposition to garner investment from the group through prestige leadership behaviours.

**MSPaM Factor Analysis**

It was possible to separate MSPaM into three factors using factor analysis – NEGESTAFF – negative esteem affect (7 items), POSESTACT – positive esteem action (5 items) and POSESTAFF – positive esteem affect (5 items). The individual items compromising each are indicated in the lists below:

*Negative Esteem Affect* spoke to the possession of a deflated and negative self-esteem with low affect, associated with a sense of threat (tension) and a need for prominence in group inclusion. It was moderately correlated with self-esteem (SISE) and depression (PHQ-9) scores (Table 59).

1. When I struggle for recognition I wake early and dwell on righting things
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2. To keep self respect I need to be included
3. I feel deflated when people won't acknowledge my contribution
4. People respect me more when I achieve my goals
5. My good standing in the group is often under threat
6. My confidence crashes when people don't support me
7. People only value me when I emphasise my good qualities

*Positive Esteem Action* spoke to a motivated attraction to hold attention, despite some resistance. The factor was positively correlated with ASRM mood elevation (Appendix Table 59).

1. I try to make myself attractive to hold people's attention
2. When people won't budge I am determined to win them over
3. I use the information I have to hold people's attention
4. I would like to impress people with my new possessions or creations
5. I get more inventive when I have to fight for good standing

The *Positive Esteem Affect* factor indicates a propensity to pursue positive affect in dealings with other people. It was weakly associated with depressive symptoms (Appendix Table 59).

1. I work hard to maintain my reputation
2. I dream that my achievements will be valued
3. It is important for people to hold me in good esteem
4. I feel over the moon when people value my efforts
5. I often think about things I have said which may have impacted on others
As already noted, Negative Esteem Affect was associated with depressive symptoms (Appendix Table 59) and was negatively correlated with self-esteem. Perusal of Figure 13 suggests that this factor may be associated with the greatest differential between the bipolar I (S1) and the control (S7) node. Further, it can be seen that Negative Esteem Affect had the greatest predictive power of the three MSPaM factors in relation to bipolar disorder diagnosis (Table 10) and was a significant predictor of MDQ (Table 11), once mood was accounted for. Therefore, a pivotal aspect of bipolar disorder appears to be the motivated desire for prestige in the context of low, or unstable, self-esteem – the latter finding mirrors the catchcry of Bentall et al. (2005). Table 9 and Figure 12 depict an ANCOVA of Spectrum into MSPaM and this revealed that the bipolar nodes (S1&S2) were significantly different to the control node. Probably because of a low n, the pseudounipolar (S3) node had only a statistically marginal difference to the control – p = .051 (though with a large d), strongly suggestive of a real difference (Le Bas, Newton, Sore, & Castle, 2015). Nodes S4-S6 were not significantly different to controls. Nevertheless, the predicted trend downward from S1 to S7 was evident – SPECTRUM contributing moderately to MSPaM (F(6,214) = 4.95, p < .001) – suggesting that prestige approach motivation may parallel the range of bipolarity – reflected in Figure 11. Depression (PHQ-9) and mood elevation (ASRM) were included as covariates because they had both been shown to affect MSPaM scores and the significance of this will be explored in the Dynamic model discussion. This result indicates that prestige approach motivation appears to be inextricably tied to the bipolar spectrum, a fact which may have impacted both our forebears and our modern society.

**MSPaM Prediction**

Is there evidence as to what predicts MSPaM? A multiple regression (Table 12) was undertaken to explore this question. As might be expected from the previous discussion, the S1 spectrum node was a significant contributory factor – the “greatest” bipolarity corresponded with the highest drive to social investment. In reflection of its low bipolarity score, the unipolar S6 node predicted a lowered MSPaM, in comparison with the control S7 group. The sociometer variables CSW.av and MSIS were associated with increases in MSPaM, suggesting their facilitatory role in the
dynamic model, where TENSION was a notable predictor also – as predicted by the dynamic model. MSIS is deemed such a large predictor because of its ubiquitous role in the dynamic process; it is a logical precursor to prestige approach motivation, where sensitivity to the cue of reduced relational value is likely to accompany the motivation to augment social relationships. Overall, the predictors accounted for around 60% of the variance in MSPaM.

We therefore have seen a rapprochement of Platonic notions of collection and division in the construction of the prestige bipolar spectrum formed by genetic and clinical gradients, which are exemplified in seven nodes reflecting a gradient of bipolarity. The differences in prestige approach motivation, which were predicted, have been found in S1, S2 and S3, when compared to controls (S7). In addition, when otherwise homogenous groups S3-S6 and S5-S7 were reviewed in T-tests, a difference in MSPaM was observed. Therefore, the bipolar spectrum so constructed has revealed differences in prestige function between these groups. MSPaM appears to reflect a motivated disposition to increase prestige leadership, driven by tension-related social inclusion sensitivity. In the following section I will explore a model which looks at the dynamic operation of these variables in the ascent to mood syndromes.

**Dynamic Model**

“Let’s be totally honest here, the world is very complex and hard to understand, no matter how brainy you are. To make things easier to grasp, reality is generally broken down into bitesize chunks. These chunks are abstracted from the real world and simplified into things called models.” (Griffiths, 2009)

The dynamic model (Figure 9) seeks to describe bipolar pathogenesis from the perspective of social investment, as influenced by prestige-related life events. Bipolarity, it has been argued, is a response to the challenge of social inclusion in a precarious ancestral social environment. Life events occur in the social milieu and consist of a day to day “microclimate”, punctuated by positive and negative “macro” incidents. The microclimate of daily life entails positive and negative flows – the latter encapsulated in the Inventory of Negative Social Interactions of Lakey, Tardiff,
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and Drew (1994). Events such as being criticised, ignored and put down entail a decrement of social investment or prestige. Lakey et al. (1994) found that such interactions, along with low social support, were consistent predictors of psychological distress, low self-esteem, mistrust and dysfunctional attitudes. The link between such events and distress was accounted for by self-esteem and cognition in their study of college students. Self-esteem mediated the relationship between negative social interactions and depression. As is reflected in the dynamic model, self-esteem was by far the largest predictor of anxiety in their research. In further support for the dynamic model initiators, Miklowitz (2011) concluded that family conflict and criticism are strong predictors of the timing of recurrence and the degree of recovery from mood episodes. Drawing on Paykel and Brown et al. Post and Weiss (1998) note that:

“Psychosocial stressors related to work, family, and social interactions, with important threats to self-esteem unbalanced by psychosocial support and compensatory adaptive mechanisms (Post et al 1996b; Post and Weiss 1997), could be sufficient to trigger or reactivate an episode of recurrent affective illness.” (p. 198).

Alongside negative social interactions, the dynamic model proposes that goal success may trigger changes in prestige and self-esteem, with flow-on effects. Miklowitz and Johnson (2009) suggested that increased reward sensitivity in bipolar disorder may also trigger sensitivity to positive life events, thereby promoting confidence and goal engagement and eventual mania. The dynamic model locates social investment (prestige) as the active and energetic driver to mood dysregulation in vulnerable individuals and there is strong evidence for a correlation between low social support and the course of bipolar disorder (Alloy et al., 2005; Cohen et al., 2004; Depp & Meeks, 2002; Johnson et al., 1999; Pratchett, 2010). But what is the psychological mechanism?

We have previously encountered the sociometer construct of Leary et al. (1995) – whereby a psychological meter arbitrates on relational value and alerts self-esteem and affect of a threat to social inclusion. Alongside biological factors, the success of humankind was predicated by close group living, which allowed the growth of culture, intelligence and technology. The success of each individual – and
his or her offspring – was therefore determined by inclusion to the group. The antithesis to this – and the antithesis to life – was shunning and ostracism. Williams (2007a) has argued that we all need finely attuned ostracism detection mechanisms, mediated by self-esteem to deal with this threat. According to Buss (2012), self-esteem becomes a motivational mechanism to improve the respect of others and locate one in the hierarchy.

The validity of the dynamic model hinges on its ability to capture the essence of the pathogenesis of bipolar states. Models represent a real world target and are rarely derived solely from either theory or data (Frigg & Hartmann, 2012). The dynamic model grew similarly from clinical data supported by perusal of the literature and is an mixture of different ontological elements (Frigg & Hartmann, 2012). The relationship of prestige to self-esteem is based on personal, experiential, and clinical phenomenology. The connection has nomological validity; drawing on the work of authors such as Barkow et al. (1975) and Stevens and Price (2000). It is therefore an a priori contention. Similarly, the anxious (tense) response to self-esteem threat is ubiquitous enough to suggest a fundamental relationship and Lakey et al. (1994) have already been cited in this respect. After Thayer (1989), tension is posited as one of his two underlying arousal states (the other being energetic arousal) and is equivalent to somatic anxiety – it was only added to the model post hoc due to a strong empirical relationship with MSPaM and depression (PHQ-9) scores. The dichotomy of responses to prestige-related contexts via tension – approach and withdrawal – has some ethological validity and converges with human experience. While there is vast evidence linking anxiety (psychic tension) with depression, the connection to prestige approach is largely derived from MOPF data only. Lastly, the iteration of affect back to prestige is intended to simulate the feedback loop that describes much of motivated human behaviour (see Figure 9).

Though the prestige model may be a valid construct, the study has several limitations which relate to its exploratory nature. The causal direction adopted is inferred prima facie from the schema and the temporal and causative sequence of the model has not been established – this would require a longitudinal method such as experience sampling. It is also possible that psychological phenomena are mere
epiphenomena to biological changes, rather than aetiological units per se. Prestige plays a large role in this theory and whilst its numerical determination enhances reliability, it remains prone to cognitive and affective bias. In any case, it is perceived social support that appears crucial to the course of affective illness (Leskela et al., 2006; Pratchett, 2010). It could be argued that spectrum nodes should only be investigated separately, though the spectral concept argues for a continuous distribution of variables in a cohort enriched for bipolarity.

_Correlation Analysis in the Dynamic Model_

Indicative correlations are a prerequisite to supporting or refuting a model and Table 13 documents the predicted trends of the dynamic model. Prestige correlated positively with self-esteem and when self-esteem was low – as Lakey et al. (1994) showed it to relate to negative social interactions – there was an associated increment in tension. TENSION was positively associated with MSPaM, which in turn correlated moderately with mood elevation in the approach group, where mood elevation also correlated weakly with prestige. These relationships support the notion that perturbations in prestige and self-esteem may drive tension up, leading to prestige approach motivation, mood elevation and a reciprocal increase in prestige. There may be some increase in depressive symptoms commensurate with mood elevation (Figure 32), consistent with a degree of mixity – that is in some circumstances tension may have a bivalent effect. Increases in tension strongly predicted an increase in depression, which in the withdrawal group (those with syndromal and mild depression) did not correlate with prestige (at clinical levels of depression) – the latter fixed a low and static level compared to depression level (Figure 26).

The correlation between depressive symptoms and MSPaM was modest for the combined bipolar groups and intermediate for the subthreshold node. The strongest relationship for these two variables was found in the control (S7) cohort. It was a negligible 0.01 \( (n = 37) \) for the unipolar node. Figure 25 reveals that, in the total cohort, there is an asymptote past a PHQ-9 score of 10, from which prestige approach motivation did not increase further. This point is curiously at the threshold at which the PHQ-9 scale considers an individual a depressed case. That the control group revealed the strongest association of prestige approach motivation with depression
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score suggests a ubiquity to this phenomenon (with the exception of the unipolar group). Therefore, in cases of non-unipolarity – when depressive symptoms are low or subsyndromal, increases in depression level are associated with increases in prestige approach motivation. This may reflect a latent compensation to counter incipient ostracism through a motivated push to prestige – group inclusion. Non-cases may show the greatest increment in prestige approach by value of their “health” and basal low MSPaM. Because bipolar cases already have high levels of MSPaM, perhaps they have already reached a correlational asymptote at higher “case” levels of depression. In bipolar persons, stealth can ensue as prestige approach and depression reach their maxima.

TENSION has a strong positive correlation with depression, suggestive of a fundamental relationship. The study found that prestige was both statically lower in syndromal depression (Figure 20, Appendix Table 48) and that it did not correlate with depressive symptoms in the withdrawal group (Table 13). This is consistent with the stealth hypothesis, which predicts a low and narrow range of prestige to facilitate social avoidance. Reduced affective, motor, appetitive and sexual demands may have allowed our depressed ancestors to “fly under the social radar”, surviving ostracism and eventually recovering (J. M. Le Bas et al., 2013). This is consistent with Allen and Badcock’s (2003) social risk hypothesis of depressed mood, albeit with a sicker population than they envisaged.

Path Analysis of the Dynamic Model

Correlation is however not causation, to quote a popular maxim. To move closer to causal inference – while recognising the cross-sectional nature of the data – I turned to a path analysis of the dynamic model (Figure 9). I was firstly able to show that self-esteem mediated the relationship between prestige and tension (Appendix Table 21), Figure 16) – quite remarkable given the ontologically distinct domains each scale was drawing on. Self-esteem corresponded moderately with prestige – conceptually detecting relational threat as would a sociometer. Secondly, it was possible to “leap frog” one step up the dynamic stem and branch to show that TENSION linked self-esteem and MSPaM as a full mediator (Table 22, Figure 17).
As an effector, tension appeared to be activating the disposition to prestige procurement, based on proximal events in prestige and self-esteem.

TENSION also partially mediated the relationship between self-esteem and depression (Appendix Table 23, Figure 17). This reflects the clinical archetype in which there is a concurrence of low self-esteem, anxiety, and depression. The data support the dynamic model axiom that depression and self-esteem will also iterate in parallel to the mediating effects of TENSION. This was formulated because self-esteem appears to be directly linked to depressive affect. Despite its success in providing mediation, it should be reiterated that TENSION was a post hoc addition to the dynamic model, included once the data were at hand.

In a recent paper Corry et al. (2013) provided evidence which appears to support the dynamic prestige model. In a sample of 142 patients with bipolar disorder they successfully tested their theory that anxiety and stress mediate the relationship between self-critical perfectionism (SCP) and bipolar depressive symptoms. They were, however, unable to show statistically that stress and anxiety (tension) symptoms mediate the connection between self-critical perfectionism and (hypo)manic symptoms. The authors discussed a volume of evidence linking anxiety to bipolar disorder clinically, prodromally and epidemiologically. Their study utilised the Dysfunctional Attitude Scale (DAS) SCP subscale (Weissman & Beck, 1978), where perfectionism is emphasised, though Dunkley and Kyparissis (2008) note this is confounded by perceptions of demand and perfection directed to the self. Such persons harbor guilt, sadness, lack of hope and loneliness – being prone to feelings of ridicule and inferiority (Dunkley & Kyparissis, 2008) – ipso facto low self esteem. The authors have therefore indirectly suggested a mediating effect for anxiety/stress (tension) when modelling bipolar depression using self esteem. The authors also used the anxiety and stress subscales of the Depression, Anxiety and Stress Scale (DASS), though it is difficult to conceptually split “acute autonomic fear” (anxiety) from the “state of persistent arousal and tension with a low threshold for becoming upset or frustrated” (stress). Not surprisingly, they found that these subscales were exceedingly correlated ($r = 0.84$), reflecting probable multicollinearity. Despite providing evidence for a mediation effect of anxiety and stress between SCP and bipolar depressive
symptoms, anxiety fell short of significant mediation \((p < .10)\) between SCP and (hypo)manic symptoms. Stress did mediate the relationship between SCP and (hypo)mania, though the effect became non-significant after controlling for depressive symptomatology. Despite the statistical shortcomings of the latter data, their work lends qualified support to the mediating effects of TENSION on the relationship of self-esteem (in its approximation of the SCP concept) to mood elevation – the approach arm of the dynamic model. In addition, their data support the mediating role of TENSION in the relationship between self-esteem and depression – the withdrawal branch of the dynamic model.

The data from Table 24 (Appendix) strongly support the dynamic prestige model – when depression (nlogPHQ-9) was used as a mediator when modelling prestige using tension, the coefficient for tension dropped to a non-significant level and the Sobel test was significant – indicating that depression was a full mediator. Rather than conceive this phenomenon as a merely a “transdiagnostic process” – as may Corry et al. (2013) – it is more parsimoniously described as transontological, where arousal, affective and sociological domains intersect in the genesis of bipolar spectrum phenomena. Thereby, the distal withdrawal arm of the dynamic model is realised.

Examination of the full cohort did not find a statistically significant mediation effect for MSPaM when modelling prestige (nlogSIPS) using TENSION (Sobel z-value 0.28, \(p = .780\)). In the subset of (hypo)manic cases (Table 27) \((n = 23)\), the \(p\) value for the C component (TENSION into prestige) was only .212, indicating that TENSION had no effect on prestige and the Sobel z-value was non-significant at 0.93 \((p = .355)\). It has been noted, however, that mood elevation (ASRM) significantly moderated the relationships of both TENSION (Table 25) and MSPaM (Table 26) with prestige (nlogSIPS). As ASRM increases to 10 (Figure 15) there was an evident positive relationship between TENSION and prestige (this also was seen with MSPaM and prestige), while with an ASRM of 0 the reverse pattern was observed. In the (hypo)manic cohort utilised (which is largely a GP sample), ASRM scores are loaded near the threshold – 12 of 23 scores at or below 6 or 7 and therefore do not show a positive correlation. Yet at an ASRM level of 10 (7/23 cases at 10 or above)
there is a strong positive relationship between TENSION and prestige, with a positive $r_p$ of 0.53 ($n = 7$), but given the low $n a p$ of only .284. Thus, in states of moderate to high (hypo)mania (as would be seen in a mental health service population), there appears to be a relationship between TENSION and prestige, although a low $n$ makes this difficult to fully substantiate. There is therefore support for the stem and the Withdrawal branch of the dynamic model, but evidence is equivocal for the Approach relationships.

Mood elevation was a moderator of Approach relationships rather than a mediator, explaining the lack of correlation between TENSION and ASRM ($r = 0.01$). This conundrum is reflected in the literature – where DSM-5 (American-Psychiatric-Association, 2013) lists irritable mood as a possible facet of mania or hypomania. Irritability may often arise out of anxiety-tension. Perlis et al. (2009) report that, in the context of a major depression study, irritability mostly occurs with anxiety and was more frequent in those with comorbid anxiety disorders. Malhi and Berk (2014) state that “… in the context of bipolar depression irritability is a common symptom that often reflects underlying anxiety or personality factors.” In the MOPF study, tension and depression were strongly related ($r_s=0.70$), though is tension-irritability really a feature of hypomania?

Parker (2008) contends that “during a true high, anxiety tends to disappear 'like snow on a summer's day”’ (p. 59). To support this contention, Figure 29 shows the relationship between tension and mood state. It can be seen that the mean TENSION score did not differ between (hypo)mania and euthymia, while the scores for depression, “mixed” (manic) states and mild depression were significantly higher. It is notable that, in spite of the equivalent means, the (hypo)mania group had a wider interquartile range than the euthymic, indicating a broader spread of TENSION scores. When all cases with categorically elevated ASRM’s – (hypo)mania and manic (mixed) – were mapped by dotplot (Figure 30), it was found that six of eight of the cases with high tension arose from S1 and S2 – the clinical nodes. High tension was nominated as being above the third quartile for the control (S5) group. Non-clinical cases had a range of TENSION scores – from what I will call status excitement to a level we may call sub-mania. There was however a trend for more tense (hypo)manic
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individuals to have higher depression ratings ($r_p = 0.42, n = 23, p = .05$) – see Figure 32 – raising the possibility that a degree of *mixity* corresponds with the increase in tension. DSM-5 (American-Psychiatric-Association, 2013) accounts for such an eventuality with its “anxious distress” and “mixed” specifiers for hypomania.

*Sociometer Sensitivity*

“Contingencies of self-worth represent the domains in which success or failure leads to increases or decreases in self-esteem, respectively. Because people seek to protect, maintain, and enhance their self-esteem, contingencies of self-worth serve an important self-regulatory role; people seek out situations and engage in activities that provide opportunities for them to achieve success and avoid failure in domains on which their self-worth is staked. These contingencies also constitute an important psychological vulnerability, however. Successes and failures lead to greater increases and decreases in self-esteem when they occur in domains of contingency. These fluctuations of self-esteem, in turn, are associated with increases in depressive symptoms. Thus, the pursuit of self-esteem, while guiding much of our behavior, comes at considerable cost.” (Crocker, 2002, p. 143)

The path leading from prestige to tension via self-esteem has received support in this analysis. Similarly, there is evidence that tension activates both withdrawal and approach behaviours. Can it be shown that relational value impacts on the dynamic model, through a “sociometer” input (Figure 9)? To address this I have tested two variables which reflect sensitivity to prestige – MSIS and CSW.av – in the dynamic model to see whether they show any evidence of modulating it.

The Contingencies of Self-worth Scale (CSW) (Crocker et al., 2003) measures to what extent self-esteem is contingent on seven domains of life – academic competence, competition, approval from others, family support, God’s love and “virtue”. It breaks from research which has proposed a binary contingent/non-contingent dimension to self-esteem, by suggesting that self-worth is domain specific. The authors, however, posit that there is an external “unhealthy” locus of self-worth, as opposed to an internal “relatively healthy” locus. Though the capacity of the contingencies to predict behaviour is relatively weak, a link to specific life events is claimed (Crocker, 2002). There is a relationship between self-worth contingencies and
unstable self-esteem, in turn predictive of depressive symptoms (Crocker, 2002). Subsequently, Crocker and Knight (2005) have written of contingent self-worth as being “tasty but not nutritious…addictive” and further “pursuing self-esteem by attempting to validate one’s abilities has costs for learning, relatedness, autonomy, self-regulation, and, over time, physical and mental health.” (p. 201). The authors advocate that people should shift their motivation from self-esteem to goals which are “larger than the self” (p. 203). Clearly, Crocker and Knight are seeing the issue as a moral dilemma for individuals who may win or lose their mental stability by good or bad decisions. This appears erroneous – as Barkow et al. (1975) have attested, “man hungers not so much the money and material goods as for prestige” (p. 553). Indeed, Crocker et al. (2003) found that very few (4%) people had no contingencies of self-worth and the MOPF results have shown that prestige approach motivation exists dimensionally between normal and bipolar individuals (Figure 12). Despite these qualifications around theory, aspects of the scale have psychometric and predictive validity and complement the MSPaM and MSIS scales well.

For the purposes of this research the “external” contingencies of competition, acceptance and approval were utilised, as the other factors seemed of limited relevance to the MOPF sample – only a small number were students and “virtue” and “God’s love” seemed like religious rather than psychometric themes. The three contingencies were averaged to establish a single prestige-contingent dimension on the premise that external contingencies depending on social prestige should form a coherent metric. The correlation of each CSW scale with the others ranged between 0.41 and 0.65 and the correlation of each scale with the CSW average ranged between 0.78 and 0.85, suggesting that each formed a consistent domain within CSW.av. The convergent validity of the individual domains was demonstrated by correlations with the MSIS of 0.46 (CSW appearance) 0.44 (CSW competition) and 0.47 (CSW approval). The CSW.av had a significant 0.55 correlation with the MSIS and 0.58 with the MSPaM scale (N = 228) (Table 62).

Table 14 shows the correlation of the sociometer sensitivity variables with those of the dynamic model. The premise for this analysis is that higher sensitivity to prestige fluctuation (as indicated by significant CSW.av and MSIS correlation) may
mediate the dynamic model of mood change. Individuals with sociometer sensitivity should be primed for both mood elevation and depression in the context of the rise and fall of prestige. It should be noted in Table 14 that in the distal reaches of the model (TENSION, MSPaM and depression) that the sociometer sensitivity variables do indeed correlate significantly. While the CSW.av measure correlates with self-esteem (SISE), the MSIS measure does not, perhaps reflecting the fact that social inclusion sensitivity (MSIS) relates more strongly to state affect than global self-esteem. Table 30 (Appendix) is instructive – MSIS does not fulfill the A criterion for mediation when modelling self-esteem using prestige. CSW.av, on the other hand, just fell short of mediation criteria, obtaining a $p$ of .070 on the Sobel test. Contingent self-worth was a major predictor of self-esteem with a $t$ value of -7.37. The greater is the dependence of self-esteem on prestige contingencies, the lower the global self-esteem. This is further reflected in Figure 23, where self-esteem is almost a perfect reciprocal to CSW.av for the spectrum nodes.

Contingent self-worth (CSW.av) was a significant partial mediator effect when modelling TENSION using self-esteem (Appendix Table 29), while MSIS failed a significance test for path A. This indicates that decrements in self-esteem increase tension, partly because self-esteem is contingent on prestige. Similarly, the capacity for TENSION to impact MSPaM was partially mediated by CSW.av, with a Sobel z-value of 5.47 and a $p$ of <.001. Table 32 (Appendix) indicates that TENSION interacted with CSW.av in the prediction of depression score. This is reflected graphically in Figure 19 – revealing that at higher levels of contingent self-worth there is a disproportionate increment in depression score as TENSION rises. What might this mean? We already know that tension predicts depression and that contingent self-worth score correlates with depression score. Yet in this instance, the two factors are interacting at different levels of CSW.av – high values of TENSION and CSW.av pushing depression scores well into the fourth quartile for the spectrum group. Thus, CSW.av moderates the relationship of tension and depression – people with high contingent self-worth are likely to become more depressed than those with low levels as tension rises. The mirror relationship appears to apply – with low levels of tension, persons with high contingent self-worth appear to be less depressed. Perhaps this reflects the value of investing in prestige objects when a person is in equilibrium and
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prestige is available – this protects mood. Once the individual experiences significant
prestige stress, this benefit evaporates and becomes a vulnerability, thereby
potentiating depression.

The MOPF Social Inclusion Sensitivity (MSIS) (http://tinyurl.com/3rqwfa8) (p. 9) measures affective response to prestige events, hinging on social inclusion. In
the Results section I tested the convergent validity of this scale with the BASrr
(reward responsiveness) scale and found a correlation of 0.41. The MSIS Cronbach’s
alpha for the MSIS is 0.80, which reflects adequate internal consistency. The test-
retest reliability of this scale has not yet been evaluated. MSIS was a partial mediator
when modelling MSPaM using TENSION with a Sobel z-value of 5.32 and a p of
<.001, indicating a significant part of the drive to prestige approach motivation stems
from the sensitivity of the self to social inclusion. MSIS was also a moderator, when
modelling MSPaM using TENSION.

There is therefore extensive evidence that the sensitivity to sociometer-
relevant contingencies affects the functioning of the dynamic model through
mediation, moderation and direct action. The dynamic model links variables which
mediate approach and withdrawal reactions, linking relational events with mood. This
is exactly as it should be. Was it shown that dimensional bipolarity has a similar role
on the dynamic tree in the ascent into hypomania and depression?

Bipolar priming in the Dynamic Model

I have previously used the Mood Disorder Questionnaire (MDQ) as a broad
index of bipolarity – useful for both categorical (via threshold effects) and
dimensional spectrum states. The sociometer sensitivity variables have previously
been shown to correlate with the MDQ. As stated, the dynamic model paves a way for
the escalation and de-escalation of mood, as linked to stem variables such as prestige
and self-esteem. In order to investigate this issue, I undertook mediation analyses for
the MDQ when modelling dynamic model regressions and this found that the MDQ
partially mediated the prestige-self-esteem relationship (Appendix Table 33), the self-
esteeem-TENSION relationship (Appendix Table 34), the TENSION-MSPaM
relationship (Appendix Table 35) and the TENSION-depression relationship
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(Appendix Table 36). It is therefore conceivable that one of the mechanisms of action for the bipolarity substrate is the facilitation of approach states and the enabling of prestige withdrawal through the linkage of dynamic model variables. This may thereby provide a conduit linking negative social interactions (and goal-success) – with hypomania and depression.

The Sociometer

This work has drawn extensively on the sociometer concept of Leary et al. (1995). It is pertinent to reiterate the functions which the sociometer is thought to serve: monitoring for relational value; responding with negative affect and lowered self-esteem; prompting a conscious assessment; and launching motivated behaviours to stabilise relational value. This represents a classic homeostatic negative feedback process, which may be designed by evolution to maintain relational value (prestige) at an optimum level. It has been argued that in bipolar spectrum conditions the sociometer is aberrant, thereby facilitating the ascent and descent of affect we see in these disorders. Rather than healthy homeostasis, mood syndromes are the product of a faulty monitor/effecter. Do the MOPF data provide any support or refutation for this thesis?

The aberrant sociometer hypothesis described here proposes that with conditions of changing prestige – be they ever so slight – self-esteem and depression level (as the functions of the sociometer) will change. The relative prestige and self-esteem levels in the five mood states are depicted in Figure 20, where it can be seen that in Euthymia the scores for prestige and self-esteem overlap, and that across the figure the levels trend together. As will be shown, prestige elevates in (Hypo)mania but self-esteem appears to lag behind; this may be an artefact of the SISE self-esteem scale, where euthymics and (hypo)manics both tend to endorse high self-esteem. The scale does not allow for elevated self-esteem. Thus, were we to use a scale that incorporated more grandiose levels of self-esteem, it is likely that prestige and self-esteem scores would correspond even more closely in (hypo)mania. The Depressed cohort has a corresponding low level of prestige, but a disproportionately lower self-esteem. The “Mixed” (manic) and Mild Depression groups have prestige scores equivalent to the Depressed group. These results may be depicted as a ratio of self-
esteem to units of prestige, much as one might relate degrees of temperature to joules of sunlight – measured in different units – but possible to compare their relative proportion. Such an analysis mirrors what has just been described; with the ratios of all groups (bar the Depressed group) falling relatively close to 1. The ratio of self-esteem to prestige for the Depressed group is significantly low at 0.74 ($p = .001, d = 0.65$). We might adduce from this that the sociometer score of the Depressed group is overly sensitive to the fall in prestige relative to the Euthymic group. This is reflected in the disproportionately low self-esteem. I therefore infer that both the monitoring of relational value (prestige) and the “output” to self-esteem change in depression. Does this extend to an increase in depression relative to relational value (prestige) in these states?

Figure 21 is a standardised plot of prestige and depression score by mood state. We see that depression score is low in Euthymia and (Hypo)mania, while escalating in Depression and “Mixed” (manic) mood states relative to a low prestige level. It is notable that while depression scores differ between Depression and Mild Depression, prestige scores are roughly equivalent. That is, some people (Depressed cohort) have a depressive response disproportionate to level of prestige. This is therefore in support of the postulate that the sociometer – in response to low relational value (prestige) – triggers a depressive reaction in susceptible persons.

Given this finding, does it lead to prestige-motivated cognition and behaviour as the sociometer hypothesis proposes? In order to assess this we again follow a methodology of comparing the relevant variable – MSPaM – to prestige level across the mood state categories (Figure 22) Low prestige states are accompanied by a high MSPaM and the relative ratio of prestige approach motivation (MSPaM) to prestige reveals a peak for the “Mixed” (manic) category, with a mean ratio of 42.93 compared to that of Euthymia at 21.85 – the difference having a Cohen’s $d$ of 1.71. An ANOVA (Appendix Table 37, Table 38) found significantly elevated ratios of MSPaM/Prestige for the Depressed, “Mixed”(manic), and Mild Depression group ratios also. These data suggest that as prestige levels fall relative to Euthymia with each depressive/”mixed” mood category, there is a compensatory response in MSPaM. Low prestige is associated with depression and elevated MSPaM levels; this is most
marked in the “Mixed” (manic) category. The “Mixed” ($p < .001$) and the Depressed ($p < .001$) categories are also significantly higher than the (Hypo)manic category – this will be considered later in relationship to mania. I therefore tentatively conclude that prestige (relational value) changes are indeed monitored and that significant decrements lead to a fall in self-esteem, an increase in depression and an increase in prestige-motivated cognitions and behaviours (MSPaM) in a bipolar spectrum sample, as the sociometer model would predict.

Does the bipolar spectrum have measurable differences in the self-esteem/prestige sociometer ratio? It would be tempting to think that spectrum groups with higher bipolarity would have more finely tuned sociometers. In order to test this, an ANCOVA with depression (PHQ-9) as a covariate (control) was performed. As noted in the results, this found no difference between spectrum groups for sociometer ratio. Therefore, the results described appear to exclusively relate to mood state and not spectrum node. The sociometer ratio indicates self-esteem relative to prestige – a construct similar to that of the Contingencies of Self-Worth Scale (CSW.av) – where self-esteem relative to socially perceived appearance, approval, and competition is measured. The two measures were found to correlate at the -0.36 level (Pearson), indicating convergent validity for the models.

**Validity of the (Hypo)manic Category**

Because 19 of the 23 (hypo)manic individuals arose in general practice, it is conceivable that they are not valid cases. Altman et al. (1997) originally claimed a 0.85 sensitivity and 0.87 specificity for his self-titled scale, based on psychiatric inpatients. Again working with inpatients, the group claimed that the ASRM was the best scale in screening for acute symptoms, with a sensitivity of 0.93 and a specificity of 0.33 (Altman, Hedeker, Peterson, & Davis, 2001). Rucci, Calugi, Miniati, and Fagiolini (2013) note that while no scale can replace clinical diagnosis, the ASRM alpha for mania was 0.79 and the correlation with observer-rated scales (MRS and CARS-M) was 0.718 and 0.766 respectively. The scale is used increasingly in the United States to monitor mood symptoms over time. Recognising a broad community representation for soft bipolarity – if not necessarily full clinical (hypo)mania – the MOPF study aimed to be inclusive of cases of suprathreshold mood elevation. Perusal
of Figure 31 reveals that in the cases designated by the ASRM scale as (hypo)manic, that MDQ scores cluster in the sub- and suprathreshold segments of the dotplot with a mean MDQ of 6.98 and 3 negative outliers (the bipolar threshold being 7.00). This is evidence of convergence of the ASRM (hypo)mania category with the MDQ scale in spite of the community recruitment. The yield rates for the MDQ in general practice are larger than those for structured clinical interviews; the MDQ predicting a bipolar case prevalence between 7.6% and 9.8%, while structured interviews nominating between 0.5% to 4.3% of interviewees as bipolar (Cerimele, Chwastiak, Dodson, & Katon, 2014). This suggests that the instrument may either inflate real rates, or else detect more subtle forms of bipolar spectrum disorder – or do both.

**Social Processes**

“…humans had in some unique fashion become so ecologically dominant that they in effect became their own principal hostile force of nature, explicitly in regard to evolutionary changes in the human psyche and social behavior…the real challenge in the human environment throughout history that affected the evolution of the intellect was not climate, weather, food shortages, or parasites—not even predators. Rather, it was the necessity of dealing continually with our fellow humans in social circumstances that became ever more complex and unpredictable as the human line evolved. Social cleverness, especially through success in competition achieved by cooperation, becomes paramount…nothing would select more potently for increased social intelligence…than a within-species co-evolutionary arms race in which success depended on effectiveness in social competition.” (Alexander, 1990, pp. 4-7)

**Social Affiliation**

The environment of evolutionary adaptedness (EEA) is not a time or a place, but the selective context which has shaped psychological change over the Pleistocene. It comprises a set of long standing and recurrent survival and reproductive problems – a niche – inhabited by Homo sapiens (Hagen, 2004). The quotation from Alexander provides a challenge to traditional notions of selection by natural environment – locating our social evolutionary context within society itself – and seemingly nominating social inclusion (prestige) as a fulcrum. Flinn, Geary, and Ward (2005) draw on Alexander’s ideas and propose a form of runaway selection, in which more adept individuals can outmanoeuvre their conspecifics to gain ecological resources, as
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well as the control of other peoples’ behaviour. These ideas are reflected in the “social brain hypothesis”, which postulates that the social environment was responsible for increased hominin brain size and hence social psychological mechanisms (Brune, 2008).

Foley (1995) has meticulously attempted to reconstruct the EEA, which selected for unique human mental states, cognitive processes and behaviours suitable for a hunter-gatherer way of life. Such persons lived in small social groups, with low population growth and low reproduction, and subsisting on available natural resources (Foley, 1995); this scenario existing for up to two million years. Indeed, Foley (1995) takes adaptive sociality back further to encompass the behaviour of all anthropoid primates – with the exception of the orangutan. Over the past 100 000 years there has been a dispersal of human groups across the globe and the rapid development of regional technologies. Symbolic behaviour has blossomed and human populations have become denser with the likely fissioning of groups and intergroup conflict. The hunter-gatherer lifestyle was a product of ancestral features and evolving demographic conditions (Foley, 1995).

In chapter 3 I introduced the notion of Hedonic Energetic Social Engagement (HESE), a putative synergistic neural system which mediates the social emotions, sociality, and – in extremis – bipolar mood states. I posited that HESE arises in limbic circuits and is responsible for prosodic speech, gesture, energisation, and emotional tone. It is involuntarily utilised by individuals to socially engage with conspecifics and communicate socially relevant material. It is veritably everywhere we look in our social environment and – alongside rank – it provides the substrate for social investment or prestige. As such, social investment predicts HESE and, in turn, arises from it. The HESE notion was based on the concept of the neural systems PLAY, SEEKING (Panksepp, 1998) and the “Hmmmmm” communication system of Mithen (2005), the latter said to arise 1.8 million years ago in Homo erectus. HESE appears to have been one of the substrates on which sexual selection operated in its “runaway selection” (Brune, 2008; Miller, 2001) of higher cognitive functions. HESE is evident early in development:
At the age of four weeks, human infants have already a strong preference for human face contours. They are responsive to human voices, particularly the mother's voice, and actively seek contact by crying or smiling... The baby's smile and play-face occurs ontogenetically much earlier than in other primates.” (Brune, 2008, p. 77)

Reciprocation (cooperation) is the most important of the ultimate traits for social navigation and relates to both kin selection and the capacity to communicate (McGuire & Troisi, 1998). It is found in all cultures, where it has a socially beneficial role, such that reciprocators may be identified and traded with. “Interpersonal constancy is maintained and close friends are protected from attack. Health and social status is thereby increased as is competitive advantage and psychological regulation. Social networks are maintained and there is a reduction of uncertainty and anxiety.” (McGuire & Troisi, 1998, p. 287). Boyd and Richerson (2009) note that cultural evolution iterates with natural selection to favour genes and practices in which more pro-social motives may emerge. Emotional expressivity – and its automatic reciprocation (Oehman, as cited in Boone & Buck, 2003) – acts as a marker for cooperation (Boone & Buck, 2003; Schug, Matsumoto, Horita, Yamagishi, & Bonnet, 2010). Emotional expression (HESE) is costly as it betrays motivation, though Zahavi (1975) and others have indicated that costly signals are more likely honest signals.

"Based upon a convergence of evidence from a wide range of sources, we argue that trust, or more accurately the communication of trustworthiness, is primarily an affective process and is governed, in part, through the communication of emotion." (Boone & Buck, 2003)

Cooperation has not only direct benefits, but increases the likelihood of mutuality with others through prestige and indirect reciprocity (Rand & Nowak, 2013).

Stevens and Price (2000) have noted the various terms used to denote the two orthogonal systems governing power/rank/dominance and social integration/affiliation/status. Wiggins and Trapnell (1996) chose to contrast agency (power) to communion. Chance and Jolly (1970) termed the affiliative inter-primate process the hedonic mode (as contrasted to the agonic mode) – the hedonic mode
defined as “An affect (q.v.) state of the mind and, at one and the same time, a set of affiliative social relations. Arousal in the individual is low, and with attention, fluctuates as part of the operational activity of the moment. This mode facilitates the exploratory, integrative, and systems-forming aspects of the intelligence.” (Chance, 1988, p. 325). Chance is therefore relating affiliative process to both affect and a set of social behaviours in the genesis of mindful being – analogous to Hedonic Energetic Social Engagement (HESE). Itani (1988) has termed the equivalent process in primates and humans “conditional equality”, judging hunter-gatherer egalitarianism to be a product of its counterpart seen in chimpanzees. In the same volume, Kemper (1988) documents the processes of power and status-accord, originating with Empedocles, the pre-Socratic philosopher, who cited “love” and “strife” as the principles which controlled the elements of earth, wind, water and fire (Kemper, 1988). Strife may be taken to be the process of enacted or agonic conflict which establishes rank, power, or Itani’s inequality principle, in societies. These principles were further enunciated by researchers in the post World War II era, who utilised factor analytic studies. Status-accord (hedonic) describes behaviours which “gratify and enhance the other” (Kemper, 1988, p. 301). The ultimate in status-accord is an open-ended amount of status – named prestige in this thesis – leading to “love”. Thus Kemper extends what is essentially HESE to prestige, in support of this thesis. He further links status (prestige) loss to anger and depression.

Unfortunately, we know more about the bones, physiology, and climate of early man than we do about his psychology and social processes. There is a vacuum in which conjecture abounds – and the prestige model is but one theory amongst many. The model posits a dynamic social structure (Figure 38) involving an ingroup, a marginal zone and an outgroup – this is hardly contentious. Prestige is cited as the “cement” which binds communities – the investment of the group in the individual. Social processes – centripetal and centrifugal – localise the individual within society, responding to forces such as rank and rivalry, gossip (Lomas, 2009), aggression and reciprocity. There is tension acting within the group which acts on the individual’s social inclusion, which in contemporary western societies may be relatively piecemeal – the average individual being able to receive social investment in a number of contexts. For example, I might socialise with a shopkeeper in the morning, come
home to my family, and attend an evening conference and a picnic the following day. All of these contexts provide my psyche with a sense of incorporation – a sense that I will live until the next day. Ancestral hunter-gatherers, however, lived in small bands of non-hierarchical individuals with a more homogenous structure and gossip which permeated all members of the tribe. As such, all of their prestige was invested in the one social locale – all of the “eggs in one basket”, so to speak. Social failures thus permeated the band quickly – which tended to respond with one voice – “you are out!...or you are still in!”. There was simply no place to escape to, except to banishment and the risk of death.

“In ancestral environments, social exclusion from the community was probably one of the most important real threats to an individual, and potentially equivalent to a death sentence. Thus, submission in conflict-laden situations may in the first place be considered a life-saving strategy that evolved under ancestral conditions despite its obvious, though perhaps transient, disadvantage in terms of reproductive success.” (Brune, 2008, p. 211)

Therefore, it is crucial to stress how dimensionally different our evolutionary social structure must have been, yet our psychological nature is equivalent to that which adapted to the environment of evolutionary adaptedness. The modern environment by contrast – with urban living, social disconnection and nuclear families – is “mismatched” (Nesse & Stearns, 2008) to our psychosocial inheritance. Modern environments share little in common with Pleistocene living conditions (Brune, 2008). Theorists such as Williams (2007a) have argued for an ostracism detection mechanism, whilst Leary et al. (1995) have suggested that relational value is maintained by mechanisms centering on self-esteem. Can we interpolate backward to ancestral societies about these processes?
Figure 38. Schematic of social zones associated with prestige. In the ingroup there is a centripedal effect of prestige – which maintains inclusion. Individuals may move to a vulnerable marginal zone and further into the outgroup (centrifugally); which poses a risk of death. Prestige approach motivation and mood elevation allow the individual to prevent ostracism and re-enter the ingroup. Depression leads to “invisible” marginalisation – prestige withdrawal – which also prevents full ostracism.

Social Exclusion

Thomas Scheff (2001) observed the intake interviews of men admitted for depression to a London mental hospital in 1965. He noted a transient lifting of mood when psychiatrists asked the patients about their activities during World War II and observed that it was the experience of reliving memories of belonging to a community that was responsible for briefly resolving shame and depression. He further proposed that the loss of communal belonging may be a subtle or insidious process, such that most patients would not include it in their self-report (Scheff, 2001). In the lexicon of this thesis, these men were suffering from a contemporary withdrawal of prestige,
equivalent to the MOPF bipolar spectrum depressed groups – where prestige (nlogSIPS) was significantly lower than in euthymia (Figure 21). Depletion of the social environment therefore appears to be a potent factor associated with depression. However, reverse causality may also be operant – depression leading to social estrangement. Such men may be said to be responding to prestige contexts, much as is measured in the MSIS scale. Respondents to this measure indicate the extent to which the following situations impact on their core affect; feeling valued, being excluded, being involved, and being included in the group. Such immediate responses may be the harbingers of a mood episode.

Rosenbach and Renneberg (2011) have reviewed clinical and non-clinical research investigating rejection sensitivity as measured by the rejection sensitivity questionnaire (Downey & Feldman, 1996) – RSQ. As defined in this regard, rejection sensitivity is the “disposition to anxiously expect, readily perceive, and intensely react to rejection.” (Downey, Mougios, Ayduk, London, & Shoda, 2004, p. 668). While the context of rejection overlaps with one of the two inputs to the MSIS, the outcome does not – the MSIS focusing on core hedonic affect – while the RSQ centers on what equates to tense arousal. Nevertheless, the MOPF Pearson correlation of TENSION and MSIS is 0.39 (N = 228), suggesting an overlap between the two constructs in the bipolar spectrum population. Rosenbach and Renneberg (2011) note that rejection sensitivity may either be either a risk factor for depression, or that depression may mitigate for rejection sensitivity. Social support may be a protective factor within this model. The rejection sensitivity literature supports the contention of the prestige model that changes in prestige may lead to depressive symptoms in vulnerable individuals. Though the RSQ emphasises tense apprehension and reaction, there may be overlap of tension and hedonic response as portrayed in the MSIS scale in the MOPF population.

Social exclusion is of course a serious predicament for modern man and a catastrophe for ancestral humans. Whilst introjected prestige – self-esteem – is unpleasant, ancestral social exclusion threatened access to actual resources such as food, water, shelter and technologies as used in hunting. Access to family would be curtailed, which rendered the individual unable to play a role in kin selection. Thus
not just life, but genetic heritage, was on the line in these contexts. For these reasons there was an acute need to be sensitive to relational value – and to act accordingly – to survive in the Pleistocene. Engelmann, Over, Herrmann, and Tomasello (2013) have shown that even five-year-olds strategically manage reputation; children share more with an unknown actor when ingroup (but not outgroup) spectators can reciprocate. The same researchers found that the human concern for, and capacity to manage, self-reputation may be unique amongst primates (Engelmann, Herrmann, & Tomasello, 2012). There are therefore two evolved mechanisms for so called “impression management” – an unconscious HESE alongside the conscious motivation to maximize gains and limit punishments (Schlenker, 1980). Both subserve the evolutionary mandate to preserve group engagement and are channeled into three broad categories of response to prestige threat – prosocial behaviour, antisocial behaviour and (our behaviour of interest) social withdrawal (Rosenbach & Renneberg, 2011). Chronic ostracism tends to “deplete coping resources”, leading to helplessness and depression (Williams, 2007a, p. 425).

In accord with the findings of Leary et al. (1995), the MOPF research found a relationship between prestige and self-esteem (SISE) – the Spearman correlation coefficient being 0.35 ($N = 228$) for the whole cohort (Table 13). There was a strong relationship between prestige and self-esteem magnitude in Figure 23 for spectrum nodes (apart from S3). The dynamic model posits that fluctuations in prestige – and threats to it – impact self-esteem, which drives approach and withdrawal reactions. A state of self-esteem contingency (CSW.av) could be said to occur when relational value is threatened – CSW.av should increase in this context. Leary et al. (1995) would therefore predict that self-esteem should thereby fall. The MOPF research found just this – self-esteem falls as CSW.av rises ($r_p = -0.47$, $N = 228$). This is reflected for the spectrum nodes in Figure 23 – the plot for self-esteem shows a reversed (mirror) relationship to CSW.av. Whilst self-esteem did not correlate with mood elevation ($r_s = 0.04$, $N = 228$) – there was no option in the SISE to indicate increased self-esteem – it did correlate with depression ($r_s = -0.52$, $N = 228$), a proxy for subjective social withdrawal. Table 42 shows that the self-esteem profile for euthymia is not discernible from (hypo)mania, while depressive cases are overrepresented in the low self-esteem cells. It was not possible to link self-esteem to
prosocial approach behaviours/affects. The relationship between prestige and self-esteem, as predicted by Leary et al. (1995), is therefore supported, as is the relationship of self-esteem to depressive withdrawal.

Of course social withdrawal does not occur in a vacuum, and Segrin and Abramson (1994) have reviewed what they nominate as negative reactions to depressive behaviours. They observed that depressed individuals reliably experience rejection from their social environment, positing that whilst depressive persons elicit this reaction, the mechanism is unknown. There is a robust association between depression and disrupted interpersonal relationships (Segrin & Abramson, 1994).

Social skill impairment in depressed individuals may lead to negative reactions from people in the social environment, consonant with communication theories (Segrin & Abramson, 1994). Yet other authors have noted that responses to depressed individuals may be positive (especially in mild cases) or mixed: “For instance, Biglan et al. (1985), Hops et al. (1987), and Nelson and Beach (1990) have all shown that depressive behavior reduces the likelihood of aversive responses from family members.” (Allen & Badcock, 2003, p. 900). In fact, Petersen et al. (1993) found a gradient of response by family members to depressed individuals, such that as helplessness increased, family became angry and avoidant of the individual. Segrin and Abramson (1994) stress that interpersonal problems result from the interaction of the subject and his or her partner and that such a focus will lead to greater understanding of the aetiology of depression. One problem with the research reviewed by Segrin and Abramson (1994) is that it does not demonstrate causality. The association of rejection and depression does not prove that depression causes rejection. In fact, the converse could be true, with negative reactions in the family leading to depression in the subject. If, on the balance, rejection (ostracism) leads to depression, then this would support the dynamic (prestige withdrawal) theory of depression pathogenesis. If depression leads to a pervasive rejection response by the family then this poses problems for the theory. Williams (2007b), by way of compromise, suggests that “It is just as plausible that ostracism leads to depression as depression leads to ostracism. It is likely that both co-occur, resulting in a vicious cycle.” (p. 244). An important caveat needs to be made; the depression research cited does not generally differentiate the various forms of depression – the response to a
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chronic dysthymic depression would in likelihood be more negative and entangled than that to a discreet episode of melancholia, where there may be briefer affective communication. Similarly, adjustment disorders involve greater distress – initially likely to engage support – but when long-term and intractable they make likely induce carer despair due to the imperative to assist, when assistance may not work. In fact, due to this fact, and due to the varying evidence, no conclusions can be drawn from Segrin and Abramson’s research to either support, or refute, the stealth hypothesis. Longitudinal research is needed with clinical depressive episodes to measure support and rejection. In the meantime, we should note that rejection and “depression” may co-occur and to not draw linear conclusions before the data are available.

Sasaki and Uchida (2013) have described a computer simulation examining cooperation and social exclusion. They note that costly punishment of freeriders by a lone cooperator barely controls the asociality of freeriders and that punishment is costly for the punisher. They found that social exclusion, on the other hand, can act as a strong incentive to participants to cooperate as opposed to using costly punishment. They concluded that powerful social exclusion, at a cheap cost, can emerge amongst “a sea of defectors and dominate them.” (p. 4). Social exclusion was still effective even in the presence of a short-term negative effect on excluder payoffs. “To resist the exclusion, it is likely that conditional cooperators capable of detecting ostracism [8] evolve.” (p. 6) – reminiscent of Williams (2007a) ostracism detection mechanism. The authors then invoked the notion of an arms race between social exclusion and exclusion detection systems. Though the technicalities of their method are complex, the results of Sasaki and Uchida (2013) suggest that cooperative systems may emerge amongst potential defectors through the aegis of social exclusion. Social exclusion may be an instrument of social sanction and a means of order. To regain group inclusion, individuals in the marginal zone of Figure 38 have options for either prosocial or depressive responses. Antisocial responses are likely to work against them and increase the likelihood of being shunned. Whilst prosocial acts may lead to social incorporation, depression may forestall full exclusion, thereby maintaining life. Social exclusion is thereby linked to adaptive responses which increase social cooperation. Could therefore, melancholia and hypomania be seen as exclusion-related processes of adaptive potential?
Sasaki and Uchida (2013) have experimentally inferred the presence of exclusion detection systems. Is it conceivable that the MOPF research may shed light on this matter? I have previously discussed the MOPF Social Inclusion Sensitivity scale (MSIS) and demonstrated that scores were elevated in nodes S1 and S4 relative to controls when mood was taken into account. In the ANCOVA, S5 was 23.59 and the S7 control 20.05 – this was not statistically significant. However, there were compromises that had to be made in allocating people to spectrum nodes to include relevant cases; those residual cases with a non-response to whether or not they had a bipolar family history were deemed “no family history”. This meant that 3 of 48 S7 control cases (where “no bipolar family history” was inferred) had in fact not responded to the family history question. Instead of using spectrum node as the comparison category, true family bipolar history in the S5/S7 cohort was assayed for MSIS. Therefore, the T-test indicated that a bipolar family history - of itself - carried with it a higher MSIS and a greater degree of affective responsiveness to prestige-relevant events.

Kernis (2005) has documented the limitations of global long-term self-esteem research and highlighted the importance of self-esteem stability. He relates this to the contingency of self-worth, impoverishment of self-concept and excessive dependency needs. These may arise from controlling or harsh family environments. His group has linked high unstable self-esteem with hostility proneness, whereas high stable self-esteem demonstrated the lowest tendencies. The more unstable an individual’s self-esteem, the greater dysphoria reported to negative events and the larger the gain from positive events. With increasing major life stressors, unstable self-esteem predicted increased depression in low (but not high) self-esteem persons (Butler, Hokanson, & Flynn, 1994). MSIS reflects self-esteem stability in the face of prestige-relevant contexts and scores are elevated for the bipolar (S1&S2), pseudounipolar (S3), subthreshold (S4) and unipolar (S6) nodes. A family history of bipolar disorder, in and of itself, is associated with a raised MSIS, and ipso facto, increased self-esteem instability. This curious fact suggests that – in both the literature and the MOPF research – that self-esteem stability is a factor in both threshold and subthreshold disorders. Bipolar genes, even in the absence of documented affective episodes, predispose to this instability. It might be inferred from this that affectivity brings an
innate fragility to the sociometer – which is overly primed to prestige ingress and egress – and a greater liability to affective episodes as a consequence.
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Affect

Adaptation

“...if variations useful to any organic being ever occur, assuredly individuals thus characterised will have the best chance of being preserved in the struggle for life; and from the strong principal of inheritance, these will tend to produce offspring similarly characterised. This principle of preservation, or the survival of the fittest, I have called natural selection.” (Darwin, 1859)

Darwin’s theory of evolution by natural selection predicts that all organisms have one or a few original ancestors. The theory proposes that the lineages of organisms are modified over time by incremental change, such that proportions of individuals with different inherited characteristics will arise – variation. Changes in these proportions occur because of their differential ability to survive and reproduce (Futuyma, 2009). Such changes lead to the emergence of adaptations – “features that appear to be ‘designed’ to fit organisms to the environment.” (Futuyma, 2009, p. 8).

Adaptation, as verb, is the process by which population members become better suited to their environment through a change in a feature which enhances their survival and reproduction (Futuyma, 2009). A reproduction-enhancing feature — as contrasted to alternative character states — evolves in response to a specific selective agent, (Futuyma, 2009). Adaptations may be biochemical, physiological, anatomical, cognitive or behavioural and are successful responses to an ecological or reproductive problem. With respect to psychology and psychiatry: “The more design features that are necessary to solve the problem, and the more such features that the nervous system appears to possess, the higher the probability that a psychological adaptation to solve the reproductive problem in fact exists.” (Hagen, 2004). Adaptations, by definition, occur in the whole population and the heritable variation is generally zero.

It is important to note that prominent evolutionary psychiatrists such as Nesse and Stearns (2008) argue that illness does not arise from adaptation, but through natural selection creating the conditions favourable to disease.

Given that natural selection selects for features which are optimally suited to the environment for the purposes of survival and reproduction, why might deleterious genes exist in a population? Adaptations may malfunction and disturbance may be the
result of noise (Hagen, 2004). Evolution by selection is mostly a “thrifty” process (Brune, 2008) and it does not promise a perfect species. McGuire and Troisi (1998) note that "those features that are preserved are often far from ideal, at least from a psychiatric perspective. Diseases and disorders of all types are everywhere apparent in nature, and the vast majority of species (> 95%) become extinct. Said another way, it is an error to assume that Homo sapiens has been selected to be mentally healthy.” (Preface IX). Thus processes such as exaptation, where adaptations are coopted for other purposes, may lead to an imperfect solution and the appearance of “bad design”.

So called “heterozygote advantage” occurs where one of a pair of genes is advantageous, but two copies are disadvantageous, as famously occurs in sickle cell anaemia under the selection pressure of malaria. Genes which are deleterious later in life such as Huntington’s chorea, but benign in reproductive years, will not be strongly selected against. Genes, such as those causing neurofibromatosis, may continually arise in a population due to recurrent mutation. Disadvantageous genes may arise through migration from areas where the feature is not a problem – eg sickle cell anaemia. Furthermore, the removal of genes by natural selection takes extensive stretches of time (Caldwell et al., 2006). Genes may also change in frequency by merit of chance and in isolated groups this may result in atypicality (McGuire & Troisi, 1998). Polygenic mechanisms may lead to advantage with low gene number, but disadvantage in phenotypes with greater numbers of genes. In pleiotropy a single genetic locus controls two or more evidently unrelated traits, often on the basis of a single mutation (Stearns, 2010). Most genes have a degree of pleiotropy and the number of traits per locus is approximately six to eight (Stearns, 2010). Stearns (2010) further notes that “…synergistic pleiotropy confined to single phenotypic modules may allow populations to rapidly evolve phenotypic novelties that produce new solutions to environmental puzzles.” As such, in pleiotropy “bad” phenotypes may be carried along with “good” phenotypes, producing a deleterious byproduct alongside one or more advantageous phenotypes. This may be the most likely explanation for the persistence of genes which code for psychiatric disturbance – that they also code for traits determining adaptational success (Gonda, 2013).

There is an approximately 80% heritability for bipolar disorder and a substantial genetic correlation between depression and mania, though a set of genes
may be specific to the manic state (Farmer, Eley, & McGuffin, 2005). Seifuddin et al. (2013) have undertaken a systematic review of genome wide expression studies and post-mortem studies in bipolar disorder. They identified 382 genes which were significantly differentially expressed and 11 of these survived corrections for multiple testing – amongst which were FKBP5 and WFS1. Recently, the Cross-Disorder-Group-of-the-Psychiatric-Genomics-Consortium (2013) found that specific SNPs are associated with a range of psychiatric disorders, which include bipolar disorder, schizophrenia, autism, ADHD and major depressive disorder. Specifically, calcium activity gene variation may have a pleiotropic effect on psychopathology. Most research has proposed that bipolar disorder has a polygenic inheritance – each gene contributing an additive effect.

**Depression**

The adaptationist project for affective disorders has a number of obstacles. Firstly, adaptations should show evidence of good design and should be pervasive in the species – lacking heritable variation. The ecological problem leading to the adaptation should be clear and the adaptation should increase fitness (Nettle, 2004). Nettle (2004) emphasises that depression, in particular, shows none of these hallmarks. Depression is generally a chronic or relapsing condition with disability and acute phase cognitive impairments. Family relationships are often strained and the condition often leads to secondary morbidity. Suicide is an eventuality in around 10% of depressed individuals and psychosis may lead to significant reality distortion. Similar qualifications apply to the bipolar disorders – especially type I. Yet cogent roles for depression have been mooted: as an “honest signal to social partners” (Watson & Andrews, 2002); for social navigation regards key social problems (Watson & Andrews, 2002); as a submission signal to mark the end of a rank contest (Price et al., 2007); and as an involuntary means of giving up on the pursuit of an unobtainable goal (Nesse & Williams, 1994). Others have argued for depression as: a means of resource conservation; as a separation protest (Bowlby, 1969); and as a method of social risk minimization when the ratio of social value to social burden is at “a critically low level” – the so-called social risk theory of depression of Allen and Badcock (2003). Gonda (2013), drawing on ideas from Akiskal and Akiskal (2005),
claims that depression is advantageous because it aids pair bonding and group cohesion. Akiskal and Akiskal (2005) further claim that depression leads to caring and support for the sick, young and weak and a tendency to self-denying devotion to others alongside conformity to social values and rules. Having personally met a large number of individuals with depression, Gonda and Akiskal and Akiskal’s claims require greater evidential support. Given the heterogeneity of theories, it can be assumed that the precise ecological problem which depression answers has not been precisely delineated.

The MOPF project found that a state of depression was associated with reduced prestige (Figure 21) – consistent with an impediment to selection. When the cases with a history of unipolar depression (S3&S6) were examined by T-test with respect to bipolar family history, the results suggested that bipolar familiarity may confer two benefits. Firstly, prestige approach motivation is higher in S3 pseudounipolars than the S6 residual unipolars. In effect, by parceling out the otherwise equivalent total unipolar cases into one cohort and then testing by the differentiating variable (FHx) controls for confounding factors more effectively than would an considering each spectrum group separately via ANOVA. Secondly, prestige is higher in the depressive cohort with a family history of bipolar disorder. Together, these results suggest the possibility that “depression is not depression” with respect to selection advantage – having a familial bipolar history may make this pseudounipolar (S3) cohort adaptive. Benazzi (2003) noted that the unipolar group with a bipolar family history made up a clinically significant proportion of the unipolar sample. The pseudounipolar group had many bipolar signs, such that there was an argument to include it in the bipolar spectrum. Is there then a prestige or prestige approach motivation benefit evident for the possession of bipolar genes in the cases that had no disorder?

Pleiotropic genes (or sets of genes) require an adaptive phenotype and a non-adaptive phenotype linked like a dipole. The adaptive end of the imaginal dipole is carried by natural selection, while the disadvantageous phenotype is carried along as a byproduct. If so, perhaps psychosis, suicide, inanition, substance abuse and physical morbidity are the disadvantageous phenotypes linked to depression. What might be
the adaptive aspect to which they are linked? I have already shown that the pseudounipolar (S3) node has increased prestige and prestige approach motivation relative to the residual (S6) unipolar group. Could bipolar family history-bipolarity reflect the adaptive “end” of the dipole? Previous models of depression have been proposed – including “depression as social navigator”, “depression as resource conserver” and “depression as help elicitor”, amongst others. These desirable facets, linked to bipolarity, may represent the adaptive bipolar depressive dipole.

Similarly, the social risk hypothesis of Allen and Badcock (2003) suggests an attractive option for the ecological problem which depression may answer. The authors propose that depressed states developed to minimize social risk, where social value falls relative to social burden. This state poses a danger of social exclusion, which triggers hypersensitivity to social threat, social risk-reducing signals and reduced socially risky behaviours, whereas the MOPF project emphasises the active motivation to re-enter society in these contexts. Allen and Badcock’s model is specific for mild and largely transient depressive states – indicating that this may be the behavioural adaptation selected for by natural selection, though the authors argue that the features should be observable in all states of depressive severity. In response to the failure of clinical depressive states to meet the ecological function of their model, they “…explicitly conceive these states to be examples of an adaptive mechanism functioning outside its adaptive range.” (p. 889). Thus their theory is consonant with the neurovegetative features of depression acting as the negative phenotype coupled to an adaptive “dipole”. The precipitants of depression involve socially contingent experiences which indicate to the individual that their ability to invest in social endeavors is low. Thus the socially “depleted” person may lay low until social conditions are more amenable through increased social value. This is consonant with the notion proposed here that social prestige mediates self-esteem and its sociometer (Leary et al., 1995) behaviours. Allen and Badcock (2003) term their process mediator (and originator of self-esteem phenomenology) social investment potential (SIP). Perhaps my only quibble with their comprehensive and persuasive theory is that there is fluidity in the cohort for whom they ascribe their adaptationist argument, such that one is not sure when they are describing a normal “down” (whatever that is) versus a
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clinically depressed state. Otherwise there is a close synchrony between the prestige model and that of Allen and Badcock.

In summary, Allen and Badcock (2003) have proposed a cogent adaptationist model for depression, amongst other models. Yet, given the existence of non-adaptive aspects of depression, problems remain for this project. The MOPF data lend support to the notion that some depressive states are adaptive – for instance those with a bipolar family history. This is attested to by the elevated prestige and prestige approach motivation in the pseudounipolar (S3) cohort relative to the unipolar (S6) cohort. The adaptive facet of the bipolar phenotype remains elusive, however. It may be easier to argue that hypomania, rather than depression is adaptive and it may be that depression is carried with hypomania as a byproduct. Yet the pseudounipolar (S3) group has never been hypomanic, yet appears to be adaptive. Perhaps such advantage relates to the shorter duration of bipolar depressive episodes? Perlis, Cusin, and Fava (2014) for instance found that the presence of at least two manic symptoms in a major depressive cohort was associated with earlier remission. Yet many bipolar II persons spend much of their life depressed – though with episodic hypomania the reproductive advantages of this group may have become evident in evolutionary contexts. Perhaps the pseudounipolar cohort has a different type of depression; one which allows for advantage. These questions remain as yet unanswered and await more real world clinical research.

The bipolar spectrum

This thesis is developing an argument that the affective disorders may have arisen as a result of natural and sexual selection. The notion of byproduct formation has been adopted to account for the occurrence of disadvantageous clinical syndromes. Current genetic evidence favours the polygenic inheritance of a range of both clinically overlapping (MDD, bipolar, schizophrenia) and distinct (autism, ADHD) psychiatric syndromes. The potentially adaptive functions of depression have been considered and evidence from the MOPF research for the possible advantage of bipolar family history to depression cited. The social risk hypothesis of Allen and Badcock (2003) has been given credence as a possible mechanism for the natural selection of the depressive phenotype.
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In *Arguments for the genetic basis of the bipolar spectrum* Kelsoe (2003) paints a broad canvass of genetic factors in bipolar spectrum inheritance. He cites the repeated finding of cases of mood disorders in the families of bipolar probands, but notes that a range of spectrum diagnoses are commonly observed. This leads to the suggestion that these conditions are genetically related via a quantitative trait. The spectrum phenotype “is neither strictly quantitative or qualitative, but a combination of the two.” (Kelsoe, 2003, p. 186) and epistasis – the interaction of genes – seems likely. A mixture of specific and non-specific genes is mooted in the origin of overlapping affective phenotypes. The environment is modelled as a contributor in a number of scenarios. Kelsoe (2003) posits a multiple threshold model of disorder vulnerability, where as bipolar gene number increases, thresholds are passed from adaptive temperaments to bipolar II then bipolar I then schizophrenia. Yet the clinical difference in phenotypes led Kelsoe to believe that some of the variance is not only quantitative, but complex and qualitative.

Affective temperaments such as cyclothymia and hyperthymia may play a role in the disposition to affective syndromes (Akiskal & Akiskal, 2005) – the genes being evolutionarily useful. As such, adaptive traits should be more common in “dilute” forms of illness than in the normal population. Evans et al. (2005) sought to test this hypothesis by comparing bipolar families with controls across a range of temperament measures. They predicted that a gradient of temperament scores would exist, ranging from: bipolar subjects; to persons with unipolar depression; to unaffected relatives; to controls. They found that the groups were different in the direction they predicted, with the exception of the hyperthymic temperament. They concluded that their data support the hypothesis that some of the temperamental dimensions are transmitted in families in a quantitative manner, consistent with a broad bipolar spectrum. The hyperthymic temperament was higher in controls than any family group – a result not explained by the theory. Akiskal and Akiskal (2005) postulate that the adaptive role of a number of temperaments, including the anxious worrying temperament, subserves altruism and potential kin protection and that cyclothymia assists romance and creativity. They conclude: “In an oligogenic model, the constituent traits of this illness may have emerged for survival and adaptive roles in the evolutionary past of our species. These roles include, among others, exploration, territoriality, leadership,
social organization, mate selection, and caring for others.” (p. 237). Despite the attractiveness of this theory, it is a long way between self-reported personality traits and demonstrating evolutionary mechanisms, as applies also to the MOPF project. Despite an important negative finding, the work of Evans et al. (2005) goes further, given that a testable *a priori* hypothesis is being examined.

Akin to the theoretical hunch of Evans et al. and utilising a prediction about an affective temperament – MSPaM – I also sought to test whether there is a gradient of scores across a spectrum of increasing “bipolarity”. Would those with higher levels of bipolarity demonstrate higher levels of prestige approach motivation? Figure 11 schematizes the relationship of MDQ score and MSPaM, indicating a rough parallel of these metrics as the bipolar spectrum is traversed. Certainly, in ANCOVA, the raw MSPaM score for the bipolar I (S1) node was some 50% higher than that of the controls and the scores reflected a stepping down in MSPaM magnitude from S1 to S7 (Table 8) (Le Bas et al., 2015). This putative relationship was tested by ANCOVA (Table 8, Table 9, Figure 12), which found that the predictors SPECTRUM, depression (PHQ-9) and mood elevation (ASRM) together accounted for 26.92% of the variance in MSPaM. Depression and mood elevation had been selected as covariates because I wanted to make a finding which was as independent of affective state as possible. It is clear from Table 9 that it is not a smooth gradient – S1 is greater than S2, but S4 is not greater than S5 – et cetera. It is notable that the predicted difference of MSPaM temperament was found for bipolar I and II nodes and arguably for the pseudounipolar (S3) node also. As already noted, T-tests in the common cohorts of “depression diagnosis” (S3&S6) and “extended normality” (S5&S7) have shown evidence of a gradient (Le Bas et al., 2015). Yet what might this signify?

Prestige approach motivation signifies the extent to which an individual is motivated to achieve the investment of others –Figure 38 – one of the centripetal vectors to group inclusion. Like the affective temperaments which Evans et al. (2005) tested, I believed that the motivation to group inclusion was a core feature in bipolarity and sought to demonstrate a “dose-response” relationship in the bipolar spectrum. Like Evans et al., my grouping included a genetic dimension, in the belief that affective temperaments should be polygenically determined according to
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threshold effects (Kelsoe, 2003). Yet why should MSPaM be linked to bipolarity – except by reason of a clinical hunch? The answer is ostensibly prestige, as the intentional object of approach and withdrawal affect as seen in hypomania and melancholia. My reading on emotion science now led to a synthesis of Ekman (2007), Panksepp (1998), Thayer (1989) and Chance and Jolly (1970), forged by Stevens and Price (2000) – the Prestige model of Spectrum Bipolarity. Yet what might the MSPaM scale have to do specifically with prestige and generally with this integrationist theory? The factor analysis of the MSPaM scale revealed dimensions pertaining to negative esteem affect, positive esteem affect and positive esteem action. It is clear that the factors reflect two poles of esteem/affect with a corresponding pole of action around positive social investment. It was negative esteem affect which best predicted a categorical bipolar disorder diagnosis (Table 10). Negative esteem affect entailed not only a low affect and low self-esteem, but the need for social prominence – the latter feature reflected in all three factors. Affect and action in bipolar disorder, whether elevated or depressed, intend prestige elevation or depression. For these reasons it is logical to look to a prestige approach motivation temperament to examine multiple thresholds of spectrum bipolarity.

Epigenetics

“Despite intense research efforts, genetic loci that substantially account for disease heritability have not yet been identified. Over the last several years, epigenetic processes have emerged as important factors for many brain diseases, and the discovery of epigenetic processes in germ cells has raised the possibility that they may contribute to disease heritability and disease risk.” (Bohacek, Gapp, Saab, & Mansuy, 2013, p. 313)

Bohacek et al. (2013) note that epigenetic marks may propagate across generations in two ways – one that is germline independent, involving social and behavioural transmission, and another completely dependent on the germline. Epigenetic regulation occurs throughout the CNS, facilitating complex neuronal processes including memory and the “persistent remodelling of the stress axis in response to adverse or stressful experiences in early life…” (Bohacek et al., 2013, p. 313). It is therefore conceivable that childhood adversity may contribute to the generation of bipolar disorder in probands through epigenetic mechanisms. Bohacek
et al. further suggest that such detrimental effects may be transferred to offspring through the methylation of sperm or posttranslational modifications of histones and protamines within sperm chromatin. In addition, mRNAs and snRNA are found in sperm cells and may be transferred into the oocyte. Major life events and traumatic stress may therefore be passed on through the germline (Bohacek et al., 2013). By way of example, unpredictable maternal separation in rodents provides a proxy for child neglect in humans and Bohacek et al. (2013) cite studies where pups subject to this stress develop depression-like behaviours and other abnormalities as adults. Quite remarkably, persistent molecular changes in the stress and serotonergic pathways are demonstrable. Transmission has been tracked to the third generation – were these mechanisms reflected in bipolar humans they may mimic patterns of apparent autosomal genetic transmission.

The MOPF research assayed bipolar disorder diagnosis and the childhood experience of abuse or neglect (PCRT). The latter was a yes/no response and collapsed the two forms of childhood privation because of their common cooccurrence and shared valence and because together they described developmental relational trauma. 191 of 228 respondents answered this question, with a high response rate for the clinical nodes (S1, S2, S3, S6), but not the subthreshold (S4), simplex (S5) or control nodes (S7). That is, the less clinical the respondent, the less likely were they to answer. Perhaps because this was overtly a mental health questionnaire they did not want to be sullied with ascriptions of child abuse. Conversely, mental health patients may be more likely to affirm or deny a connection between their condition and childhood experience (whether positive or negative), making them more avid to record a response. There is no doubt that this question is broad, impressionistic, retrospective, and open to interpretation. For these reasons, alongside the response rate issue, it is important to treat the results with a modicum of caution.

Given the caveats mentioned above, one more needs be highlighted – the effect of mood on the estimation of childhood experience. We might predict that depression could colour the individual’s perception of their upbringing, as the depressed patient recollects instances of negative treatment from caregivers. On the
other hand, negative early life experience is a recognised antecedent to depression. A T-test was undertaken to investigate the cross-sectional relationship between perceived childhood relational trauma (PCRT) and depression score. This found that persons with PCRT had a mean depression score of 12.34 (making the bulk of them depressive cases), while those not reporting PCRT had a mean score of 7.55 – the 95% CI of the difference being [2.25, 7.34], $t(189) = 3.72$, $p < .001$ and $d = 0.59$. Clearly, there is an association between perceived childhood relational trauma and depressive mood in this bipolar spectrum cohort, though the direction of causality may be bidirectional. It is possible therefore that depression colours childhood recollection.

The Prestige model posits that the evolutionary experience of relational trauma involved in ostracism has selected for responses such as mood elevation and depression. Those that were able to maintain prestige through mood processes were likely to survive and reproduce. I found that perceived childhood relational trauma (PCRT) was associated with MSPaM – the $d = 0.56$ and the $p$ being <.001. In addition, social inclusion sensitivity (MSIS) was increased in those who indicated childhood relational trauma, with a similar magnitude of effect. These combined results suggest that the process of neglect and abuse may sensitise the individual to group inclusion threats and motivate them to augment their prestige. The association between a family history of bipolar disorder and PCRT (Appendix Table 39) suggests either a developmental or genetic genesis to the perception of abuse or neglect in vulnerable families. Curiously, PCRT in the spectrum cohort related specifically to a proband diagnosis of bipolar disorder and not to a depressive diagnosis, suggesting that prestige processes may indeed be at play (Appendix Table 40). McGuffin, Katz, and Bebbington (1988) found that not only did depression aggregate in families, but that recent life events did also, while El Kissi et al. (2013) found that life events aggregate in bipolar families. PCRT is a partial mediator of the relationship between bipolar family history and bipolar caseness (Appendix Table 54, Table 55, Figure 37), indicating that abuse and neglect appear to interact with bipolar family history in the genesis of bipolar disorder. Social inclusion sensitivity (MSIS) has been shown to be related to the bipolar illnesses and may affect the perception of life events.
In terms of classic genetic inheritance, traditional estimates rate the heritability of major depression at around 40 per cent (Joyce, 2009). Childhood maltreatment is said to double the risk of depression (Craig, 2010) and I have discussed increased depression scores in those who described childhood neglect or abuse. Are those increases in depression spread through the spectrum group or confined to one diagnosis? Table 40 (Appendix) indicates that there is an overrepresentation of perceived childhood relational trauma (PCRT) in bipolar disorder. This finding does not extend to depressive disorder, where the rate is as expected. Those with no diagnosis were significantly underrepresented with respect to PCRT and 64 of 67 recorded no PCRT. These results suggest that in this bipolar spectrum cohort, that PCRT and bipolar disorder have a strong and specific association. However, in a review which included articles describing “affectionless control” in unipolar and bipolar conditions, Alloy, Abramson, Smith, Gibb, and Neeren (2006) found fairly consistent evidence that this form of parenting was associated with unipolar depression. The authors found “suggestive but less consistent” (p. 58) support for the association of this form of parenting and bipolar disorder. Cumulative disadvantage is more strongly associated with adult depression than any single developmental variable in isolation (Joyce, 2009).

Depression

"In all, to interact socially is to engage in a highly complex and subtle process that, during any extended interaction, continually undergoes modification—a process in which the average individual engages during approximately 40% of his or her waking day, and a process whose success is contingent on the efficient operation of multiple infrastructures. Given this complexity, it is no wonder that minor system alterations (e.g., missing connections, reduced neurotransmitter levels) have disruptive effects." (McGuire & Troisi, 1998, pp. 169-170)

“In mourning it is the world which has become poor and empty; in melancholia it is the ego itself.” (Freud, 1917)

Clinical depression is a disorder manifested by anhedonia and lowered mood associated with reduced self-esteem, guilt and social withdrawal. There are associated cognitive, biological and behavioural symptoms. It forms a part of both bipolar and
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unipolar conditions and may be triggered by psychosocial events. Following on from my thesis that HESE is central to affective disturbance, one typically finds the following in clinical depression:

1. reduced social engagement
2. psychomotor slowing
3. reduced goal pursuit
4. diminished hedonic activity
5. reduced hedonic display
6. reduced prosodic patterning
7. psychic inhibition
8. appetitive inhibition

Clearly clinical depression has a communicative function which reduces social engagement, though this may or may not be an ultimate relationship. The individual with depression is no longer acting centrally in their polydiadic group and the neural system HESE is postulated to be underactive. Social engagement signals are reduced, leading to impaired prosodic, motoric, gestural and affective signalling. Emotions show a reduced range and, rather than enjoyment, amusement and excitement, the individual displays sorrow. Such output leads to reductions in investment (prestige) from dyadic and polyadic groups. In turn, social withdrawal is mirrored by social invisibility as the process of prestige withdrawal sets in. In accord with Allen and Badcock (2003), the affected individual is attuned to social risk and communicates reduced personal threat to others, whilst avoiding the risk of exclusion. The agency of the individual is therefore reduced as they tread water socially. The condition should cause distress and impairment according to DSM-5 and should be pervasive and persistent (American-Psychiatric-Association, 2013).

**Self-esteem**

Self-esteem is a central component of the dynamic model’s stem, and forms an intimate relationship with prestige; the two are acted on by prestige-related events.
through the sociometer. From time immemorial, depression has been noted to be associated with low self-esteem, though the causal relationship between the two has not been clear. In the spectrum population there was a -0.52 (n =228) Spearman correlation between depression (PHQ-9) and self-esteem (SISE) (Table 13), indicative of a moderate relationship. It can be seen in Figure 14 that self-esteem and depression have a mirror relationship to each other across the bipolar spectrum – where one is high the other is low. While TENSION carried a large portion of the partial mediation between self-esteem and depression, the direct (C’) link between self-esteem and depression remained significant. In this thesis, self-esteem is treated as an introject from prestige – the Spearman correlation being positive 0.35 (Table 13). Clearly, they are related but not identical. It is evident that biological, developmental and personality factors make a significant contribution to self-esteem. In Figure 2 self-esteem is considered as the central locus of an explanatory circle including life events (macro and micro), cognition, subthreshold mood, and social support, driving the vulnerability to mood episodes. Unfortunately the MOPF data shed no direct light on life events or negative social interaction and was therefore not able to further clarify sociometer function. The data at hand make a strong case for a relationship between self-esteem and depression, supporting both clinical experience and the tenets of the dynamic model. Further, Sowislo and Orth (2013) found in a meta-analysis that the effect of self-esteem on depression was significantly greater than the effect of depression on self-esteem. The vulnerability rather than the depressive scar model was supported. Symister and Friend (2003) found that self-esteem was a mediator between social support (a proxy for prestige) and level of adjustment. Pavlickova et al. (2013) found that momentary self-esteem was associated with both depression and mania. Self-esteem, reflecting the interaction of HESE and prestige, appears to be a central pivot in the ascent to depression.

**Tension**

Just as depression is a state of mind, so is it a state of body. As seen in Figure 29, TENSION is high in depression when compared to euthymia. Tension is the substrate for the Fight/Flight System (FFS), originally conceived by Cannon in 1929 and mapped by Gray to involve the midbrain, amygdala, hypothalamus and the central
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grey matter. When stimulated, the circuit produces alarm and escape responses which mirror panic in humans (Durand & Barlow, 2005). Bracha et al. (2004) have noted that there are, in fact, four stages in Cannon’s FFS: freeze, whereby the organism becomes hypervigilant; followed by flight; which precedes fight; and ending in fright, where tonic immobility eventuates. It may be argued, therefore, that tension, as measured in the MOPF survey, indicates preparedness for some kind of action. Given the extreme levels of tension that the depressed responders manifest, what action could they be preparing for? To what are they attuned and why? It may be that they are in the process of generating a “cry for help” or that social rumination is leading to worry and action-readiness. The rank contest of Price et al. (2007) could still be having physiological effects, or a state of loss could be at play. Alternately, tension in the depressed subject could arise from social risk. Such a state results from the risk of exclusion due to social burden and entails hypersensitivity to signals of social threat along with an active inhibition of risk-seeking behaviours and signals. In bipolar I (S1) and unipolar (S6) subjects the correlations of depression (PHQ-9) and TENSION were 0.65 (n = 53) and 0.64 (n = 37) respectively.

TENSION is linked to self-esteem; as self-esteem falls tension rises – the correlation being -0.47 (Table 13). Conversely as individuals demonstrate a higher self-esteem there is a corresponding fall in tension. This strong relationship between disparate psychological variables suggests a fundamental relationship and this has been linked to the influence of negative social interactions (Lakey et al., 1994) and expressed emotion (Miklowitz & Johnson, 2009). We have already seen in path analysis that TENSION fully mediates the relationship of self-esteem and MSPaM while it partially mediates the relationship between self-esteem and depression. Therefore, tension appears to be providing a common pathway to both approach and withdrawal reactions to changes in prestige and self-esteem. How can one variable mediate such opposing responses? Presumably, other factors involved in the generation of affective swings tip the balance; determining how an individual responds to prestige stressors. So-called approach tension seems also to have a different flavour to withdrawal tension. In the former individuals may be more “keyed-up” and anticipatory, while in the latter the affect is more likely one of dread and worry. The intentional emotional tone – prestige approach motivation – appears to
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colour approach (tension) motivation, while depression colours withdrawal (tension) motivation, both giving tension a different phenomenology and experience.

Angst, Gamma, et al. (2011) suggest that trait anxiety may in fact mediate the link between developmental adversity and chronic mood disorder. Merikangas et al. (2007) found that close to ninety per cent of individuals with bipolar disorders I and II had comorbid anxiety disorders. Bipolar Spectrum disorders involve disturbance in prefrontal and limbic network activity along with autonomic nervous system disturbance (Outhred et al., 2014). Increased heart rate, indicative of increased arousal, characterises BSD's and there are also decreases in heart rate variation, which may relate to mania and depression sensitivity. Galvanic Skin Response magnitude seems to be a trait marker of BSD's, however phasic responses do not relate directly with the disorders (Outhred et al., 2014). Unfortunately this evolving physiological literature does not emphasise differences in measured arousal at different phases of the illness. Lastly Thayer (1989) has described interaction between tense and energetic arousal, such that both covary at low levels but mutually inhibit each other at high levels. Therefore there is an intimate relationship between bipolar disorder and tension, described in a range of contexts. Further, the interrelationship of tension and energy – and its hedonic partner – strongly suggest relevance to the clinical conditions.

**Prestige**

The dynamic model presupposes a feedback loop between dynamically impaired prestige states and two compensatory arms – the approach arm intended to drive up prestige and the withdrawal arm intended to diminish prestige to a preset point and thereby “fly under the social radar” till conditions improve. Absent social support (prestige) has a more significant effect on mood than the buffering effects of positive social support (Craig, 2010). Prestige is thereby governed by a system input and an output, levels being metered by the sociometer. The withdrawal state intends prestige withdrawal, once categorical levels of depression have been achieved. This is evident in Figure 26, which compares prestige with levels of depression and shows that up until a PHQ-9 score of 10, prestige falls as depression increases. Once the
subjects show clinically significant levels of depression, the prestige score asymptotes at a low level (the nlogSIPS in euthymia being 3.42 and in categorical depression it was 2.90) and mean prestige levels remain steady despite increasing levels of depression. Having said this there is a significant spread of prestige scores at these levels of depression. In the spectrum cohort overall, the Pearson correlation coefficient between prestige (nlogSIPS) and depression (PHQ-9) was -0.32 (Table 13), while the Spearman correlation in the withdrawal group (PHQ-9 ≥5) was only -0.08. I have taken this to suggest that in the categorical depressions there is a “destination” for depressive increases – as depression increases prestige does not continue to fall. I have termed this destination stealth.

**Stealth**

The term stealth originated from the old English to steal – *stǣlth* and implies covert or furtive action or theft. One definition is “Having or providing the ability to prevent detection by radar: a stealth bomber; stealth technology” (American-Heritage-Dictionaries-Editors, 2011). Stealth actions are hidden, even from the most powerful of scrutiny. On this basis, affective stealth is a metaphor for the social consequences of melancholia, where social withdrawal and riskless signalling place an individual in a covert position. There they can be in a form of stasis, interacting little and therefore failing to further threaten their already impaired prestige. Social processes will happen around them, but not impact them dramatically and ostracism will not eventuate. Where an individual no longer contributes to their own prestige, stealth steals the power of ostracism by presenting the depressed individual as a motiveless target. As Allen and Badcock (2003) suggest, this phenomenon occurs because the individual perceives that their social value to social burden ratio has fallen to a critically low level. This is analogous to triggering of the sociometer (Leary et al., 1995) to contexts of falling relational value, where the behavioural output is unique to those with depression. The consequence is a type of labour strike where there is a loss of interest in virtually all activities (Hagen, 2003).

MSPaM initially rises as depression scores increase (Figure 25) and as already noted, this was most marked in the control (S7) group – moreso than the subthreshold
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(S4) and even more so than the combined bipolar group. Yet the unipolar (S6) withdrawal group does not muster a correlation between depression and MSPaM. What might this mean? Firstly, the control group result – more marked for the euthymic than the mild depression persons – suggests a fundamental relationship akin to homeostasis; as mild withdrawal reactions (with increments of PHQ-9 of only around 2-3) occur, the psyche compensates with approach (MSPaM). This approach is strongest in those without hint of bipolarity. With increasing bipolarity – moving from the subthreshold (S4) to the combined bipolar (S1, S2) cohorts, the magnitude of the compensation (MSPaM) increases, but the correlation wanes like an asymptote. Yet the unipolar (S6) group does not muster a relationship between approach and withdrawal.

The largely bipolar depressed group, shown in Figure 33, has an admixture of approach and withdrawal. This is at odds with the risk-aversive ascription to depression of Allen and Badcock (2003). The negative esteem affect factor is significantly elevated in bipolar disorder and indicates a depressed, threatened cohort with low self-esteem who still strive for group inclusion. Bipolar depression, on this account, indicates a motivation to re-enter society and may represent a “time-out” rather than a unipolar “hibernation”; it may signify the combined effects of prestige withdrawal processes and prestige approach motivation. Bipolar depressions are briefer, may be part of cycling and are often mixed. This ambivalent disposition may be reflected in atypical reactivity.

Triggering

Depressive reactions in the context of the bipolar spectrum rarely happen out of the blue – between 66 and 90% of depressive episodes may be linked to a significant event in the preceding six months (Craig, 2010). There is a suggestion that with advancing illness course episodes may become semi-autonomous (Post & Weiss, 1998). For this reason – when considering the aetiology of depression – it is important to look for psychosocial triggers. May these triggers entail a unifying theme? Brune (2008) has noted that forms of depression in young monkeys result from separation

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\(^9\) Compare with the findings of Miklowitz and Johnson (2009) who also found such a relationship, which was supported by limited evidence.
from their mothers – a break in the attachment system. From a human developmental perspective, he describes the phase of despair which ensues in parental separation, with increasing “hopelessness, withdrawal and disengagement” (p. 79), resembling depression or mourning. In severely depressed adults there is often a cessation of social investment and depressed persons typically fail to converge with interviewers regards expressive behaviours and increase their non-verbal submissive signals (Brune, 2008). Brune (2008) further opines that: “Depression, in this line of reasoning, represents the extreme of submission or appeasement strategy, however inappropriate in terms of context, duration and/or intensity compared to adaptive submissive behaviour, and occurs foremost in situations associated with acute or chronic social stress.” (p. 212). He goes on to note the primary social or interpersonal context of depression, rather than that due to potential losses in non-social domains. As Allen and Badcock (2003) have suggested, Brune (2008) contends that depression is a “pathological extreme of harm-avoiding strategy in socially competitive interactions.” (p. 212).

Depression represents the intersection of low self-esteem, adverse environmental factors, life events and lack of social support (Craig, 2010). Sentinel research by Brown, Harris, and Hepworth (1995) has found that humiliation and entrapment are the predominant life events causing depression. Similarly, Kendler, Hettema, Butera, Gardner, and Prescott (2003) found that the onset of depression was strongly associated with humiliation and loss. Humiliation implies a significant loss of face or self-esteem, while entrapment signifies being stuck in a punishing situation (Craig, 2010). Depression, therefore, appears to be triggered typically by events which involve a loss of esteem and social entrapment, which involves a loss of prestige. But what of the apparent triggering of depression by the loss of an important other? Separation leads typically to a state of sadness marked by full distress emotions rather than the void emotions of depression. Adjustment reactions can usually be separated from clinical depression, but not always. DSM-5 (American-Psychiatric-Association, 2013) notes that losses may “include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in Criterion A (of Major Depressive Disorder) which may resemble a depressive episode…the presence of a major depressive episode in addition to the normal response to a significant loss
should also be carefully considered.” (p. 161). It should be noted that some states of separation distress become prolonged and morph into depression, perhaps in a manner akin to the protest gateway of Panksepp (2010). Yet, if sadness is not to be conflated with depression, why should the two conditions sometimes merge over time – why should depression sometimes replace grief? To follow the argument of this thesis, loss may first be manifest by sadness and the individual may become socially disadvantaged. No longer does the bereaved receive the flurry of phone calls or have people dropping in. She withdraws to “lick her wounds” and over time society takes less note of her. She has fallen into a state of low social support or prestige. In this state of abandonment, her self-esteem falls and her mood changes to one of sorrow and unchecked this leads to depression. Thus, in the first instance, loss of an attachment figure was the driver of her mental state, while in the second case her enemy has become prestige-loss. Put another way, it could plausibly be stated that prestige-loss underlies the origin of clinical depression.

**Play**

“The creation of something new is not accomplished by the intellect but by the play instinct acting from inner necessity. The creative mind plays with the objects it loves.” (Jung, 1875-1961)

The purpose of the proto-emotion PLAY (Panksepp, 1998) has remained elusive. It has been suggested that play promotes skill attainment, which in turn may lead to natural selection. Human play entails the positive emotions, which frequent Chance’s hedonic mode, maintained by affective signals and creative objects. Play is about generative creation; about *externalising the interior*. Dutton (2009) and Miller (2001) have linked this to sexual selection. PLAY is an approach emotional system, homologous with the rough-and-tumble system of Panksepp (1998) while other related forms of play include object, fantasy, exploratory and relational play, amongst others. According to Panksepp (1998), the parafascicular and posterior thalamic nuclei mediate play urges, with recruitment of movement sites such as the basal ganglia.

Through phylogenetic linkage with higher centres, it has been argued that PLAY, in its widest sense, is the currency of social engagement. The neocortex is not
essential for play and that frontal lesions may in fact increase play (Panksepp, 2004). Whilst knowledge of the neurochemistry of play (Table 18) is nascent, the synergism of opioids, dopamine and the endocannabinoids is operant (Trezza & Vanderschuren, 2009) and muscarinic acetylcholine and glutamate further play a role. The principal proponent molecules for PLAY, however, remain to be discovered. There are a number of opponent neurochemicals which antagonize play, including serotonin and GABA (Panksepp, 2004). There is a complex dopaminergic contribution, with both dopamine antagonists and dopamine agonists being opponent to play (Trezza & Vanderschuren, 2009). PLAY is important for social engagement, the development of cooperation and for the establishment of dominance hierarchies. Panksepp and Harro (2004) argue that play has a primary endogenous mammalian function. PLAY is inhibited by hunger, anger, fear, separation distress and is synergistic with CARE. Panksepp and Harro (2004) further note that in primates, with established social attachment, that social isolation has a devastating effect on the urge to play.

It is posited that the primary factor leading the group to invest in the individual is play, conceived in its widest sense. Play essentially involves the creation of a symbol (intervening object), between the subject and the object of the play. In humans, play is ubiquitous in contexts of social communication and PLAY is evident with the expression of the emotions of enjoyment, amusement and excitement. Amongst freely interacting persons, these emotions are expressed and mirrored in a dance of affect where touch may become evident. Gestures correspond to affect, the head bobbing and the torso moving in synchrony. Information, both narrative and affective, is exchanged in an often non-linear manner, while dramatic pose and expression are often evident. PLAY is the bed of creativity, song and humour, with its often goalless commonality. PLAY is the individual drive to “be” and the cement of culture, leading to creativity, social engagement, prestige and selection. Those in play are the instantiated ingroup, while those unselected remain on the margins of social interaction. Intrasexual prestige competition occurs within the ingroup, where attachment, status and physical beauty signals are also relevant. Play signals, however, remain the prominent method of intersexual selection.
As Panksepp and Biven (2012) have argued for an essential motive connecting cultural creativity and play, so Zepp-LaRouche (1994) advances Schiller’s argument for an instinctive basis to play. Herbert Spencer (1911) one century later connected play to art, while Nachmanovitch (1990) contends that “Improvisation, composition, writing, painting, theatre, invention and all creative acts are forms of play, the starting place of creativity in the human growth cycle, and one of the great primal life functions.” Meares (2005) emphasises the centrality of play, noting its “generative” linguistic capacity. Meares and Coombes (1994) further argue that culture is unique to human societies, arising from a consequence of a genetically given propensity – the play drive. Jung (1971) had earlier opined that play was the source of symbols, where language, myth, ritual, music, games and magic, the objects of culture, represent such symbolizing (Meares & Coombes, 1994). Play is thus a source of symbolisation, and as such, is constitutive of self, culture and art.

PLAY conceptually accords with the dimensions of extroversion and openness, as described in the Big Five personality typology (Goldberg, 1993). Extroverts are interpersonally activated, enthusiastic and favour energised group contexts. People with the openness trait have active imaginations and intellectual curiosity (Piedmont, 1998). PLAY is developmentally linked to the ATTACHMENT system, as it arises under the guidance of the parent, though with time PLAY extends to progressively wider social contexts. PLAY facilitates the establishment of rank, establishing younglings into the structures of society (Panksepp, 2004). There is a multiplicity of play forms in humans and Panksepp asks whether these are subsumed by multiple circuits, or one unified system. The author sides with Panksepp (2004), in subscribing to a single “command circuit” model – PLAY (Panksepp, 2004).

Hawes (Hawes, 1996) has vividly evoked the varieties of play in avians and mammals, where it is proposed that it prepares the young animal for the necessities of adult life. In humans, socially-oriented play may be categorised into the following forms: rough and tumble, socio-dramatic, social, creative, communication, dramatic, deep, fantasy, imaginative, role play, recapitulative (narrative), locomotor, constructive, symbolic (Hughes, 1996), flirtatious and games-with-rules play (Anonymous, 2009). Play is essentially a social enterprise. Juvenile play is also about
necessary skill development; “…a lion cub is wrestling with a young peer, pouncing and pawing, at one time going for the kill, and at another playing the victim” (Hawes, 1996). Social play facilitates cooperation, helps develop alliances and encourages innovation in social behavioural patterns (including dominance and aggression) (Hawes, 1996).

“Animals, it turns out, communicate playful intentions with certain stereotyped signals. The most widespread play signal is the play face, a relaxed, open-mouth expression seen in many mammal species, used virtually from birth. The human smile almost certainly evolved from this ancestral trait. For great apes, the extension of one's arms, hand contact, and an exaggerated gait similarly relate that ‘what follows is play’.” (Hawes, 1996)

Males, generally, engage more often in rough-and-tumble play, while juvenile vervet monkey (and human) females engage more often in “play mothering” (Hawes, 1996). Evolutionarily, play corresponds to the phylogensis of intelligence and the length of developmental latency (Hawes, 1996).

In The Art Instinct Dutton also argues that creativity is a process guided by an innate drive. This is linked to Darwinian sexual selection, whereby individuals are selected by their mates for favourable features, which are then handed on to offspring (Dutton, 2009). Partners select physical features indicating fertility, status and markers of likely attachment. Overwhelmingly, however, the features selected are features of social engagement, including: intelligence, language, humour (Buss, 1992), decorative speech (Darwin, 1871), emotion induction, affective mirroring, humour (Buss, 1992), creativity, music, song (Miller, 2001), appearance enhancement (“body-as-art”) (Buss, 1992), dance, courtship, ostentation and excitement, along with many others. The centrality of HESE to sexual selection is thus supported by research.

Dutton draws an example with the male New Guinea bower bird, which makes an ornate bower to attract his mate:
“His woven bower, which may be six feet or taller, is extravagantly ornamented, both inside and outside. On the floor and on the interior walls, the bird arranges clumps of berries, red leaves, displays of flowers, acorns, bright feathers from other birds, iridescent beetle elytra, and if available, brilliantly coloured human detritus...The desire to impress – and bed – a member of the opposite sex with displays of artistic creativity or the ownership of rare objects tastefully arranged is not unknown in our species.” (Dutton, 2009, p. 8)

Artistic objects for sexual selection are seen in birdsong and within the panoply of PLAY emotions, gestures and behaviours. Creativity, flirtation, novelty, social pleasure, an expressive face, unpredictability and variety are further cited as indicators of attraction (Miller, 2001). Vocal prosody is a likely factor. In The Art Instinct, Denis Dutton states that courtship, dancing, an exciting personality, dramatic discourse, and art ornaments (constituting an “extended phenotype”) contribute to selection (Dutton, 2009). Gaudy ostentation furthers the power of play attractants, as does the effort, cost and essential non-utilitarian nature of the ornament (Dutton, 2009). Zahavi (1975) has advanced the handicap principle, which argues for the selection advantage of prodigious waste in selection ornaments. High cost of the ornament to the individual indicates high fitness, as low-fitness pretenders could not afford to display such a costly ornament. Consider the peacock’s tail. It will be argued that the handicap principle pertains to the aetiogenesis of hypomania.

Certainly, social engagement is not only about sexual selection, but also about broader issues of prestige – social selection. Prestige is described as the standing or estimation one has in the eyes of people and signifies influence (The-Editors-Webster's, 1972). Modern models of prestige emphasise deference, proximity, collegiality and approval based on an acknowledgment of merit (Henrich & Gil-White, 2001). Psychodynamically, prestige also involves the introjection of collectively acquired esteem. As such, self-esteem is formed from a distributed network of the prestige attributions from important others. This is analogous to the fictional Lord Voldemort, who distributed his soul into seven horcruxes to attain immortality (Rowling, 2005). In developing self-esteem, humans ascribe their souls (self-esteem) to the ingroup as a means of engagement, advancement and protection. This is a strategy with obvious advantages, but with the disadvantage that one
becomes beholden to the fortunes and impressions of others. Social Attention Holding Power (Gilbert, 2006) occurs when group members evaluate themselves according to their power to attract interest. It is argued that the human infrastructure of social interaction arises within play-driven social engagement, leading to prestige and the realisation of the self.

“The esteem needs. -- All people in our society (with a few pathological exceptions) have a need or desire for… self-esteem… for confidence in the face of the world… Secondly, we have what we may call the desire for reputation or prestige…” (Maslow, 1943, p. 10)

Hypomania, Mania and “Mixed” States

“I roll on like a ball, with this exception, that contrary to the usual laws of motion I have no friction to contend with in my mind… I am almost sick and giddy with the quantity of things in my head – trains of thought beginning and branching to infinity, crossing each other, and all tempting and wanting to be worked out.” (Ruskin, as cited in Goodwin & Jamison, 2007, p. 37)

Whilst in depression play is retarded, in hypomania it is universally released. Hypomania is a singular condition; one patient described it such: “if I’m ill, this is the most wonderful illness I ever had.” (Campbell, as cited in Goodwin & Jamison, 2007, p. 32). Persons with hypomania may be oblivious to risks as their self-esteem soars. They tend to take a central “performing” position in social settings, being more voluble in their expressiveness. Playfulness is the rule and affect is elated, while goals are pursued with alacrity and appetitive and energetic functions are increased. Motor function is raised and persons may describe feeling “wired or energised”. Such is hypomania, with its frenetic pace, enlivenment and engagement with the world. Hypomania represents an unequivocal change in affect and function and should be observable by others. The episode does not cause marked impairment in social or occupational functioning (American-Psychiatric-Association, 2013).

Figure 6 depicts the prestige model of hypomanic escalation. Goal success may directly trigger this cascade (Alloy, 2013), or conversely negative social
interactions and life events may precipitate self-esteem change in cognitively and affectively predisposed persons. In turn, \( HESE \) is upregulated as individuals move toward social objects, in the process displaying the *ornaments* of play. In contrast to the *dormant* position of the depressive, the hypomanic person is a “performer” in the group and social investment is generally evident. I have argued that, according to the handicap principle of Zahavi (1975), hypomania reflects profligate waste, which indicates to prospective partners that the hypomanic individual is fit enough to carry this handicap. In this sense I have focused on the adaptive aspects of the condition—those likely to increase sexual selection. The elevation of mood in the simplex S5 group suggests that the possibility that subclinical mood elevation may be a possible consequence of the possession of bipolar genes. It is notable that all of the four individuals with ASRM-defined categorical mood elevation in the simplex (S5) group had either sub or supra-threshold MDQ scores—both variables suggestive of a degree of spectrum bipolarity. Similarly, it has been noted that the elevation of ASRM in the subthreshold (S4) group may be an artifact of the transfer of nine participants with categorically elevated mood from the control S7 node. Of relevance, however, these individuals with categorically elevated mood had MDQ scores statistically higher than their euthymic compatriots, confirmatory of subclinical spectrum bipolarity in those transferred persons.

Given the above description, hypomania should be associated with increased prestige. Figure 20 and Table 48 (Appendix) demonstrate that this is the case and the nlogSIPS score for the (hypo)manic cohort was significantly higher than the euthymic group with a Cohen’s \( d \) of 0.74 (moderate to large effect). This of course may reflect a response bias, as hypomanic individuals may exaggerate their number of greetings and available friends and family. In order to minimize this risk, the SIPS scale utilised numerical rather than subjective values. Therefore, hypomanic persons perceive increased social prestige, be it primary, imaginal, or secondary to the condition—or a combination of these. In an ancestral tribal context, such persons may be viewed as imbued with spirits or even be seen as shamans. Certainly mildly and moderately affected individuals are likely to be productive in tribal undertakings and may have prestige leadership roles. Unfortunately we are bedeviled by a modern fallacy, through which hypomanic individuals are perceived as *ill* rather than *blessed*. This has
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arisen through better communication and education on medicine and psychology. In evolutionary times hypomania would not have been formulated as illness, but as a special state of self and behaviour which may demand respect or even reverence. Therefore, mild hypomania is likely to have socially advantaged the individual and led to greater fitness.

"From this perspective, mania perpetuates the once-fragile sense of connectedness, the one feeling that was so excruciatingly discontinuous for the child. The time of feeling deeply linked to the mother or caregiver becomes the experience of the whole world: now everything is linked together, love is everywhere, good vibes and possibilities for the future." (Leader, 2013, p. 29)

Did the hypomaniac predisposition arise through a process of adaptation? Sociometer mutations may have arisen randomly (and in stages) and been governed by frequency dependent selection, responding to social triggers with goal success, high affect and social prowess. Such individuals could then compete in a landscape where prestige is the currency. The faulty sociometer in this instance responds with a feedback response to raise self-esteem and prestige; the apogee being hypomania. Negative social contexts are thereby dealt with and reversed. In Figure 38 we see the hypomaniac individual in the socially marginal zone, though being centripetally focused. As prestige approach motivation (MSPaM) rises in hypomania, so too does prestige (Figure 28) with a Pearson correlation of 0.51. Therefore, through this curious condition, in ancestral contexts group inclusion may be restored.

DSM-5 (American-Psychiatric-Association, 2013) differentiates hypomania from mania. While the B core features are identical, the syndromes differ only in terms of minimal duration and social and occupational impairment – a manic episode requiring marked impairment or hospitalisation – or psychotic features. With respect to the prestige model, mania involves the action of the tense arousal system acting superordinately to the HESE arousal system. This results in the ranking behaviours of aggression, competition and dominance (Brune, 2008) in addition to increased socialisation, grandiose and goal-driven cognition, elevated affect and behavioural features of HESE-driven hypomania. In this sense, mania is not just more hypomania, but an emergent syndrome with additional diagnostic features. According to this
model, mild to moderate hypomania is adaptive while – in current social and historical contexts – mania is not. I would further suggest that apparently maladaptive hypomania is in fact “sub-mania” either in disguise, in treatment, or in prodrome. Of course in ancestral contexts even mania may have had a benefit with respect to leadership and resource allocation. Manics are often terrifying and may have been high ranking, especially where mania may seem legitimate or spiritual and not the product of an illness, which had in ancestral time not yet been conceived.

It may seem strange that there are two (hypo)manic individuals in the unipolar (S6) group. This is evidently fallacious and may represent a failure of the analytic method to fully and accurately demarcate groups. Of course the finding may also represent two false positive Altman Self Rating Mania (ASRM) results, though this seems unlikely with ASRMs of 12 and 15. One of the unipolar respondents had a PHQ-9 score of 8 and a subthreshold MDQ of 5, indicating mild depression, which may have in fact been a part of a subthreshold and misdiagnosed “mixed” state, while the other had a PHQ-9 score of 0, indicating a “pure” (hypo)mania. Such a situation is relatively common given the findings of Angst, Azorin, et al. (2011), where apparently unipolar patients are in fact bipolar spectrum cases. Another individual was deemed to have a “mixed” mood state by merit of a concomitant PHQ-9 score of 16 and an ASRM of 8 (and a subthreshold MDQ of 4). This would be consistent with DSM-5, which allows for mixed features in major depression (American-Psychiatric-Association, 2013) and these may in fact represent an incipient bipolar spectrum condition. Minor anomalies in mood state were therefore detected in unipolar respondents and one of these appears to have been inappropriately allocated to the unipolar (S6) cohort, a situation which is common in the real world of affective diagnosis.

“Mixed” states were defined by means of a concomitantly suprathreshold ASRM and PHQ-9. Of course this is a contrivance, as the presence or absence of mixed features should be made on the basis of clinical interview. In mania and hypomania mixed features can be diagnosed on the basis of at least three specifications on the majority of days. These include the presence of dysphoria or depression, anhedonia, psychomotor retardation, fatigue or anergia, worthlessness or
guilt and suicidal ideation or action (American-Psychiatric-Association, 2013). It was necessary to designate a “mixed” group to maintain the fidelity of the depressive and (hypo)manic groups. In so doing, something of relative value may have been incepted, though it may not approximate actual clinical mixed status. What might the greater than expected co-occurrence of significant depression and (hypo)mania signify? Perhaps it is in fact a proxy for a real mixed state, as the PHQ-9 is based on the DSM-IV quite closely and picks up the items described above? Or perhaps it represents severity of affective disturbance suggestive of mania rather than hypomania? DSM-5 (American-Psychiatric-Association, 2013) stipulates that if the full criteria for mania and depression are simultaneously met, then mania with mixed features should be diagnosed. In support of this, six of nine of the “mixed” group were situated in S1, while two were from S2 and the final one from S6 (the case mentioned above with the MDQ of 4); this speaks to the logic of the diagnostic algorithm.

Several results are worth noting: The ”mixed” group ($n = 9$) had a prestige (nlogSIPS) score significantly lower than euthymic and (hypo)manic individuals and not significantly different to depressed individuals (Figure 20, Appendix Table 47, Table 48). This is despite the presence of mood elevation (alongside the high depression score). In furtherance of the discussion of theory, tension is far higher in “mixed” states than in pure (hypo)mania (Figure 29) with a difference of 2.75, a $p$ of .001 and a $d$ of 1.83. The total score for MSPaM in the “mixed” mood category was 14.75 points higher than the (hypo)manic category (Appendix Table 49, Table 50, Figure 22). Though this difference failed the NHST criterion for difference ($p = .330$) the $n$ for the “mixed” group was only 9 and the $d$ was 0.74. These results together suggest that the “mixed” group may be categorically distinct from the (hypo)manic cohort by merit of low prestige, high tension and high depression, while MSPaM may also be higher – certainly the MSPaM/prestige ratio was higher (Table 37, Table 38). What might this mean? Perhaps the “mixed” group is in fact a manic group, while the (hypo)manic group is in fact hypomanic. The former would seem possible, given that there are only nine “mixed” cases, eight of which are situated in the bipolar nodes – approximating the expected incidence of mania in this cohort; it seems unlikely that there would be more mixed than manic cases. Similarly the majority of the (hypo)manic group was situated in non-clinical nodes S4 and S5, with only four in S1,
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making it extremely likely that the majority were hypomanic rather than manic. Goodwin and Jamison (2007) note that “most (manic) patients, on average, are depressed (46 percent) or labile (49 percent) nearly as often as they are euphoric (63 percent) or expansive (60 percent); they are irritable (71 percent) even more often.” (p. 40). This supports the contention that the “mixed” group here, with categorical mood elevation in the context of high tension, high depression and low prestige are in fact manic with mixed features. The (hypo)manic cohort, on the other hand has elevated prestige and a low mean TENSION, echoing the contention of Parker and Eyers (2008), already cited, that anxiety (tension) melts like snow on a true hypomanic’s summer day.

The discussion above argues that the conditions of hypomania and mania are different, but overlapping. In DSM-5 (American-Psychiatric-Association, 2013) the only valid categorical differentiator between the two is psychosis and this is not mandated for the diagnosis of mania. Otherwise the only differentiation between the syndromes in DSM-5 is dimensional – impairment. Clearly, mania and hypomania are related; they occur in the same bipolar conditions, they may temporally overlap and, most importantly, they share key criterion B features. How then can we have overlapping conditions that earn different disorder appellations? The most parsimonious explanation is to adopt a superposition model that sees the conditions arising through the interacting dysfunction of two arousal systems. In the simplest condition – basal hypomania – the hedonic energetic social engagement (HESE) system is aberrant and pure approach hypomania (status excitement) ensues. At a certain threshold – for biological, psychological or social reasons – the second stratum may be activated, triggering mania, and this involves significant tense arousal, alongside opposed approach-withdrawal. In mania both strata are concurrently dysfunctional – one sees evidence of both increased hedonic social engagement/withdrawal and dominance at proximal times. The relationship and causation of affective syndromes is clearly complex. Hypomania (and I will categorically call it that now) is a state of high prestige, low tension, benign prestige approach motivation and elevated affect. Mania (dropping the “mixed” appellation) is similar to major depression, with the exception of high mood elevation. While hypomania is a disorder of social engagement, mania is a disorder of dominance and
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dysphoria, where prestige approach motivation is in overdrive, relative to prestige level (Figure 22).

Having proposed that these variables assist in defining the mood syndromes of mania and hypomania, we are left asking “why?” Are these accidents which occurred in the arousal systems in deep evolutionary time? Or do they serve an adaptive purpose? I have previously argued that hypomania is a mechanism for promoting social investment – an extra “oomph” – where there is goal success. Or, else prestige uncertainty – as demonstrated in the MSPaM rise to increments in depression – may trigger a homeostatic increase in prestige approach motivation. Mania is another matter and involves both approach emotions and behaviours, alongside withdrawal affect. In fact Phelps (2008) conjectures that mania and depression may be situated on orthogonal axes which may give rise also to mixed states. Mania is a global, physical, disturbance of the affective brain, where the individual tries to completely dominate their social environment through charm or aggression.

What of the evolutionary origins of mania? In a seminal paper Territory, Rank and Mental Health: The History of an Idea Price et al. (2007) link elevated mood with increased rank, allowing the assumption of a leadership role. This idea followed from a number of developments linked to the assignation of social roles to territory holding, which in turn led to social asymmetry through the aegis of rank. Affective disorders were linked to rank signalling and intense mood states were needed for “the creation and reversal of asymmetry” (p. 533). Invoking the triune brain hypothesis of MacLean (1982), Price et al. (2007) nominate escalating and deescalating strategies as the basis of agonistic (rank) competition. They cite “fighting on”, “being assertive”, “angry or hostile”, and (having) “elevated mood” (Table 1, p. 538) as part of the escalating strategy, whilst not explicitly linking this to mania. They then introduce the notion of prestige competition, which overtook agonistic competition and “the pursuit of goals replaces the decision to attack” (p. 540) and these goals are typically prestige related. Self-assertion replaces anger, while at the emotional level mood elevation takes place. We are reminded that mood change is an all or none phenomenon. Reminiscent of Nesse and Williams (1994), the authors cite depression as being due to continued escalation at the rational level and ongoing de-escalation at the reptilian level.
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While linking this model with depressive illness (as a deescalating strategy), the authors are obscure about their model’s role in hypomania and mania. This may be because they link excessive mood change with disorganisation and failure to succeed in agonistic contests – “extreme levels are clearly maladaptive, taking the form of the disorganisation of clinical mania and the incapacity of psychotic depression” (p. 546). As such they have failed to explicitly separate more adaptive hypomania from disorganised and destructive mania. The authors go on to describe a number of clinical scenarios which appear to be linked to maladaptive mixed escalating and deescalating strategy sets at different triune levels. It is unclear whether they ascribe pathology to either agonistic or prestige competition; it is possibly both forms of competition depend on context. It seems apposite to speculate that mania is a form of tonic and extreme agonistic escalation, while hypomania arises from a disturbance in prestige competition. As such, mania is evolutionarily an older pathology, only later becoming contrasted to prestige mechanisms. Adapting the model of Price et al. (2007), hypomania may be a pure form of escalation in prestige competition, while mania may involve mixed opposing agonistic strategies at different levels of the triune brain.

In investigating a behaviour Tinbergen urges us to consider:

1. What adaptive function does the disorder perform in aiding survival and reproduction?

2. What are the proximate biochemical, neural and environmental factors involved?

3. “Why did the behaviour develop in the individual patient in the course of ontogeny?”

4. When did such behaviour evolve at a phylogenetic level? (Stevens & Price, 2000)

I have argued that depression may provide a prestige withdrawal function to mitigate ostracism and thereby promote survival and reproduction. I have also argued that mild to moderate hypomania (excluding “sub-mania”) confers a fitness advantage through the augmentation of prestige and subsequent sexual selection, though it is
unique to Homo sapiens and therefore has no direct animal homologies. The major ontogenetic factor in bipolar disorder was found to be perceived childhood relational trauma (PCRT) and the disorder may have arisen genetically and also as a means of maintaining relational space. At a phylogenetic level, bipolar states may have two convergent origins; in both the dominance struggles of reptiles, birds and primates and also in prestige competition, which is said by Stevens and Price (2000) to have arisen in the ancestor common to both chimpanzees and Homo sapiens. Mania may have its origins in older reptilian behavioural repertoires, to which HESE aberration has more recently contributed. Proximate mechanisms will be considered in the discussion of Table 18.

**Complex Systems**

“That is, these systems change and reorganize their component parts to adapt themselves to the problems posed by their surroundings. This is the main reason the systems are difficult to understand and control – they constitute a ‘moving target’. We are learning, however, that the mechanisms that mediate these systems are much more alike than surface observations would suggest. These mechanisms and the deeper similarities are important enough that the systems are now grouped under a common name, *complex adaptive systems.*” (Holland, 1992, p. 18)

The brain is a complex non-linear system which operates below the threshold for chaos. It is adaptive to its environment and changes its component parts in response to problems occurring in its surroundings – evolving, aggregating, and anticipating (Holland, 1992). Within adaptive systems complex global patterns with new properties may arise from local interactions—this is termed *emergence* (Lansing, 2003). Systems may become chaotic, frozen (periodic) or complex – in the latter islands of stability emerge, shifting shape at their borders (Lansing, 2003). Networks which are frozen or chaotic do not transmit information, thereby being unable to adapt; complex networks that are at the “edge of chaos” are able to do both (Langton CG as cited in Lansing, 2003). Complex systems may tend to evolve toward particular sets of physical properties and these are called *attractors*. Where connections are small, periodic behaviour may ensue, in which tiny attractors arise quickly, trapping the system in simple state cycles by which information may be stored but not communicated between islands (Lansing, 2003). “So networks with lots of
connections exhibit chaotic behavior, whereas networks with very sparse connections decompose into an archipelago of isolated subsystems that either stop twinkling or follow simple repetitive patterns.” (Lansing, 2003, p. 189).

In 1995 Gottschalk, Bauer, and Whybrow (1995) undertook a dynamic analysis of mood in individuals with rapid cycling bipolar disorder. They cited the biological rhythms model, which posited that mood swings were governed by 48 hour multiples and that endogenous rhythms may drive the disorder. Patients and controls maintained diaries with a visual analogue scale over one to two and a half years. The bipolar patients demonstrated less noisy mood fluctuations compared to the controls, whose mood responded more to their social environments. The result indicated lower complexity, but higher structure, for the mood expression of bipolar persons (Heath, 2004). They concluded that, while the condition is not cyclic as the biological rhythms model would suggest, it is highly organised and characterised by the existence of a low-dimensional chaotic attractor. In a similar vein, Woyshville, Lackamp, Eisengart, and Gilliland (1999) found evidence for non-linear dynamics in which “chaotic” clinical patterns resulted from a less complex chaos-theoretical perspective than normals. Their 36 clinical subjects were included based on the phenomenology of “affective instability”, regardless of diagnosis. Heiby, Pargano, Blaine, Nelson, and Heath (2003) investigated mood dynamics in two young women, one of whom had depression while the other did not. Mood was rated over six months using a Likert scale, which was calibrated against the Beck Depression Inventory. The control subject showed greater mood variability. The authors suggested the “maladaptive determinism hypothesis”, whereby the ill state has more deterministic and predictable behaviour than occurs in healthy individuals. With increasing determinism, individuals adjust less well to environmental complexity (Heath, 2004). Bonsall, Wallace-Hadrill, Geddes, Goodwin, and Holmes (2012) described a time-series approach to characterise mood stability in bipolar disorder. Their results indicated that mood variability was nonlinear and deterministic, when comparing a stable with an unstable cohort of bipolar patients over six months. These results together – and this is informed conjecture – suggest that mood states in bipolar individuals may generally arise from low complexity and deterministic attractors in a “frozen” state, while the
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attractor for normal mood may arise as part of a region of complexity “at the edge of chaos”.

I have previously suggested that prestige has a landscape – indeed this was the starting point and the title of this thesis – onto which affective states may be superposed. According to the prestige model, the terrain forms a topography of three dimensions; prestige (x), prestige approach motivation (y) and mood state (z). If hypomanic or subthreshold mood elevation is the positive elevation for this landscape, then depression is logically the negative or downward deviation. The level stratum is euthymia. This results in a landscape, formed by the dimensions of prestige and prestige approach motivation and punctuated by hills and troughs of affect. This is construed as a dynamic form, in which individuals move laterally and vertically according to their states of prestige and affect. Such would tend to be attracted to areas of high prestige and affect, so as to facilitate social inclusion and survival in the ancestral timescape. Conversely they may become “stuck” in a region of low prestige/affect, which is known as mild or moderate depression. Given the previous discussion, these regions may be described as low dimensional or “frozen” basins of attraction. Such locations do not readily transmit or receive information.

The MOPF data were plotted by means of contour plots of depression and mood elevation according to the topography described (Figure 35, Figure 36). Figure 35 shows the result for depression (PHQ-9) score overlaid upon the interaction of prestige approach motivation and prestige. It reveals three rough bands of affect – euthymia (PHQ-9 <5) in the high prestige-low MSPaM quadrant. This accords with the everyday conception of normal mood, where individuals feel included (prestiged) and need not be motivated to achieve prestige, as they already hold it. There is a band running across the graph indicating mild depression (PHQ-9 5-10), where individuals have subthreshold low mood and vary diagonally in the degree of prestige and prestige approach motivation. Finally, there is a region of high depression (PHQ-9 >10) located in the low prestige-high prestige approach motivation quadrant. These individuals are clinically depressed and arise largely from bipolar I (S1), bipolar II (S2) and pseudounipolar (S3) cohorts, with eight unipolar (S6) individuals represented. There is a small island of high depression in the low prestige-low
MSPaM quadrant and this includes unipolar (S6) individuals. There was no pattern for the location of unipolar (S6) individuals in this landscape – such persons were spread across all four quadrants in a non-focal manner.

This relationship was subject to multiple regression, but because regression into PHQ-9 yielded an inconstant “fanned” variance in residuals, the natural logarithm of PHQ-9 was used. This found a strong relationship between MSPaM, prestige (nlogSIPS) and log depression score (nlogPHQ-9) – explaining just over twenty percent in the variance in nlogPHQ-9. The contour plot of this relationship showed a marked quadrantic relationship, such that logarithmic euthymia was clearly located in the high prestige and low MSPaM quadrant, while logarithmic high depression dominated the low prestige-high MSPaM quadrant. In the topographical sense, this result is consistent with the notion that bipolar depression forms a basin of attraction in the prestige-prestige approach motivation landscape in the left upper quadrant. As has been indicated, the high depression response reflects an underlying overrepresentation of bipolar I (S1) respondents (45.46% of the quadrant and 23.25% of the spectrum) and an underrepresentation of control (S7) cases (6.78% of the quadrant and 21.05% of the spectrum) (Appendix Figure 43).

The unipolar group is a sample of the population with a more heterogenous condition and was split relatively evenly between mental health and general practice collectors. I am inclined to interpret this as follows – the bipolar and attenuated bipolar depressions reflect a more overt biological dimension, while the community unipolar (S6) cohort includes those with adjustment disorders, clinical depressions and personality-related depressions. As such, there is an evident pattern in the bipolar end of the spectrum, but an absent pattern for the heterogenous unipolar cases. This is reflected in the lack of correlation between unipolar (S6) MSPaM and depression scores ($r_p = 0.01$). It follows from this discussion, that bipolar (and attenuated bipolar) cases that find themselves depressed and low in prestige are motivated to increase prestige though MSPaM leadership behaviours, while their unipolar cousins are not. What now of mood elevation?
Figure 36 depicts the map of prestige approach motivation (y) against prestige (x) indicated by mood elevation (z). The most obvious feature of this topography is that moderate to high mood elevation is largely focused in the high prestige-high prestige approach motivation quadrant. These individuals were overrepresented by bipolar II (S2) and pseudounipolar (S3) nodes, with elevated mood (and also “mixed”-manic cases). Unipolar (S6) cases were underrepresented (Appendix Table 51). Low ASRM euthymic cases were largely located in the high prestige-low MSPaM (lower right) quadrant. When hypomanic cases were plotted on this map, they lay along a line increasing positively toward the right upper corner (high prestige-high MSPaM), with a correlation of 0.51. Seemingly, as prestige approach motivation increased in this cohort with categorical mood elevation, prestige increased also. The regression of the contour relationship needed to again use the natural log of mood elevation (ASRM) due to inconstant variance in residuals. It found a significant relationship between the three variables, which explained a modest 6.73% of the variance in nlogASRM. What do these results mean? As with the depression results, these data are consistent with a three dimensional map wherein mood elevation forms a peak in the landscape, located in the right upper quadrant of high prestige and high prestige approach motivation and to a lesser extent the very high end of the left upper quadrant (Motivated Withdrawal Figure 39). Mood elevation intends individuals to this location, apparently driven by increasing MSPaM and resulting in elevated social investment (prestige). As with the depression basin, Motivated Approach (Figure 39) may be an attractor for bipolar spectrum individuals. This peak conceptually correlates with high HESE activity and may be frozen at its nidus (manic and transmitting no information), or complex at its margins (hypomania) and therefore communicating with its environment.
Figure 39. Graphical representation of the principal contour plot regions of approach and withdrawal prestige approach motivation according to an ideal free model of population distribution. Reference lines are drawn at the median for each axis. Textual description to follow.

Figure 39 depicts a topographic representation for the data thus far described. It shows the four regions of affective intensification overlaid on the landscape of prestige. The relative location of the mood states suggests a fundamental relationship with the intersection of prestige approach motivation and prestige. I have made a supposition that true unipolar (melancholic/endogenous) depression is located in the left lower quadrant. While the highly depressed unipolar (S6) subjects tended to have low prestige, there was no localization to either the high or low MSPaM quadrants. There was no pattern evident when the general practice and mental health unipolar (S6) groups were divided and, as described, depression level did not correlate with MSPaM in the unipolar (S6) group. Therefore this particular supposition may be
nothing more than uneducated guesswork. What might this general localisation for euthymia, bipolar depression and mood elevation mean? It suggests that there is an inherent relationship between the x and y axes and that as these parameters vary in relation to each other that mood syndromes flux – or else reverse causality may apply. It is perhaps more self-evident that prestige will change in relation to mood status, but not so obvious that MSPaM will. The results are supported by the earlier ANCOVA (Table 8, Table 9) which shows that high MSPaM occurs in the bipolar (S1, S2) and attenuated bipolar (S3) nodes, when compared to controls. The ANCOVA (Table 8, Table 9) found a residual structural effect for spectrum on MSPaM (evident in S1, S2 and S3), but found that depression (PHQ-9) and mood elevation (ASRM) predicted MSPaM also. I have further shown correlations between mood states and parameters such as prestige, self-esteem, TENSION and MSPaM in the dynamic model analysis. These results together suggest that there is a trait difference in MSPaM between spectrum categories, alongside a dynamic flux with mood. Figure 39 therefore captures these findings topographically and clearly links mood to both prestige and prestige approach motivation.

**Evolutionarily Stable Strategies**

In *An Introduction to Behavioural Ecology* Davies, Krebs, and West (2012) explore the distribution/configuration of species where there is competition for limited resources. Often the best option for an individual depends on the actions of its competitors and payoffs are therefore frequency dependent. The Evolutionarily Stable Strategy (ESS) occurs when all of the population members adopt it and it cannot be bettered by an alternative strategy. The authors cite the Hawk-Dove game, where in simulated contests Hawks always fight and may injure themselves and their opponents, while Doves never engage in fights. It may seem obvious that the Hawks would win and dominate the population, but that isn’t so. I won’t go into the mechanism, but the outcome of this simulation is that it leads to a frequency dependent selection – an equilibrium between Doves and Hawks, where each strategy does best where it is relatively rare. A similar situation has been proposed for antisocial personality disorder, in that it is likely to prosper where its frequency in the population is low. This ESS may arise in one of two ways: there are dimorphic states.
of pure strategies (Hawk or Dove), or individuals play an alternating strategy of Hawk and Dove, morphing from one to the other with a 50% probability. This morphing state of strategies is analogous to that seen between depression and mood elevation in the individual with bipolar disorder.

Davies et al. (2012) further describe a model in which there are two habitats, one rich in resources and the other not. Each individual is free to exploit its habitat of choice, but for the fact that someone may get to the rich habitat first. “Eventually a point will be reached where the next arrivals will do better by occupying the poorer quality habitat where, although the resource is in shorter supply, there will be less competition.” (p. 119). Profitability becomes equivalent in each habitat. This has been termed the ideal free distribution in behavioural ecology. In a broad sense, the resource studied may be access to a checkout counter, food or access to mating partners. I will argue that this resource availability model is applicable to prestige acquisition. The ideal model does not account for the fact that some competitors are unequal to others at resource acquisition and therefore “despots are part of the habitat to which the subordinates respond when deciding where to search.” (p. 123). It turns out, that despite unequal competition, the consequent net dispersal observed is hard to tell from an ideal free distribution. Therefore, individuals may tend to distribute themselves according to resource acquisition, sometimes avoiding plentiful supplies which are competed for, and despite unequal competition the form of the ideal free distribution is often observed.

The distribution of individuals by mood state, superimposed on the prestige by prestige approach motivation landscape, is depicted in Figure 42 (Appendix). This complements the single-affect dimensional contour plots already described. I will focus on just a few observations. Firstly, the euthymic group is clustered in, or close to, the right lower quadrant – I have called this the Secure Approach quadrant (Figure 39), notable for euthymic or elevated mood and high prestige, with minimal prestige approach motivation. This group is able to engage with social objects with relative equanimity. The left lower quadrant I have called Insecure Withdrawal; this is largely occupied by a sparser number of mildly and moderately depressed individuals. I believe that this quadrant represents a risky environment from which ostracism is
likely. The ideal free model posits that such an unappetising place will hold fewer competitors, but that some individuals will be forced by prestige resource dispersal to dwell there. The left upper quadrant is frequented largely by moderately depressed individuals, with three manic (mixed) persons. Motivated Withdrawal comprises those in a state of withdrawal and low prestige accompanied by high prestige approach motivation. These persons are sensitive to their state of low prestige and motivated to reverse it. I have termed the right upper quadrant Motivated Approach, comprising a mixed group of depressed, euthymic and (hypo)manic and manic (mixed) persons, in a state of high prestige and high prestige approach motivation. The trend of this quadrant is for elevated mood (ASRM). The pleomorphic nature of the quadrant is evidenced by the range of mood diagnoses – though with raised ASRM – in the context of high prestige and MSPaM, contrary to the relatively uniform mood states in the other quadrants.

The relative presentation of spectrum groups in the Motivated Approach quadrant was examined (Appendix Table 51). This found that bipolar II (S2) and the pseudounipolar (S3) groups were overrepresented relative to their total numbers in the bipolar spectrum. The unipolar (S6) node was underrepresented. Together, these results suggest that the S2 and S3 nodes are preferentially motivated for approach, while the S6 node is less likely – noting the cell’s clinical heterogeneity. The vast majority of bipolar I (S1) individuals are located in, or on the boundaries of, the Motivated Withdrawal quadrant. Together, these results suggest that the state of MSPaM “motivation” is predominantly a bipolar rather than a unipolar phenomenon, whether it occurs as part of a withdrawal or approach response.

The results are consistent with an ideal free model of prestige acquisition, allowing for the possibility that individuals may monopolise this commodity. Persons in the Secure Approach quadrant, largely controls (S7) and subthresholds (S4) (Appendix Figure 43), are minimally affected by prestige challenges, buoyed by their possession of good self-esteem and nourishing relationships. Individuals with perceived or actual low relational value may respond excessively to prestige challenge, dropping self-esteem and raising TENSION (see Figure 9). According to intrinsic and extrinsic factors, a response of approach or withdrawal will eventuate
and persons will become dispersed across the prestige landscape according to the relative possession of prestige and prestige approach motivation. Prestige acquisition is harder in the withdrawal quadrants; hence in the wider population these areas are relatively sparser in their occupancy. Individuals from particular bipolar spectra such as bipolar I (S1) and control (S7) may monopolise particular “pastures” – Motivated Withdrawal and Secure Approach respectively (Appendix Figure 43). Occupancy of each of the four quadrants tends to co-occur with the adoption of discrete mood states, corresponding to the basins and peaks described in complexity theory. Such attractors may become “frozen” and thereby impede the ongoing flow of information, as may be the case in major depression and mania. While depressive states may have a signal such as “let me be!” communication beyond this point is lacking, such conditions may become static and persist for extensive stretches of time. The Motivated Approach quadrant is constituted by individuals with various mood categories, albeit with higher relative mood elevation. This quadrant represents a common point for those subjected to prestige challenge and able to strategically increase their motivation to prestige. In all of these senses, we live in a landscape of prestige, which has profound effects on our capacity to survive and which locates individuals in states of loss and repair.

**Bipolar Disorder Causation-Correlation**

We now move tentatively to discuss possible causal relationships in relation to bipolarity, beginning with an examination of the links between bipolar family history, perceived childhood relational trauma (PCRT), MSPaM, and categorical bipolar disorder (of which there were 77 subjects). Each of the three predictors was found to have an independent predictive role into the diagnosis of bipolar disorder when several predictor binary logistic regression was undertaken (Appendix Table 52). The role of bipolar family history in the development of bipolar disorder in offspring is already well known and studies discussed earlier have highlighted the contribution of childhood relational trauma on the development of bipolar disorder. The role of MSPaM is novel and curious, especially if we inspect Table 16, where high MSPaM has an OR in predicting BD of 6.8 – but only in the absence of a bipolar family history. By way of reciprocation, the OR for a positive family history in predicting BD was 11.33 in the presence of low MSPaM, but far less in the presence of high
MSPaM (Table 17). This is curious indeed, seeming to indicate that MSPaM and bipolar family history predict bipolar disorder, but in an interacting way – and moderation was demonstrated. When both factors occur together, they seemingly cancel out any aetiologic effect. This opens up the possibility of some sort of *epistatic* effect; perhaps the family history genes suppress genes associated with MSPaM and vice versa. MSPaM may be a “rogue” contribution to bipolar disorder diagnosis where there is no familial influence. However, the OR of 1.98 indicates that bipolar family history may also predict high MSPaM.

It was possible to test this causal discourse in another way – by recourse to the Mood Disorder Questionnaire (MDQ). As has been already noted, the sum of MDQ items may be added together to get an approximation of dimensional bipolarity. Table 56 (Appendix) shows the relative independent contributions of FHx, PCRT, and MSPaM (binary) to MDQ score. All three factors had a significant contribution. Does the same interaction between bipolar family history and MSPaM hold out with MDQ? Yes, a significant interacting term was evident in Table 57 (Appendix). There was no significant interaction between PCRT and MSPaM (binary), as shown in Table 58 (Appendix).

As an aside, there was a significant association between bipolar family history and PCRT (Appendix Table 39). This suggests that in the subset of persons with a family history that resulted from *parents* with bipolar disorder (as opposed from other relations), parental bipolarity may have been significantly traumatic to the proband. Other explanations include the possibility that bipolar genetic factors increase the temperamentally-based experience of neglect or abuse, as the perception of childhood environment may be partially under genetic control (Hanscombe, Haworth, Davis, Jaffee, & Plomin, 2010). Cross sectional mood abnormality (i.e. depression) in persons with inherited bipolarity may also skew their perception of their developmental history.

In summary, we come therefore to Figure 37, which summarises the model, where bipolar family history is the established contributor to bipolar disorder diagnosis. It has a direct effect on diagnosis, while this relationship is also partially
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mediated by perceived childhood relational trauma (PCRT). PCRT is predicted by a familial history of bipolar disorder, presumably due to the relational disruption that a disorder in a parent may cause. This relational disruption partially carries the effect of family history on BD, while perceived childhood relational trauma, in and of itself, appears to increase the risk of bipolar disorder. The OR for PCRT was 4.31, against the OR of 2.93 for bipolar family history. While bipolar family history and MSPaM are related (OR 1.98), MSPaM acts generally as a “rogue” contributor to BD diagnosis and has its major impact in the absence of a bipolar family history. When the two are combined, they seem to cancel each other out through either an epistatic or other proximate mechanism. When high MSPaM is not present, bipolar family history has a large odds ratio of 11.33 with respect to BD diagnosis. When each of the three factors is compared for their independent contribution, all predict categorical and dimensional bipolarity (MDQ). The model is thus a robust schema for the contribution of childhood relational environment, bipolar family history, and MSPaM to bipolar causation. Nevertheless, it is only correlative and causation cannot be proven.

Figure 23 is added for the sake of reflection – line plots of mean prestige, self-esteem, and the contingencies of self-worth scale (CSW.av) are drawn by spectrum node. As the results section indicates, prestige aligns with self-esteem, except for the S3 (PsUni) pseudounipolar node, where we have noted that prestige is high, possibly by merit of bipolar genes. When we compare self-esteem with the contingencies of self-worth plots we see that the graphs almost perfectly mirror each other. States of low self-esteem in the nodes are married to highly contingent self-worth. I note that the bipolar 1 (S1) node has a contingent self-worth score appreciably higher than the controls. As with MSPaM this extreme value may be a marker of the group prone to mania, where self-worth is conditional on prestige and where prestige approach motivation becomes compensatory. Of necessity then, these individuals are prone to prestige challenges.
Empirical Considerations

Survey Method

The Measurement of Prestige Factors (MOPF) research (Appendix survey) utilised a combination of paper and online surveys. These were a facsimile of each other and only therefore differed only in means of access. The choice of either survey form was determined by ease of use and access. Many of the general practitioner – and some of the mental health service – surveys were done in the waiting room, while others chose to do it at home. Return rates were approximately 60 per cent. In relation to the SIP scale (“MOPF-EP Scale” on survey) only the first three of five listed items were included because of a poor correlation of items 4 and 5 with the other items and scale total. I had researched survey methodology and plain English as part of the development process. The survey had a gestation of one year before implementation and was therefore in a mature phase once it was finally implemented.

It took continual work to keep the surveys being dispensed and a team of “champions” assisted the process in all facets of the mental health service. I made a point of recurrently visiting teams to remind them of the process. Champions met monthly and an incentive trophy was won each month. Managing the survey in a busy, multi-sited health service was at times challenging, although the clinical context was overall a strength of the methodology. It ensured that we were assaying real cases in real time. My feeling is that, overall, the survey process was successful, particularly as sufficient numbers for most of the statistical tests were obtained. Several respondents complained that it was long, though in general it took fifteen minutes or less. There were no informal or formal complaints about the survey process and no communications to either the phone or email contact modes.

Scales

The Prestige Approach Motivation scale (MSPam) (Appendix) is described in the Research design section. It measures the effortful pursuit of prestige leadership, through strategically becoming valued in efficacy, invention, skill, narrative, integrity, energy, attractiveness, and knowledge. It was developed from interviews with bipolar patients – identifying core elements which related to motivation for social investment.
Scoring was developed and an informal comparison of bipolar and normal groups was made: items which correlated significantly with the total score were retained. It is a 20 item, 7 point Likert scale, where the individual items are added to a score ranging from 20 to 140. The Likert rating descriptors are shown in the MOPF survey (http://tinyurl.com/3rqwfa8). An exploratory factor analysis found that the full scale had sufficient internal consistency to warrant use of a total scale score and that there were three meaningful and psychometrically adequate sub scales. The negative esteem affect factor appeared to be closely related to both depressive symptoms and MDQ bipolarity. These findings suggest a core relationship between this factor and bipolarity; perusal of the items highlights the relationship between the threat of loss of esteem and the need to repair this. The MSPaM scale is normally distributed with a skewness of -0.44 and a kurtosis of -0.16. The mean for the spectrum cohort was 77.95 and the median 81.00. The Cronbach’s Alpha for the scale is 0.93. The convergent validity of the scale has been tested against the CSW.av scale and the correlation found to be 0.58.

The MOPF Social Inclusion Sensitivity (MSIS) scale seeks to mensurate an individual’s affective sensitivity to social inclusion and exclusion. It complements the MSPaM motivational scale by its emphasis on affect in a state of prestige loss or gain. It is a five item scale (http://tinyurl.com/3rqwfa8) which utilises a seven point Likert measure. The items are summed to a total score. The scale was developed solely on the premise that social inclusion is a prime factor in the development of affective disorders. In preliminary tests it was found that individuals rating highly on sensitivity to social inclusion also rated highly on sensitivity to social exclusion and therefore the both valences were added together in the total score of between 5 and 35. In the spectrum cohort the mean was 24.13 and the median 24.50 and the scale was normally distributed, with skewness of -0.49 and kurtosis 0.02. The Cronbach’s Alpha was 0.80 and the small number of items did not allow for factor analysis. The scale has been compared to the BASrr (reward responsiveness) subscale of the Behaviour Activation Scale (BAS) and found to have convergent validity. MSIS is considered one of the sociometer sensitivity scales and a proxy for HESE and was found to be a mediating factor in several of the dynamic model’s relationships. Most particularly, the values for MSIS against spectrum node are depicted in Figure 24.
Similarly the hypomanic, depressed, and manic (mixed) cohorts (Appendix Table 44) all have higher social inclusion sensitivity scores than the euthymic cohort. Yet causality is unclear – does a mood swing make one more sensitive to social investment or is sociometer sensitivity a precondition for a mood swing? The data depicted suggest that social inclusion sensitivity (MSIS) is a both a structural factor implicit in spectrum subthreshold and clinical groups and a factor which changes with mood.

The Social Inclusion and Prestige (SIPS) scale was extensively utilised in the research to depict actual community social investment. This is clearly difficult for a cross sectional subject-based survey, as prestige conceptually involves all of the people in the individual’s social world and their composite level of social valuing. This is not only a self-assessed social phenomenon, but one which is dynamic over time. There are other scales which measure social inclusion, though these were not known at the time of survey inception. To undertake the task of measuring prestige, three items were chosen and these measured annual number of birthday wishes, size of support group and the number of various messages received each week. Each was mensurated according to a numerical range, with the intention of making the measure objective rather than subjective. The means of each range were added to form a score. In multiple regressions the residuals for this measure were not normally distributed and hence the score was log transformed to nlogSIPS. This reduced the skewness from 1.07 to -0.24 and the kurtosis decreased from 0.23 to -0.67. While nlogSIPS is used throughout the study, it features prominently in the theory and measurement of the dynamic model, where it interfaces with self-esteem, MSPaM, and depression. All of the relationships in the dynamic model were confirmed by path analysis, including those of prestige. Prestige (nlogSIPS) was also used as the denominator for the sociometer calculation, which calculated the ratio of self-esteem (numerator) relative to prestige. This sought to provide a measurement of how individuals were valuing themselves relative to their actual prestige. In a similar vein, a measure of prestige approach motivation relative to actual prestige was obtained and found to be highest in “mixed” (manic) conditions. All of these manoeuvres generated outcomes with face validity. With a Cronbach’s Alpha of 0.76 the SIPS scale has internal consistency, though its test-retest reliability, convergent and construct validity remain untested.
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The TENSION scale is a single item seven point Likert scale which rates the statement “Lately I have been feeling tense”. It was included because of the ubiquity of tension in mood syndromes. Rather than assess anxiety, which has significant cognitive overlay, it attempts to tap a biological fundamental – tense arousal (Thayer, 1989). The scale was used throughout the exploratory analysis, particularly in the areas of manic (mixed) and hypomanic states. Results from it were incorporated post hoc into the dynamic model by reason of its strong relationship to MSPaM and depression and the fact that it contributed strongly to the path analysis. It was found to predict prestige – as was MSPaM – but only in the presence of high mood elevation (Figure 15). Tension became a differentiating factor in the demarcation between “mixed” (manic) states and hypomania. It helped to demonstrate that depression and tension scores paralleled each other across the spectrum. The TENSION scale was close to normally distributed with a skewness of -0.22 and a kurtosis of -1.08. It therefore has content validity and utility within the context of this work, but its reliability remains untested as does its convergent validity with extant scales.

The SISE scale measures self-esteem on a single item five point Likert scale and has been found to be valid and reliable (Leung & Xu, 2013; Robins et al., 2001). In the MOPF sample the scale was mildly platykurtic (skewness -0.03 and kurtosis -1.11). The scale has been utilised ordinally in MOPF dynamic model correlation analysis (Spearman correlation) and parametrically in line charts and in path analysis regressions. Likert scales are ordinal and some authors have considered the parametric evaluation of ordinal data to be a “sin” (Kuzon as cited in Norman, 2010). Opinions are divided between purists and liberals. Norman (2010) opines that “one of the beauties of statistical methods is that, although they often involve heroic assumptions about the data, it seems to matter very little even when these are violated.” If the data is adequately distributed, inferences about a wide range of measures can be drawn on the numbers, even if inferences about the “underlying, latent, characteristic reflected in the Likert” structure cannot. Perhaps more importantly, the measures were all treated as continuous and produced acceptable residuals in simple and multiple regressions. This discussion relates as much to the 7 item Likert TENSION as to the 5 point SISE scale and the same conclusion is drawn in both cases. Given the cogent views of Norman (2010) and the valid model assumptions it seems reasonable to use
Likert scales parametrically, even though this remains an area of some contention. The 13 item MDQ scale, though largely used for its threshold arbitration, is summed and therefore parametric and has been shown to have a cogent factor structure and utility in dimensional assays (MacIntyre, 2013). It is therefore reasonable to use the scale in correlation analysis and regression.

**Diagnostic Allocation and the Mood Disorder Questionnaire**

A potential limitation of this study is that clinically selected psychiatric and general practice cases nominated their own diagnosis. Fifty two of 53 bipolar I (S1) subjects originated in the mental health population – where clinical diagnosis was applied for case selection. Forty eight (91%) of the bipolar I (S1) group endorsed a suprathreshold MDQ score (strongly suggesting bipolar disorder) – including 1 subject with a self-nominated “no psychiatric disorder”. Six bipolar I (S1) respondents (all arising in the mental health population) indicated a diagnosis of “depression” and this was rectified by means of the MDQ. Forty six of 51 (90%) of S1 responses indicated they had been hospitalised, while 31 (61%) said they had been told they were psychotic when most ill. Control subjects, on the other hand, indicated “no psychiatric disorder”, scored below 3 on the MDQ and were non-cases on the ASRM and the PHQ-9 and had no bipolar family history. Therefore, for bipolar I (S1) and control (S7) cases, in particular, that there is a strong case for diagnostic fidelity – and these groups constitute the principle comparison. Cases in the spectrum between extremes – justifiably – entail some spectral ambiguity, principally relating to the imprecision of the diagnostic group depression in general practice which makes allocation to these nodes probabilistic. Diagnosis was augmented by the utilization of family history, MDQ and mood scales in the diagnostic algorithm.

Stuart et al. (2013) have described a study in which they tested the reliability of self-reported depression – compared to the Structured Clinical Interview for DSM-IV-TR (SCID-I/NP) – in a large community cohort. They found that the level of agreement between methods was reasonably high – 0.61 sensitivity, 0.89 specificity and overall agreement (kappa) 0.5. The authors note that past research has validated self-report for chronic well-defined conditions like hypertension. They concluded that
simple self-report methodologies may be used in the identification of depression “with some degree of confidence.” (p. 1). Given the validity of self-reported depression, it is to be expected that self-reported bipolar disorder – being a prototypal condition usually diagnosed by specialists – will have a higher reliability still. In support of this, 60 of 75 (80%) MOPF respondents judged to be bipolar had previously been told by a health professional that they had either manic depressive illness or bipolar disorder. Mental health service respondents (52/53 bipolar I) were generally treated over an extensive period through hospital and community phases of illness. All respondents had regular doctors and care coordinators and psychoeducation was an integral part of treatment. The small number who misquoted their diagnosis were likely to have had their diagnosis correctly re-allocated by the MDQ. Other cases with non-volunteered current state mood syndromes of relevance were detected using the PHQ-9 and ASRM scales and diagnosed accordingly. The bipolar I cases approximated the Longitudinal Expert and All Data (LEAD) gold standard of Spitzer (1983). Family history data were reliant on the MDQ prompt and did not entail a structured interview of respondents or families, therefore the data arising are provisional, though as good as one can get in a survey.

There is a potential criticism of the allocation between bipolar I and bipolar II on the basis of hospitalisation or reported psychosis – because hospitalisation may arise for non-manic causes and psychosis may occur in depressive illness. I believe that the boundary used demarcates more simple community-arising bipolar II (S2) – where hospitalisation has not occurred – from mental health service selected (52/53) bipolar I (S1). Yet there will be true bipolar II persons who have been hospitalised for depression and its complications or comorbid personality disorder who may be mislabeled as bipolar I (S1). This leaves the distinction as a working approximation, but nevertheless the best that could be achieved in this survey. Cases diagnosed by MDQ as bipolar II (as opposed to those self-nominating diagnosis) were included in the S2 category to obtain as broad a spread of community type II “bipolarity” as possible, albeit using an approximation.

The Diagnostic Algorithm and Spectrum Calculator (hierarchical) (Appendix) reveal that original strict MDQ criteria of Hirschfeld et al. (2000) were adhered to.
This involved a moderate or severe rating of impairment for MDQ positivity. A diagnosis was first allocated and subsequently a spectrum group was selected. The algorithms follow a hierarchy, such that a positive bipolar disorder *trumps* indications of depression and that this trumps *control* status etc. Self nomination of “bipolar disorder” was sufficient for allocation to either S1 or S2 (pending confirmation of polarity). Secondly, if the bipolar quotient (Diagnostic Algorithm – Appendix) – involving previous clinician diagnosis and MDQ – was positive, the diagnosis was also “bipolar disorder”. Undiagnosed – but PHQ-9 positive – depression indicated a broad depression diagnosis, though this may have been inflated by reactive, characterological and organic conditions. Self-nominated depression followed and so forth. The Spectrum Calculator (hierarchical) (Appendix) was subsequently applied and is relatively straightforward, with the exception of the subthreshold group (point 7.). I found that there were nine cases of categorical ASRM mood elevation ((hypo)mania) in the control (S7) node and this posed a problem for the underlying construct. Evidently the algorithm had overlooked what probabilistically were subclinical mood elevation cases and these were essential for making inferences about the bipolar spectrum. After discussion with my supervisor, I chose to break the protocol and situate these people in the S4 subthreshold node. I remain unsure what the finding of ASRM positivity in a normal population may mean – whether subclinical status – or a false positive test, or a combination of the two. Figure 31 demonstrates that all bar three (of twenty three) of all cases with categorical mood elevation in the total cohort had either subthreshold or suprathreshold MDQ scores. I therefore decided that, in accord with the spectrum *philosophy* of defining broadly distributed bipolarity, normal population ASRM positivity should be taken to mean something of pertinence – subthreshold bipolarity (S4). This, of course, has implications for the residual control group that may be of relevance, as it is possible that the nine cases moved had higher MSPaM ratings. This might have deflated the control (S7) group mean MSPaM and inflate the difference between the S1 and S7 nodes. As spectrum allocation cannot be undone, I am not able to directly test this, though Table 9 reveals that the S4 node mean MSPaM was not significantly higher than controls (S7) and the analysis in this case was powered to detect difference.
The MDQ was therefore used in some instances to “bump” people up the bipolar spectrum and self-nominated diagnosis was, in these instances, overridden. A categorical positive MDQ rating (allied with the symptoms occurring in the same time period with moderate or severe impairment) meant that a person was allocated to bipolar nodes S1 or S2. Is this reasonable? Zimmerman (2011) has railed against the MDQ as a diagnostic test, particularly in the general (and general practice) populations. His main criticism is that in contexts of low prevalence, with a low positive predictive value, the majority of positive-testing persons will have actual diagnoses other than bipolar disorder. Other diagnoses included borderline personality disorder, depression, anxiety and substance use disorders. In addition, Zimmerman (2011) notes that the MDQ was poor in detecting bipolar II disorder when compared with bipolar I disorder. However high specificity (0.87 overall) and high negative predictive power means that a negative test is likely to indicate an absent bipolar diagnosis. Zimmerman (2012) emphasises that despite its use in prevalence studies, the MDQ is a screening test and not a diagnostic proxy. In a general practice study on patients with depression treated with antidepressants, Hirschfeld et al. (2005) found that 21.3% screened positive for bipolar disorder, utilising the SCID as the gold standard. In this study the sensitivity – true positive rate – was 0.58 and the specificity a high 0.93. How do the MOPF data stack up with this?

Compared to the SCID, the main MOPF standard to compare MDQ positivity with is self-professed diagnosis (Appendix Table 60). Those with no professed diagnosis had an MDQ positivity of only 0.06, indicating a 0.94 specificity using this silver standard, comparable to Hirschfeld et al. (2005). In those professing a bipolar diagnosis the MDQ sensitivity was 0.75. In persons with self-professed depression the MDQ positive rate was 23.8%, strikingly similar to Hirschfeld et al. The second comparator employed was by comparing MDQ positivity against whether or not the individual had been told they have bipolar disorder by a health professional. Forty seven of 67 of those told they had bipolar disorder were MDQ positive (0.70 sensitivity), while only 15 of 152 of those not told they had bipolar disorder were MDQ positive (0.90 specificity). These findings suggest a brighter interpretation of the MOPF data than Zimmerman (2011) would predict. With sensitivities of 0.74 (self-diagnosis) and 0.70 (health professional diagnosis) there are likely to be
individuals without bipolar disorder who get bumped up the spectrum, though this is unlikely in the mental health survey sample, where prevalence of bipolar disorder was high. Inappropriately bumped persons are more likely to have come from the general practice cohort, given the MOPF MDQ’s overall positive predictive value of 0.87 in the total sample. In the GP sample of 105, there were 6 MDQ positive cases, of whom 4 professed not to have bipolar disorder. While this indicates a positive predictive value of only 0.33, the low n, and the probability that some of these individuals in fact had bipolar disorder, makes it exceedingly unlikely that the use of the MDQ significantly skewed the results.

Causality

The claim for causality is a major issue for science and Sutherland, Spiegelhalter, and Burgman (2013) have recommended tips for interpreting scientific claims. Science is imperfect, they opine, and its main challenge is to tease apart competing possibilities. Measurement is inexact with extant scales, let alone scales such as the MSPaM, which has not been replicated in other centres. Yet it can be claimed that standard statistical methods yielded significant results for the MOPF data, some of which were corrected for multiple analyses (ANOVA, ANCOVA). Biases are an issue in research; for instance the author’s biomedical training on causation of mental disorders. The early inception of the study was based on hunches and followed from a small trial sample. Yet hypothesis formation is a type of bias and without it the process of science cannot progress. I took particular care in the formulation of the survey scales not to use leading questions and, through the Likert method, to provide an unbiased response scale. It was paramount to provide little inkling in the survey of the research’s focus on prestige as a contributing factor to spectrum conditions.

Sutherland et al. (2013) argue that bigger samples sizes are usually better. While they suggest drug trials with tens of thousands of participants, power analysis indicated that the MOPF sample of 228 would be adequate for the ANCOVA analysis of structural differences between spectrum groups. However, the pseudounipolar and simplex groups had only 17 members each, while the hypomanic cohort numbered only 23 and this limited some statistical inferences. Overall, with respect to the other
analyses, the sample size was reasonably large and statistical inferences could be soundly drawn. Yet being a case-control study with a cross-sectional method we run into a classic problem in research, that being that “correlation does not imply causation” (p. 336). We cannot see the causal “force” transmitted between MOPF groups, only the association between each. Association may be due to common cause or common effect relationships rather than linear causality. Whilst path analysis in the dynamic model supported a causal set of relationships, it could not prove this. In fact, were the claims proven, they would form part of a causal homeostasis. Arguments are therefore inductive. Multiple regression provides some amelioration of the problem of causality, though only partially.

With respect to the dynamic model – the stealth and hypomanic theories – at least a causal mechanism is suggested and this was part of a priori hypothesis testing. Putative mechanism preceded analysis of the data. With respect to the structural model, prediction testing was also applied, though no mechanism was initially suggested – it was based on clinical observation and hunch.

Sutherland et al. (2013) warn against extrapolating beyond the data. The MOPF research can make a tentative correlative truth claim for male and female angloceltic persons in the adult age range in Australia, though matters might be different for other populations such as different ethnicities. Sampling and survey delivery methods may impact on results; the MOPF research obtained its control group from a single general practice. While this was likely to be representative of the general help-seeking population, the possibility of sample bias exists. Sutherland et al. (2013) further advise for replication and, where possible, meta-analysis to improve statistical power. The MOPF research has not been replicated and this limits validity and reiterates the proof of concept nature of the study. The authors also advocate the adoption of results with large effect sizes; in the MOPF study Cohen’s $d$ was routinely utilised to provide meaningful analysis of the data. For example, in the structural model the pseudounipolar (S3) node had a 16.98 point difference to the control (S7) group with a $p$ of .051 – according to rigid NHST this is insignificant. Yet this group had only 17 members and the $d$ was large at 0.86 and, as such, a meaningful association between MSPaM and pseudounipolar status was proposed.
Apart from the structural and dynamic analyses, which are central to the study, some of the MOPF analysis is based on non-primary hypothesis methodologies. This may be seen to limit inference about some of the aspects of prestige theory. Yet, as the study was exploratory and sought to provide proof of concept, rather than definitive findings. The findings in the study were made on the basis of hypotheses as they evolved and not as part of “data dredging”.

Claims for causality may be enhanced by temporal precedence; bipolar family history and perceived childhood relational trauma (PCRT) are instances of this. Bipolar family history was found to be a factor in the development of high MSPaM when the simplex (S5) and control (S7) groups were compared. This supports claims that bipolar genes and prestige approach motivation are linked, though further studies would be required to support this. It is supported also by the cross-sectional relationship between bipolar caseness and high MSPaM. Similarly, there was a relationship between PCRT and social inclusion sensitivity with a $p$ of $<.001$ and a $d$ of 0.56. This supports the claim that childhood adversity makes spectrum persons more likely to be socially insecure, though the measure of PCRT is prone to confounds such as depression level. A further problem exists – it may be possible that persons with social inclusion sensitivity have a faulty recall bias to their childhood – hence a higher PCRT! This is reverse causality despite putatively temporal precedence. Confounds are many in correlational research – they require longitudinal and experimental enquiry to disentangle. I emphasise again, this research is exploratory and “proof of concept”, not definitively causal.
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

Implications and Future Directions

In this section I will consider the prestige theory of spectrum bipolarity and attempt to draw meanings for the professions of psychiatry and psychology, for society and for the individual with a bipolar spectrum condition. I will draw on the original theoretical premise that life events, cognition, social support and mood state revolve around the axis of self-esteem and that this, in turn, is inextricably married to perceived societal investment. We will revisit the models of hypomanic reentry, depressive stealth, dynamic bipolarity, the prestige spectrum, and systemic contouring to reach a conclusion about the utility of the model. Some new concepts will crystallise the work that has gone before.

Hypomanic Adaptiveness

One of the implications of this research is that mild to moderate hypomania is adaptive to social role function. This may be an erroneous assertion, given that hypomania is a clinical condition which may be associated with some degree of impairment in social or occupational functioning (American-Psychiatric-Association, 2013). Indeed, opinions about the adaptiveness of the condition are divided – while clinicians may think of it as a morbid state – Judd et al. (2005) entertain the idea that mild and subthreshold hypomania may enhance functioning in bipolar II disorder. Judd et al. found a nonsignificant increase in psychosocial function as patients transited between asymptomatic and subsyndromal hypomanic status. Hypomanic bipolar II persons have the same level of psychosocial function as when they are asymptomatic. Further, Akiskal, as cited in Judd et al. (2005) suggested that the positive hypomanic signs of cheerfulness, jocularity, gregariousness, confidence, sexual drive and vitality potentially improve functioning. In a similar vein, Tijssen et al. (2010) found evidence that hypomanic symptoms were a common adolescent phenomenon and that lower grades of this rarely came to clinical attention. Nevertheless, in light of the lack of statistical significance to the increase in function between baseline and subthreshold hypomania, the suggestion of Judd et al. (2005) merits caution. Higier et al. (2014), however, found a significant increase in sociability and verbal functioning in twins of bipolar probands compared with both
community controls and bipolar patients. They concluded that the liability to bipolar disorder may confer traits which impact positively on reproductive fitness.

The MOPF research relies on the finding that prestige is higher in the (hypo)mania category than in euthymia (Appendix Table 47, Table 48). Prestige approach motivation and prestige are furthermore positively correlated in the (hypo)manic group \( r = 0.51 \), while social inclusion sensitivity (MSIS) appears to be higher in (hypo)mania also (Appendix Table 43, Table 44). It is possible that mood elevation may distort prestige (nlogSIPS) by merit of extreme response bias – a sense of bravado which leads to an inflated number of birthday wishes, increased intimate circle and social contacts. Such inflated ideas may be seen in hypomania and this possibility cannot be ruled out. It was found that the subthreshold (S4) node had the highest prestige of any category (Figure 23), though this was not significantly different from the control (S7) node – the difference in nlogSIPS being 0.29 points (95% CI [-0.83, 0.25], \( p = 0.70 \). Therefore, statistically speaking, the prestige of the subthreshold (S4) group is at least no worse than the controls (S7), even given undiagnosed bipolar symptoms and history. Given the positive MOPF findings and the suggestive Judd et al. (2005) results – and in the absence of refutation – it seems reasonable to entertain the possibility that mild hypomania improves function, until further evidence is available. It should be noted that adaptiveness is not equivalent to adaptation. In the latter, if it could be shown that hypomania leads to increased fitness, then it would represent a type of adaptation, albeit only in a frequency dependent manner.

If hypomania has function – if it increases social inclusion in accord with the prestige model – then there are major implications. The possibility of function changes the “aboutness” of the condition and demarcates it strongly from mania, where in modern societies dysfunction is the unmitigated rule. Mania may be a pathological byproduct of adaptive hypomania. People with subthreshold/mild hypomania have a socially attractive presence. They energetically push boundaries with self-confident ideas, performing for conspecifics and the group. Elevated affect in the actor prompts elevation in the recipient. Such states prompt individuals to achieve social goals where incorporation is the object. From the prestige landscape
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view, these individuals (broadened to include hyperthymic persons) are the affective/prestige peaks in Motivated Approach, around which society flows. They are the prestige leaders of the present. Of course hypomania is transient – the “flame” burns out – and other persons with mood elevation take their place until the next cycle.

Another implication to this alternate way of viewing mild hypomania is that it should no longer be viewed as a disorder. Indeed, such a novel move would recognise four levels of mood elevation:

1. Normative happiness consonant with an achievement – *enjoyment*
2. Subthreshold or mild hypomania – *status excitement* (adaptive)
3. Hypomania (that which is truly “sub-mania” – *agitated (tense) excitement*
4. Mania – mixed and cycling states of *anger, excitement, agapic joy and distress (lability,) and sorrow*

We all know the feeling of enjoyment when something which increases prestige occurs, while continuous states of excitement indicate a deviation from normative functioning. These may be conceived of as subthreshold or mild hypomomic conditions. One of my insightful patients could describe a difference between (2.) subthreshold/pleasant and (3.) tense and irritable hypomania, while many bipolar I persons are able to recognise the difference between all four states. It is conceivable that this differentiation is dimensional rather than ordinal, with tension increasing monotonically as the transition occurs from (2.) to (3.) and so on. As currently conceived, hypomania subsumes (2.) and (3.), though for theoretical reasons the two may be divided. The division may have significant diagnostic and therapeutic implications.

In the regression fitted line plot of TENSION into prestige (Figure 15), as tension increased – in the context of an ASRM level of 10 ((hypo)mania) – so prestige increased (Figure 34). The manic (“mixed”) group had TENSION levels categorically
higher than the hypomanic group ((hypo)manic in the study) \( p < .001 \) and prestige levels categorically lower. As tension levels increased in the (hypo)manic cohort, so too did depression level (Figure 32) – suggestive of mixity. According to the dynamic model, tension is the prime driver of prestige approach and withdrawal. Though the mean levels of tension were equivalent between the (hypo)manic and euthyemic cohorts, there was clearly a greater spread in the hypomanic cases (Figure 29, Figure 30). According to the model described above, status excitement entails moderate excitation and low levels of tension, while clinical states emerge as tension increases and prestige rises – to a point (TENSION \( = 5 \)) – when sub-mania and eventually mania supervenes. It may be that when tension levels are extreme – where both approach and withdrawal co-occur – that the system tilts toward mania.

Whilst hyperthymia may be a static form of subthreshold hypomania, typically hypomania is a cyclic condition. Why might this be so? It has been posited that mood elevation may be a response to either goal success or else unstable prestige status. As such, it is designed for a task – to rectify or improve prestige. Low prestige may be associated with depression, which is clearly a damaging neural state (Neumeister, Charney, & Drevets, 2005) and it may be beneficial to have a “circuit breaker”. Cycling may be a consequence of opponent process motivation (Solomon & Corbit, 1974). But surely tonically high prestige would be a good thing and cycles would be counterproductive? It is possible that persistent hypomania is biologically unsustainable and may cause neural damage as might be suggested by inflammatory and oxidative stress models of bipolar disorder (Berk et al., 2011). Individuals with persistent mood elevation would therefore be at an evolutionary disadvantage. Similarly persistent hypomania may be a precursor to mania, which is likely to be neurally and socially toxic. It is possible also that hypomania has its social advantage by means of its novelty. Persons chronically affected may “outstay their welcome” and become aversive. In this sense the functionality of mild hypomania may be tied to its cyclic nature.

**Depressive Adaptiveness**

The case for the stealth model of depression has already been made and the similarity to the model of Allen and Badcock (2003) drawn. What does this model
mean for psychiatry, psychology and society about the nature of depressive affect? The dynamic model path analysis showed that depression is a full mediator in the relationship between tension and prestige. Figure 26 depicts the relationship between prestige and depression level and indicates that at low levels of depression prestige is high. As depression increases further, prestige falls, but past the clinical level of PHQ-9 ≥ 10 the two variables do not covary – they reach a plateau. Why does prestige not continue to fall as depression rises, in accord with common sense? The asymptote level is around 2.75 – above the first prestige quartile of 2.60, though significant spread is notable. I have suggested that this pattern occurs because depressive cases find a niche where lower prestige is stabilised – so-called stealth. This may have something to do with the sick role that is earned by clinically ill individuals, but not earned by the subsyndromally unwell. Prestige may be stabilised by a caring social milieu and this may be an unconscious object of depression.

This relationship is not maintained when the PHQ-9 impairment item is utilised in place of the prestige item. Conceiving PHQ-9 depression score as the independent variable – and PHQ-9 impairment as the dependent variable – the correlation (Spearman) between the two was + 0.81 (n = 205). Therefore, as depression increases impairment closely follows. Combining the prestige and impairment results, we may speculate that while people have increasing difficulties with work, home and sociality, their investment from the community need not fall. Stealth allows individuals – as they increasingly become incapacitated from depression – to maintain a statically low prestige and not go down the “social sink”.

Following from the abovementioned ordinal scale for elevated affect, a scale for lowered affect is suggested:

1. Normative low mood consonant with prestige loss – sorrow

2. Subthreshold or mild melancholia – status sorrow

3. Agitated depression – which may be considered a form of subthreshold mania – agitated (tense) dysphoric melancholia
4. Mixed depression and mania – mixed and labile cycling states of tension, anger, sorrow and excitement

Nosology

"He felt that he had to make people laugh, as if this were 'almost a duty', and mania is perhaps a way of trying to keep one's audience alive, afloat, to keep an addressee right there in front of you." (Leader, 2013, p. 26)

DSM-5 American-Psychiatric-Association (2013) classifies mental conditions in an atheoretical manner. Psychiatric caseness may be defined where there is a persistent and pervasive disorder which causes distress, dysfunction and disability. Data and modelling from the MOPF study may in the future inform psychiatric categories and dimensions and Figure 40 draws together the strands of this thesis and presents a conceptual framework based on the interaction of HESE approach and withdrawal as they intersect with tense arousal.

In Figure 40, prestige approach (MSPaM) and prestige withdrawal (PHQ-9) are retained as basic motivational responses of animate creatures; they are drawn in this figure as two orthogonal axes. Similarly, Phelps (2008) drew a model in which he juxtaposed axes of mania and depression, thereby locating mixed states. Tension is conceived as an axis running orthogonally ($90^\circ$) to approach (appearing as $45^\circ$ on the XY plane). However, the 0.70 correlation between depression (withdrawal) and tension indicates that these axes are not orthogonal, but related – and therefore running at perhaps $-30^\circ$ to each other on the XY axis. Tension is synonymous with tense arousal (Thayer, 1989) and with danger orientation. DSM-5 (American-Psychiatric-Association, 2013) describes this construct under the rubric of anxious distress, where individuals experience being “keyed up or tense”, “unusually restless”, “difficulty concentrating because of worry”, “fear that something awful may happen” (p. 149) or fear of loss of control. In this model tension may become superposed on the hedonic axes through the bipolar I and II dispositions and lead to a modification of the original hedonic substrate.
Figure 40. Theoretical three dimensional schematic of the bipolar spectrum mood syndromes according to the interaction of prestige approach (Y) and prestige withdrawal (X) motivation along with TENSION (Z).

On the approach axis, increasing levels of approach (without tension) from euthymia on lead to increasing excitement, which in subthreshold cases may become a fixed state – status excitement. Status excitement is notable for a persistently elevated mood which is abnormal and which is a subset of the clinical condition hypomania. It is unlikely to lead to disability. Indeed, as Judd et al. (2005) intimate, it may lead to an increment in function. This is contrasted to the comments of Nusslock and Frank (2011), where subsyndromal hypomania was associated with role impairment, though it appears that their samples had coexistent major depression. Certainly, in the MOPF data, subthreshold (S4) cases had tension and impairment levels which were grossly equivalent to controls. Status excitement is a component of the subthreshold conditions which Merikangas et al. (2011) discuss; they cite evidence that this group
of conditions may affect up to 6% of the population. In contradistinction to Tijssen et al. (2010), they note a high conversion to bipolar disorder in youths with subthreshold mania. Therefore, subthreshold hypomania may lead to a clinical condition, but it is uncertain whether tensionless status excitement forms part of this progression.

Should tension commensurately increase, then clinical hypomania (submania) arises. These varying levels of tension and approach describe the progression from the bipolar II to bipolar I diatheses. In the MOPF research, hypomanic cases had non-simultaneously higher MSPaM and significantly elevated prestige, while tension was spread around a mean which was equivalent to controls. Some hypomanic individuals therefore had reduced tension (compared with controls) and it may be that they were status excitement cases. The MOPF study was not powered to otherwise differentiate between low and high tension hypomanic subjects.

Prestige loss may trigger sorrow in susceptible individuals, which in its full manifestation takes the form of status sorrow. This is a condition of low-tension depression, which may be transient or persistent and may be seen clinically as retarded depression. Concurrent activation of tense arousal – with an increment of approach – results in more agitated forms of depression as individuals move within the withdrawal arm of the bipolar I and II diatheses. In support of this claim, while there was no correlation between TENSION and depression level in the mild depression group ($r_p = 0.09, n = 38$), there was a moderate relationship between these variables in the clinical depression cohort ($r_p = 0.38, n = 65$).

I am suggesting that these two approach and withdrawal excursions – with or without mild to moderate tension – fully describe bipolar II disorder. Key elements to this model are fixity over time – where mood states become locked on the axes (hence a persistent disorder) and mixity – where two or three mood states become superimposed by merit of the three dimensional structure. It needs to be mentioned that other arousal axes are often involved in mania, including the LUST (Panksepp, 1998) system evident in raised sexual desire, and ATTACHMENT – Panksepp’s Integrative Emotional System for Social Affect (Panksepp, 1998) – where agapic feelings rapidly oscillate with distress in affective lability.
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The clinical condition of unipolar melancholia is best described by the range of psychomotor retarded status sorrow to agitated depression; in the latter, psychosis may become evident as tension levels rise. Psychotic melancholia appears more common in agitated states (Parker, 2007), consistent with high tension, while if tension is in extremis, states of approach and withdrawal may occur together, accounting for the high levels of depression in mania. At these levels the manic person is prone to psychosis. In more extreme states still, mixed states may ensue as demonstrated by depression and mood elevation co-occurring with lability of mood and psychosis. Bipolar I disorder thus occupies a narrower range of extreme tension with higher combined approach and withdrawal motivation than bipolar II disorder in this model.

In Figure 33 the plot marries with the predictive model depicted in Figure 40, somewhat flattened perhaps because of the postulated -30° separation between TENSION and depression. The contour plot indicates that euthymia and hypomania are proximal, while mild depression has a similar approach score, but a slightly higher TENSION level. Depression (largely bipolar) is an approach-withdrawal high tension state, as indicated in Figure 40. Mixed mania has a higher approach level still. Disorder severity is linked to tension level, as predicted.

The structural prestige spectrum (Table 6) presented seven affective categories for evaluation. It found that prestige approach motivation was raised in the bipolar I (S1), bipolar II (S2) and pseudounipolar (S3) categories. Factor analysis found that negative prestige-motivated self-esteem (NEGESTAFF) was the only factor which predicted bipolar disorder. High social inclusion sensitivity (MSIS) was shared by all of the clinical nodes (pre-ANCOVA Figure 24) – and also the S4 subthreshold node. How might these findings affect nosology? The data suggest that bipolar I (S1), bipolar II (S2) and pseudounipolar (S3) disorders may be provisionally validated diagnostic categories, by merit of differentiation on the basis of MSPaM, as well as clinical features. These findings were enhanced by extracting subthreshold and bipolar family history cases from the non-cases, suggesting that this maneuver may have diagnostic utility also. Subthreshold (S4) cases had higher MSIS than controls. Similarly, the possession of a bipolar family history predicted higher MSPaM in both

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depressed and otherwise normal categories according to T-tests, with the caveat that some subthreshold MDQ cases were included in those with positive family history (F5). It seems reasonable that family history was of a higher diagnostic order than non-case level MDQ. Together, these findings suggest that prestige may be woven into the bipolar tapestry and that diagnostic differences may be found by combining several factors together – symptom profile, bipolar family history, tension level, subclinical MDQ rating, prestige approach motivation and social inclusion sensitivity. These would profitably be added to those features proposed by Ghaemi (2008).

Unipolar (S6) cases failed to hold a pattern in the contour analysis and depressive level was not correlated with MSPaM, as it was in the control, subthreshold and bipolar groups. This group did not follow the “norms” for bipolarity or spectrum behaviour. Clinically, people commonly qualifying for a unipolar diagnosis are constituted by adjustment disorders, characterological cases and a subgroup of true melancholia. “Major” (severe) depression is a loose and imprecise proxy for diagnostic subtype” (Parker, 2007). Diagnosis may be based partially on evident distress rather than intrinsic anhedonic and anergic depressive features. For these reasons, it is understandable that unipolar depression is different to bipolar depression with respect to prestige behaviour. Apart from agitated varieties (sensu Figure 40), unipolar depression entails little of an approach dimension, thereby limiting its termination capacity. Frankland et al. (2015) have reported significant differences between bipolar and unipolar depressive phenomenology. In particular, the bipolar I depressive episodes show more psychomotor retardation while bipolar II episodes show greater mixed features – the mixed features consistent with higher tension, approach and withdrawal per Figure 40. Hypersomnia (atypical depression) – and also waking when no one is up (EMW) – along with psychomotor retardation all limit interaction with the tribe. Rejection sensitivity, as seen in atypical bipolar depression, reflects social vigilance per the model of Allen and Badcock (2003). These factors together indicate the utility of bipolar prestige withdrawal. There are no currently evident advantages to mixed features, except perhaps incapacity and sick role entitlement.
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The path analysis for categorical bipolar disorder found that MSPaM was a predictive factor, but only with absent bipolar family history. This suggests that there may be bipolar subtypes based on both prestige and inherited factors, alongside the customary bipolar I and II division. Future researchers will unite these and other findings from initiatives like the Research-Domain-Criteria-(RDoC) (2012) to find better models and categories for the bipolar spectrum. Whilst acknowledging that the project of the bipolar spectrum seeks to identify common aetiogenic pathways and establish improved nosology and treatment, Strakowski, Fleck, and Maj (2011) urge caution until research provides evidence of greater validity.

Treatment

“Consciousness is not always in this form. For example, when we are alarmed, reflective awareness is lost and attention is directed towards the source of the threat. This kind of consciousness is ‘adualistic’. The characteristic consciousness of those people damaged by the impacts of the social environment takes this form. The aim of therapy is to restore, generate, and potentiate that particular kind of consciousness that we are calling self.” (Meares, 2004, p. 54)

Putting biological treatments aside for the moment, the psychosocial management of bipolar spectrum conditions is a matter for debate. Schöttle, Huber, Bock, and Meyer (2011) have found that research to date has used heterogenous methods for heterogenous outcomes. The majority of studies have, however, shown positive results for relapse rate, quality of life, functioning or symptomatic outcome. Interventions touch on issues such as self-esteem and performance expectations, for example. An example of an effective real world group approach was that of Castle et al. (2010), where a randomised sample of participants received either a group psychoeducational multimodal package or treatment as usual. The study found that the intervention led to fewer pooled relapses and lower morbidity. The effective component(s) of the treatment was not elucidated, though the model was based on what the authors term a Collaborative Therapy Framework, using a stress vulnerability model. Detection of stressors, monitoring of mood and coping strategies were pivotal.
A novel approach was applied by Perlick as cited in Schöttle et al. (2011) where the intervention focus was caregivers and not patients. The study found that 12-15 sessions of family-focused CBT, versus a health education video intervention, reduced caregiver depression ratings and health risk behaviour. What was most interesting was that patients’ depressive \( (d = 0.67) \) and manic \( (d = 0.34) \) symptoms were significantly reduced. Part of this finding was mediated by reduced caregiver depression. These results are consistent with the MOPF premise that improving social investment improves the dynamic symptoms of mania and depression in the patient. This may be through a self-esteem pathway. Schöttle et al. (2011) effectively conclude that “some kind” of psychosocial intervention is likely to augment treatment as usual, but that we don’t know who will benefit from which approach. The thrust for new therapies is the focus on families as mediators of the social milieu in which the patient lives.

Treatment, in an ideal world, should follow directly from pathogenesis, though in the real world it is often predicated by the orientation of the therapist: medical doctors prescribe medicine, social workers dispense social interventions, while psychologists may focus on cognition. The core premise of the prestige model is that life events impact self-esteem and social investment and form an inseparable whole as they act on mood. For this reason, therapies arising from the import of this research will target the self and the social world.

The structural model provided evidence for a high loading for prestige approach motivation (MSPaM) in the S1, S2 and S3 nodes with a trend for other nodes to also have raised MSPaM. Central to the issue of causation – and therefore remediation – is whether MSPaM is a causal or a compensatory factor in bipolar aetiogenesis. The prestige model places relational value as the trigger to compensatory self-esteem, TENSION and MSPaM fluxes. These are part of the causal chain to hypomania and mania (Figure 9). This situation is analogous to extreme fame ambitions (Johnson, Carver, & Gotlib, 2012) and goal setting (Johnson & Carver, 2006) in bipolar disposition as being compensations to potential low self-esteem. There may be a manic defence against self-esteem/prestige dysfunction and persons with bipolar disorder must manage wins as well as losses. This will be augmented by
insight to the role that self-esteem and prestige approach motivation may play in the escalation of approach tension and consequent (hypo)manic relapse.

MSPaM may be a compensation for low prestige in the bipolar depressed, manic and mild depression groups. Through the MOPF research we can garner some sense of what to treat, but not necessarily who to treat. For example, status excitement arising in the subthreshold and simplex cohorts may need no treatment. Tijssen et al. (2010) and Goodwin (2010) describe a relatively large cohort of hypomanic adolescents and young adults who need no treatment. Status sorrow may be transient and mild.

The bipolar spectrum is beginning to impact therapy; for example psychiatrists are cautious to give persons with subthreshold bipolarity antidepressants and ipso facto, may use mood stabilisers in some “unipolar” individuals. Cuijpers (2013) has reviewed psychotherapy for depression and found that all mainstream therapies, including supportive therapy, were of roughly equivalent effect when administered in a routine manner. Is it therefore possible to select a therapy specific to the prestige model? Johnson and Tran (2007) note the inconsistency of results in the psychotherapy of bipolar disorder and they draw attention to the low self-esteem, negative self-beliefs, self-blame and need for achievement that bipolar persons show. Bipolar individuals are very sensitive to environmental feedback, mirroring the emphasis the prestige model makes on social investment. It may be important, venture Johnson and Tran (2007), for clients to understand that cognitions which are self-relevant can arise directly through mood state. Ascertaining meaning is important, in the context of a strong reflective therapeutic alliance.

Prestige and self-esteem were very low in the bipolar I (S1) and bipolar II (S2) cohorts (Figure 23). The MOPF research found the bipolar I (S1) and subthreshold (S4) groups had overly strong affective reactions to social inclusion (MSIS) – be it positive or negative (Figure 24). These results mirror the prestige model’s emphasis on the relationship between positive and negative prestige events and the core of self evaluation. As a novel finding, the MOPF research found an increased Contingencies of Self-Worth score in bipolar I disorder (Figure 23). This outward locus of self-
Esteem may be contributory to the approach and withdrawal escalation we see. This is potentially amenable to treatment – in trying to disentangle the individual’s self-worth from the vicissitudes of competition, approval and appearance. Deitz (1995) – an adherent of self psychology – sees the difficulties bipolar patients have in mood regulation as a primary (i.e. innate) abnormality in self-structure and/or function. The MOPF research found that bipolar family history was associated with perceived childhood relational trauma, which itself was an independent predictor of bipolarity in the spectrum population (Table 15). Thus the origin of the disorder, at least in this cohort, appears to have both biological and developmental roots, which are both a target for remediation. Yet Freud’s therapeutic approach provided no answers: “horribly chaotic (often disabling) regressions were the rule, rather than the exception, when bipolar patients were classically psychoanalysed.” (Deitz, 1995, p. 478).

Given that the locus of pathology in the bipolar spectrum may be the interaction of self with society, therapeutic modalities which target this dimension may be especially beneficial. The Conversational Model (Meares, 2004) aims to “potentiate the emergence and amplification of that dualistic form of consciousness that William James called self.” (p. 51). Conversational Model therapy was originally used in borderline conditions, but has a general application, though a quick literature search found no references to the treatment of bipolar conditions. Conversational therapists focus on the “shifts, the movements, the waxings, and wanings” of “one’s being in a place or among persons.” (p. 52). The characteristics of the Jamesian self include duality (reflective awareness), a sense of movement (vitality) and non-linearity, amongst others. Meares (2004) argues that there “is no such thing as a self. It does not exist in the absence of an environment.” (p. 54), analogous to the self-prestige unity. He promotes therapy in which self can emerge through the experience of relatedness. Therapeutic “doubling” entails the other portraying – in vocal tone, facial expression and words – something of the patient’s experience (Meares, 2004), emphasising the role which hedonic energisation plays in the sense of self and of the other. Conversational therapy utilises non-linear forms of “play” and as such may be said to involve the PLAY system of Panksepp (1998). The role of relational trauma is central to Conversational theory, especially how it affects the “central feeling of self” (Meares, 2004, p. 60). Damage to this core can be through “attacks upon value” (p.
60) and may take the form of shaming, ridiculing and invalidation, reflective of the role which negative social interactions (Lakey et al., 1994) may play in the dynamic model’s constructs of self-esteem and tension. Conversational therapists therefore aim to potentiate self and integrate disruptive traumatic memory through utilising associative speech in the context of relatedness (Meares, 2004). Dualistic consciousness, the sense of “I” and “me”, may assist bipolar spectrum persons to understand their condition if they can reflect on the relationship of self to the pursuit of prestige. Would such an internal dialogue ameliorate the condition were it to be addressed through inner speech? The strongest of the MSPaM factors pertinent to bipolarity was negative esteem affect, which spoke to the possession of a deflated and negative self-esteem with low affect, associated with a sense of threat and a need for prominence in group inclusion. If the core of this damaged self-system can be explored in a safe relationship, then perhaps some of the consequences of bipolarity can be undone. MSPaM correlated highly with MSIS (social inclusion sensitivity), highlighting the possibility that through the transference – the relationship with the therapist – bipolar persons may be able to address their interpersonal fragility. One of the advantages of the Conversational Model is its focus on ontogeny and the basic processes of self and interrelatedness.

Bipolar escalation through the dynamic model is a fundamental problem and a target for therapy. It can be seen, from the fall in coefficients and the positive Sobel tests, that dimensional bipolarity – as reflected in the MDQ – exerts a role as a partial mediator through the complete dynamic model stem and branches (Appendix Table 34, Table 35, Table 36). While the genetic drivers of this may not be directly addressed at this point in time, the substrates – real biopsychosocial elements, the perceptual bias, the self-esteem fragility and the tension – may be engaged as therapeutic targets. On the other hand, Smith (2013) highlights the problematic nature of biopsychosocial formulation, such as in the model presented here. Such formulations reduce the totality of a person’s life to abstractions and are based on an admixture of axioms and hypotheses. Nevertheless, reasoning is required to explain why this person (or group of persons) came to be this way at this point of time. Smith goes on to explain that models require both theory and testing and the prestige model offers all three levels of formulation, albeit at a proof of concept level by inference.
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and eclecticism. Smith – were he being generous – might call the model an intuitive integration

Depression may be another matter. In the prestige model depression reflects a state of being “frozen” on the social landscape. People may “fall” into its basin of attraction – into a state of prestige withdrawal which is relatively safe from ostracism. In this clinical location, prestige approach motivation is high, but unwavering (Figure 25) and prestige is fixed at a low level (Figure 26). Not only are there genetic influences on depression, but the MOPF study found that perceived childhood relational trauma had a moderate relationship with depression score. Psychotherapies which can break the dominant effects of trauma, and assist the person to stand separate from it, may assist to thaw the ground on which depressed persons stands. Narrative therapy (Morgan, 2000) may be a means to assist people to create new stories contradicting the dominant storyline of depression. Koch, Morlinghaus, and Fuchs (2007) describe the use of dance in alleviating depression via communally stimulating hedonic movement. Activity scheduling is often used in depression – using the environment to have reverse effects on affective tone. All of these modalities may assist to disrupt the depressogenic state of prestige withdrawal. Cognitive behaviour therapy may assist here to increase hedonic motivation (eg. motivational interviewing) and challenge dysfunctional prestige-based ideas.

Panksepp (1998) and Panksepp and Biven (2012) have done extraordinary work on the mapping of neurochemicals and neuropeptides to arousal systems. This is noted and expanded in Table 18 with a view to developing a new biological substrate for the prestige model, which has implications for future directions in research.
Table 18  Neurochemicals and Neuropeptides Involved in Three (Triaxial) Arousal Systems (SMAS’s) by Approach and Withdrawal Orientation

<table>
<thead>
<tr>
<th>ATTACHMENT CARE</th>
<th>TENSION RAGE</th>
<th>HENSE PLAY</th>
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</thead>
<tbody>
<tr>
<td><strong>APPROACH</strong> PROONENT</td>
<td><strong>OPIOIDS</strong></td>
<td><strong>DOPAMINE</strong></td>
</tr>
<tr>
<td><strong>OXYTOCIN</strong></td>
<td><strong>NORADRENALIN</strong></td>
<td><strong>OPIOIDS (LOW DOSE)</strong></td>
</tr>
<tr>
<td><strong>PROLACTIN</strong></td>
<td><strong>OPIOID WITHDRAWAL</strong></td>
<td><strong>ACETYLCHOLINE (MUSCARINIC)</strong></td>
</tr>
<tr>
<td><strong>LOW SEROTONIN</strong></td>
<td><strong>TESTOSTERONE</strong></td>
<td><strong>GLUTAMATE</strong></td>
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<tr>
<td><strong>(MODULATORY?)</strong></td>
<td><strong>SUBSTANCE P</strong></td>
<td><strong>CANNABINOIDS (ANANDAMIDE)</strong></td>
</tr>
<tr>
<td><strong>ACETYLCHOLINE</strong></td>
<td><strong>NULLITY</strong></td>
<td><strong>OPPONENT</strong></td>
</tr>
<tr>
<td><strong>WITHDRAWAL</strong> PROONENT</td>
<td><strong>SEROTONIN</strong></td>
<td><strong>DOPAMINE (ANTAGONISTS)</strong></td>
</tr>
<tr>
<td><strong>OXYTOCIN</strong></td>
<td><strong>NORADRENALIN</strong></td>
<td><strong>NORADRENALIN (SNRI’S, TCAD)</strong></td>
</tr>
<tr>
<td><strong>PROLACTIN</strong></td>
<td><strong>OPIOID ANTAGONISTS</strong></td>
<td><strong>OPIOIDS (BUPRENORPHINE ETC)</strong></td>
</tr>
<tr>
<td><strong>GABA AGONISTS</strong></td>
<td><strong>CRF</strong></td>
<td><strong>CANNABINOIDS (ANANDAMIDE)</strong></td>
</tr>
<tr>
<td><strong>OPIOIDS</strong></td>
<td><strong>ACETYLCHOLINE (NICOTINIC)</strong></td>
<td><strong>ANTICHLINERGICS</strong></td>
</tr>
<tr>
<td><strong>NICOTINE</strong></td>
<td><strong>GABA</strong></td>
<td><strong>(SCOPOLAMINE)</strong></td>
</tr>
<tr>
<td><strong>CLONIDINE</strong></td>
<td><strong>OXYTOCIN</strong></td>
<td><strong>GLUTAMATE</strong></td>
</tr>
<tr>
<td><strong>SOMATOSTATIN</strong></td>
<td><strong>DOPAMINE (AMPHETAMINES)</strong></td>
<td><strong>(NOR)ADRENALIN</strong></td>
</tr>
<tr>
<td><strong>NULLITY</strong></td>
<td><strong>SEROTONIN (SSRI’S)</strong></td>
<td><strong>DOPAMINE</strong></td>
</tr>
<tr>
<td><strong>DISTRESS</strong></td>
<td><strong>NORADRENALIN (SNRI’S, TCAD)</strong></td>
<td><strong>SEROTONIN (ACUTE)</strong></td>
</tr>
<tr>
<td><strong>FEAR</strong></td>
<td><strong>OPIOIDS (BUPRENORPHINE ETC)</strong></td>
<td><strong>CCK/CRF</strong></td>
</tr>
<tr>
<td><strong>SORROW</strong></td>
<td><strong>CANNABINOIDS (ANANDAMIDE)</strong></td>
<td><strong>GLUTAMATE</strong></td>
</tr>
</tbody>
</table>

*Note.* Proponent molecules drive each affect program (eg PLAY), while opponent substances oppose arousal in the affect program. Systems (with the exception of TENSION) are generally unidirectional across approach and withdrawal. Neurochemistry taken from Panksepp (1998) and other sources.
Table 18 depicts three arousal systems pertinent to both prestige and affective disorders – ATTACHMENT, TENSION and HESE. Each has an approach and a withdrawal arm (affect program) in which neurochemicals and neuropeptides act. These agents may be proponent (pink) for the relevant affect program or opponent (blue) (Solomon & Corbit, 1974) in that opponent neurochemicals tend to return the affect program to nullity. As an example, HESE is constituted by approach (PLAY affect program) and withdrawal (Sorrow affect program). Dopamine, cannabinoids (anandamide) and opioids etc. (at the right level) facilitate play, while serotonin and dopamine antagonists oppose it. Molecules which oppose Sorrow (depression) tend to promote PLAY; in this group are agents acting on dopamine, opioid and cannabinoid (Mangieria & Piomelli, 2007) receptors.

Serotonin acts to oppose proponent systems and this may explain its antidepressant, anxiolytic, antianger and sometimes general emotion-blunting effects. Therapeutically, dopamine antagonists oppose mania and hypomania (PLAY & RAGE) and benzodiazepines have a useful opponent function in this regard also via GABA potentiation. Drugs opponent to Sorrow run the risk of triggering hypomania (PLAY extremis). Panksepp (2010) advocates for the utility of the mu-opioid agonist-antagonist buprenorphine in treating depression, while Giovanni and Maremmani (2013) note that naloxone – an opioid (PLAY) antagonist may be antimanic. Methadone induces major mood stabilisation in bipolar I patients (Giovanni & Maremmani, 2013), possibly by treating depression or through non-specific sedation. With respect to the proponentcy for Sorrow, Mineur et al. (2013) recently found that cholinergic signalling may be a factor in the aetiology of depression. Scopolamine, a muscarinic antagonist, was opponent to depression (Sorrow) (Drevets & Furey, 2010). Clearly further results are needed as the action of serotinergic, opioid and muscarinic molecules in humans is complex.

Tension is an important mediator of manic mood escalation in the dynamic model (Figure 29, Figure 34), though as a SMAS it does not have the linearity of ATTACHMENT or HESE. Phenomenologically, FEAR does not relate to ANGER as does DISTRESS to CARE; while the latter are opposed, the former are somehow conjoined or “folded”. TENSION is archaic and a number of the molecules proponent
for each affect program are synergistic, rather than opposed. Exercise, CBT, psychoeducation, meditation and mentalisation may have prophylactic effects for tension in bipolar subjects, though the evidence for the effectiveness of those interventions which target anxiety in BSD’s is modest overall (Stratford, Cooper, Di Simplicio, Blackwell, & Holmes, 2015). In the Triaxial schema (Figure 40) increasing tension drives status states of sorrow and excitement into the clinical conditions – agitated depression, hypomania (sub-mania), mania and mixed states. The use of dopamine antagonists is consistent with reducing RAGE and FEAR in the Panksepp model (Table 18). In the same light, opioids, benzodiazepines and SSRI’s all treat anxiety and are opponent for FEAR. It is no wonder, given their role in SORROW, FEAR and DISTRESS circuitry, that opiates are so ubiquitous in society and prone to dependence. Because mania and mixed states typically involve admixtures of approach, withdrawal and tense arousal, it is important to use medications with multiple sites of action.

Lastly, we should not overlook the role of social factors in the remediation of prestige conditions. Through social projects, we may be able to prevent 25% of depressive cases from occurring (Cuijpers, 2013). In the MOPF research Bipolar I cases were partnered less, had lower incomes, less employment and had longer durations of illness (Table 7). Remediating these factors, through social action, and through improving prestige, self-esteem and tension may go some of the way. Recognising the bipolar disorders as part of a spectrum – which includes normality – may further reduce stigma and promote social inclusion.
Chapter 9: Conclusions

"The weight here of the marketing of bipolar should not be underestimated. As references to it multiply and as studies presuppose its existence as a valid diagnostic category, it becomes equated with an immutable biological entity. More and more people come to see themselves as bipolar, suffering from a 'disorder' that follows its own set of externally classified rules. One of the results of this is that the specificity of each case is lost, as we saw above with the example of the woman who felt discouraged from having another child, as if all cases were the same. The meaning that each individual might give to the events of their life vanishes as they become just another instantiation of an 'illness'". (Leader, 2013, p. 87)

It is compelling – and perhaps common medical sense – to think of bipolar disorder as a unitary medical condition – a molecular accident – afflicting the unfortunate person pacing in the clinic. The reality at hand may be yet more complex and this thesis has sought to explore realms of bipolarity derived from evolution and societal prestige. A concept is an abstract or general idea formed in the mind and ideas about bipolarity have their origins millennia ago with Aretaeus of Cappadocia, whose mind presaged modern models of these conditions. Aretaeus was observing phenomena which had their origin tens or hundreds of thousands of years earlier in the Pleistocene, where social investment – mediated by prestige competition – was a matter of life and death. In this ancient context basic bipolar mutations arose and propagated through human populations, following the great northern and eastern migrations out of Africa. Bipolarity, as broadly defined to include subthreshold forms, appears to be distributed widely in the global population and may be subject to multiple threshold effects. Social investment – our putative driver of bipolarity – is further internalised through the sociometer to self-esteem, which forms part of a homeostatic system. The intentional object of this system may be ingroup membership for the purposes of survival and reproduction. Byproducts such as mania, suicide, and psychosis may have arisen through mechanisms akin to pleiotropy.

Proximal antecedents to bipolar spectrum conditions include childhood relational trauma, which may echo ancestral traumas of social inclusion. A microclimate of life events pertinent to social relationships may act on bipolar individuals to drive escalation of tension through the dynamic cascade. Psychosocial
stress represents a threat to prestige-relational value which may lead to a recapitulation of the mood responses which were selected for in evolutionary time. For unknown reasons either an escalating or de-escalating hedonic-energetic path may ensue. The substrate on which dynamic factors operate is posited to be an hedonic energetic social engagement system, anticipated by Mithen (2005) in his Hmmmmm communication model. Mood syndromes may be usefully mapped onto a three dimensional space drawn from the intersection of prestige approach, prestige withdrawal and tension. The model dismisses the notion that there are two poles to the “bipolar” phenotype, but draws on four arousal systems – the hedonic-energetic, the tense, the agapic and the sexual, each being oriented to approach and withdrawal. Tension appears to drive affective systems from adaptive to byproduct forms.

Yet the individual level of analysis is insufficient, as these reactions are part of a frequency dependent selection. Hypomanic and depressive traits may have competed with euthymia for a resource located on a landscape of prestige – a complex adaptive system. While euthymia predominated in the wider population, the low frequency of elevation and depression may have made the mood states effective competitors. Basic animate dispositions to approach – or withdraw from – social objects intersected with tense arousal to map the mood disorders.

However, are the MOPF theory and data important? They may not be, given their exploratory nature and the “weirdness” of their ideas. Theories which offer an all embracing mechanism for a phenomenon as clinically and aetio logically complex as bipolar disorder should be treated with caution. That said, the theory is simple and coherent and the data remarkably consistent in meeting most predictions and then in supporting the exploratory framework. MOPF represents an attempt to go beyond surface phenomena to fundamental building blocks, much as the RDoC (Cuthbert, 2014) project aims to do. Research-Domain-Criteria-(RDoC) (2012) has chosen to investigate five domains: negative and positive valence, cognitive systems, social processes and arousal/regulatory systems (Cuthbert, 2014). This will incorporate, amongst other dimensions, threat orientation (tense arousal), approach motivation and affiliation (social engagement), along with the perception of self and others. The RDoC methods will include control groups using “liberal inclusion criteria” (p. 33)
and subthreshold *forme fruste* cases, as have been used in this broad prestige spectrum. “Each disorder is seen not as a unitary disease entity, but rather as a particular range within the overall gradient.” (p. 34). These are praiseworthy ambitions, though RDoC (Insel, 2014) runs the risk of being consumed by the *brain*, as chemical imbalances become neural circuits, behavioural disorders become brain disorders and the mind’s view becomes the brain’s view. Brain exists in a system of interacting micro and macro-social objects – psychology is an emergent process, which can never be mapped identically to the brain. Rather than a reductionistic biological focus, I have chosen to emphasise basic biopsychosocial systems which are represented in the emotional Brainmind – a term from Panksepp (2010).

The MOPF research may be tested from a large number of vantage points, which from the outset would entail replication – using standardised diagnostic instruments - to allocate cases to spectrum nodes. Psychometric scales need to be tested and prestige (nlogSIPS) – in particular – needs to be subject to further conceptual and empirical investigation. Some form of 360° estimation of actual societal investment should be generated as a part of this process, whilst the importance of perceived social investment is recognised. The dynamic model has been subject to correlation and path analysis, while the temporal sequence has yet to be determined – perhaps through experience sampling. The nosological model presented has utilised notions of mood level, MSPaM, depression and tension to develop mood disorder categories which would require field testing for validity and reliability. Studies of impairment – or functional gain – and tension in hypomania would add much to conceptualisation around this enigmatic condition. Similarly, testing of the differences between unipolar and pseudounipolar depression would clarify the MOPF edicts. Drawing on Panksepp’s research, Table 18 offers options for empirical validation or refutation for target molecules in specific conditions. We already have information about effective ingredients in psychotherapy, however the role of perceived social investment is nascent. These steps together would add, not only to MOPF theories, but to the wider domain of psychiatric understanding. Yet what of the evolutionary theories entailed in this work – can they be profitably tested – for example that mild hypomania provides a selection advantage through prestige. The research found that prestige approach motivation and prestige were strongly
correlated in hypomania. Replication of this finding could compliment studies of sexual fecundity in hypomania to test common assumptions about selection advantage.

Feynman (2006) has said “The atoms come into my brain, dance a dance, and then go out—there are always new atoms, but always doing the same dance, remembering what the dance was yesterday.” (p. 486). The elucidation of complex biopsychosocial causality in affective disorders is a hard problem for psychiatry – a problem which has lacked theoretical and empirical foundations. I propose that developing a greater understanding of the adaptive drive for social investment – *the dance* – may provide clues for the evolutionary basis of the bipolarity in individuals – *the atoms*. This thesis provides preliminary evidence to support this approach. It is certainly not the first or final word on the matter – as science grows by conjecture and testing – yet it is one attempt to approximate the truth of affective pathogenesis from a novel perspective.
References


THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

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offspring. *Psychological Medicine, 34*(5), pp787-793. doi: 10.1017/S0033291703001491


Appendix

MOPF Survey

May be found at http://tinyurl.com/3rqwfa8

Includes:

1. Plain Language Statement
2. Mood Disorder Questionnaire (MDQ)
3. Presence of Condition Question
4. Illness Impact Questions (impairment scale not used)
5. PHQ-9 Questionnaire
6. ASRM Scale
7. MOPF-EP (SIP) Scale
   Tension Scale
8. MOPF-SPM (MSPaM) Scale
9. MOPF-PR Scale & Others
   MSIS scale
   BIS/BAS scale
   SISE scale
10. Contingencies of Self-worth Scale (abbreviated)
11. Demographics
Plain Language Statement

What is the MOPF questionnaire?
You are invited to take part in MOPF web questionnaire, a part of the principal researcher's PhD project. It measures a number of psychological factors and seeks to clarify the relationship of bipolar disorder to other conditions. It is intended for people with or without a mood disorder. Participants are being recruited from several “collector” sites (which will be recorded) – a hospital & clinic setting and in general practice. The survey will run for up to one year.

Who can do it?
People doing the questionnaire should be 18 to 65 and be able to understand the purpose of the survey. You will need a computer or a paper survey and 15 minutes to spare. The questionnaire should be done promptly, but only once.

What will I be asked?
There are a number of questions about your mood, your illness, your self-esteem and your life. Doing it is voluntary. Please don’t feel you need to indicate to your health professional whether you have done it or not.

Why do the questionnaire?
Some people find it interesting to be asked about matters to do with their life. We hope that analysis of the survey sheds light on the causes of mental illnesses.

How can I give my consent?
You have been asked to give consent to do this survey by the health professional who gave you this invitation. By completing the survey you are freely consenting to provide personal and health information, which will be recorded anonymously. The information you provide will be treated confidentially.

What if I have any questions or concerns after the survey?
Doing surveys can lead people to reflect on their current situation. If you have concerns then please discuss these with your health professional, or the no-cost survey counsellor, available on the number at the end of this document. Alternatively, the researchers may be contacted on the same phone number or e-mail. Alternately, if you have any concerns about the conduct of this research project, you can contact the Executive Officer, Human Research Ethics, The University of Melbourne, ph: 8344 2073; fax 9347 6739 or The Convenor, Peninsula Health Human Research Ethics Committee ph 9788 1474.

Security and Privacy at SurveyMonkey:
SurveyMonkey is a free public online survey tool which operates from a high security site with data protection, including SSL technology (a protocol for transporting documents on the Internet confidentially). It stores your IP address (though the researchers do not), as all web computers do, and uses this information for its administration. SurveyMonkey allocates cookies when you log on. These may be disabled in your browser program if you are concerned. Your survey responses are kept in strict confidence and used in compliance with legal requirements. The researchers remain the controllers and owners of the data collected.

Where is the information stored?
SurveyMonkey offers high levels of privacy and security. The information will be downloaded securely to the researcher’s PC without any data that could identify you. Because it is anonymous, you will not be able to get any personal summary feedback on your responses, but the research findings may be obtained by emailing the researchers (though publications may take several years). The information will be kept for fifteen years.
How do I do it?
By entering the following link into your web browser: www.surveymonkey.com/s/mopf5ph
or by filling in a paper questionnaire

Lastly, thank you for doing the MOPF questionnaire. We thank you for the part you are playing.

Dr James Le Bas FRANZCP (PhD candidate)
Peninsula Health Mental Health Service
(03) 9784 6999
surveymopf@gmail.com
Professor David Castle MD FRANZCP
University of Melbourne
Department of Psychiatry

Ethics approved Melbourne University & Peninsula Health December 2011: PLS version 2.5
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

Prestige Approach Motivation (MSPaM)

Answered on a seven point Likert scale:

<table>
<thead>
<tr>
<th>Disagree strongly</th>
<th>Disagree somewhat</th>
<th>Neutral</th>
<th>Agree somewhat</th>
<th>Agree</th>
<th>Strongly agree</th>
</tr>
</thead>
</table>

1. If I come under scrutiny I feel uneasy so I strengthen my supports
2. When I struggle for recognition I wake early and dwell on righting things
3. To keep self respect I need to be included
4. I try to make myself attractive to hold people's attention
5. When people won't budge I am determined to win them over
6. I work hard to maintain my reputation
7. I dream that my achievements will be valued
8. I use the information I have to hold people's attention
9. It is important for people to hold me in good esteem
10. I feel over the moon when people value my efforts
11. I feel deflated when people won't acknowledge my contribution
12. Setbacks push me to get back in the good books with people
13. I would like to impress people with my new possessions or creations
14. People respect me more when I achieve my goals
15. I get more inventive when I have to fight for good standing
16. My good standing in the group is often under threat
17. I often think about things I have said which may have impacted on others
18. My confidence crashes when people don't support me
19. If my self-esteem is hurt I become determined to fix the problem
20. People only value me when I emphasise my good qualities
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

Diagnostic Algorithm

MDQ category

If the MDQ responses (MDQ 1.1) occurred in the same time period (MDQ 2.2) AND they caused a moderate or severe problem (MDQ 3.3) then the MDQ category is equivalent to the MDQ threshold rating of 0 or 1. If the responses did not occur in the same period OR were "minor" or "no problem", despite the MDQ threshold rating, the MDQcat is 0. (Hirschfeld et al., 2000)

Bipolar Quotient

The following are summed:

• If told you had bipolar illness by a health professional (0,1)
• If you volunteered that you had bipolar illness (0,1)
• The MDQ category (0,2)

If the sum was 2 or more the person is allocated a positive bipolar quotient.

Diagnosis algorithm (hierarchical)

1. If the diagnosis you allocated yourself was bipolar disorder, the recorded diagnosis is "bipolar disorder"
2. If your bipolar quotient is positive, the recorded diagnosis is "bipolar disorder"
3. If you have scored 10 or above on the depression inventory (PHQ-9), your diagnosis is “depression”
4. If you allocated yourself no diagnosis, your diagnosis is "no diagnosis"
5. If you allocated yourself a diagnosis of depression, your diagnosis is "depression"
6. If you allocated yourself a diagnosis of "other psychiatric condition", your diagnosis is "other psychiatric condition"
7. If none of these apply the cell is empty

Polarity estimation

If the diagnosis is bipolar disorder and the person has been hospitalised or admitted to psychosis, the result is 1(bipolar I disorder).

If the diagnosis is bipolar disorder and the person has not been hospitalised or has admitted to psychosis the result is 2 (bipolar II disorder).

If neither applies the result is 0.
Spectrum Calculator (hierarchical)

1. If diagnosis is empty, cell is empty

2. If polarity is 1, spectrum is bipolar I (S1)

3. If polarity is 2, spectrum is bipolar II (S2)

4. If there is a diagnosis of depression and a positive family history of bipolar disorder, the spectrum is pseudounipolar (S3)

5. If there is a declared family history of bipolar disorder and the diagnosis is of "no diagnosis", the spectrum is simplex (S5)

6. If the diagnosis is depression, the spectrum is unipolar depression (S6)

7. If none of the above apply and the individual has a suprathreshold (ASRM) (hypo)mania score OR an MDQ score between 3 and 6, then the spectrum is subthreshold (S4)

8. If there is neither a diagnosis, a declared family history of bipolar disorder, or subthreshold condition, the spectrum is control (S7)

9. If diagnosis is simply "other psychiatric disorder" the spectrum cell is empty and the response is deleted

10. If none of these conditions apply the spectrum cell is empty and the response is deleted
## Tables and Figures

### Table 19: Descriptive Statistics for Numerical Variables (Scales)

<table>
<thead>
<tr>
<th>Scale</th>
<th>N</th>
<th>Mean ((M))</th>
<th>Standard Deviation ((SD))</th>
<th>Minimum</th>
<th>Median</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>nlogSIPS</td>
<td>225</td>
<td>3.19</td>
<td>0.85</td>
<td>1.50</td>
<td>3.16</td>
<td>4.68</td>
</tr>
<tr>
<td>TENSION</td>
<td>222</td>
<td>4.29</td>
<td>1.94</td>
<td>1.00</td>
<td>4.50</td>
<td>7.00</td>
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<tr>
<td>MSPaM</td>
<td>227</td>
<td>77.95</td>
<td>21.37</td>
<td>20</td>
<td>81</td>
<td>125</td>
</tr>
<tr>
<td>MDQ</td>
<td>228</td>
<td>6.12</td>
<td>4.14</td>
<td>0.00</td>
<td>6.00</td>
<td>13.00</td>
</tr>
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<td>ASRM</td>
<td>224</td>
<td>2.71</td>
<td>3.03</td>
<td>0.00</td>
<td>2.00</td>
<td>15.00</td>
</tr>
<tr>
<td>PHQ-9</td>
<td>228</td>
<td>8.26</td>
<td>8.16</td>
<td>0.00</td>
<td>5.00</td>
<td>27.00</td>
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<td>MSIS</td>
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<td>5.47</td>
<td>10.00</td>
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<td>35.00</td>
</tr>
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<td>CSW.av</td>
<td>225</td>
<td>4.15</td>
<td>1.03</td>
<td>1.73</td>
<td>4.07</td>
<td>7.00</td>
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Table 20  Descriptive Statistics for Ordinal and Nominal Variables (Scales)

<table>
<thead>
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<th>Measure</th>
<th>Level</th>
<th>Description</th>
<th>N</th>
<th>%</th>
<th>Cum%</th>
</tr>
</thead>
<tbody>
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<td></td>
<td>45</td>
<td>20.55</td>
<td>20.55</td>
</tr>
<tr>
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<td>19.18</td>
<td>39.73</td>
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<td>64.84</td>
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<td>4</td>
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<td>57</td>
<td>26.03</td>
<td>90.87</td>
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<td>5</td>
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<td>20</td>
<td>9.13</td>
<td>100</td>
</tr>
<tr>
<td>MOODcat</td>
<td>0</td>
<td>Euthymic</td>
<td>90</td>
<td>40.00</td>
<td>40.00</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>(Hypo)manic</td>
<td>23</td>
<td>10.22</td>
<td>50.22</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Depressed</td>
<td>65</td>
<td>28.89</td>
<td>79.11</td>
</tr>
<tr>
<td></td>
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<td>“Mixed”</td>
<td>9</td>
<td>4.00</td>
<td>83.11</td>
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<tr>
<td></td>
<td>4</td>
<td>Mild depression</td>
<td>38</td>
<td>16.89</td>
<td>100.00</td>
</tr>
</tbody>
</table>

*Note.* MOODcat is a nominal variable
### Table 21
Simple and Multiple Regression Analyses Examining Self-Esteem (SISE) as a Mediator When Modelling TENSION using Prestige (nlogSIPS)

<table>
<thead>
<tr>
<th>Path</th>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>Estimate</th>
<th>SE</th>
<th>Standardized</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>nlogSIPS</td>
<td>SISE</td>
<td>0.50</td>
<td>0.10</td>
<td>0.34</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Model: TENSION = nlogSIPS

| C    | nlogSIPS             | TENSION            | -0.57    | 0.15| -0.25        | <.001   |

Model: TENSION = SISE + nlogSIPS

| B    | SISE                 | TENSION            | -0.65    | 0.10| -0.43        | <.001   |
| C'   | nlogSIPS             | TENSION            | -0.21    | 0.14| -0.09        | .143    |

*Note.* The Sobel z-value for mediation was -3.96 (*p* < .001).
Table 22  Simple and Multiple Regression Analyses Examining TENSION as a Mediator When Modelling MSPaM using Self-Esteem (SISE)

<table>
<thead>
<tr>
<th>Path</th>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>Estimate</th>
<th>SE</th>
<th>Standardized</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>SISE</td>
<td>TENSION</td>
<td>-0.70</td>
<td>0.09</td>
<td>-0.46</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Model: MSPaM = SISE

| C    | SISE                 | MSPaM              | -3.75    | 1.11 | -0.22        | .001    |

Model: MSPaM = TENSION + SISE

| B    | TENSION              | MSPaM              | 4.97     | 0.76 | 0.45         | <.001   |
| C'   | SISE                 | MSPaM              | -0.54    | 1.15 | -0.03        | .639    |

Note. The Sobel z-value for mediation was -5.01 (p <.001).
Table 23: Simple and Multiple Regression Analyses Examining TENSION as a Mediator When Modelling Depression (nlogPHQ-9) using Self-Esteem (SISE)

<table>
<thead>
<tr>
<th>Path</th>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>Estimate</th>
<th>SE</th>
<th>Standardized</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>SISE</td>
<td>TENSION</td>
<td>-0.70</td>
<td>0.09</td>
<td>-0.46</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Model: nlogPHQ-9 = SISE

| C    | SISE                 | nlogPHQ-9          | -0.38    | 0.05| -0.47        | <.001   |

Model: nlogPHQ-9 = TENSION + SISE

| B    | TENSION              | nlogPHQ-9          | 0.30     | 0.03| 0.56         | <.001   |
| C'   | SISE                 | nlogPHQ-9          | -0.20    | 0.05| -0.24        | <.001   |

Note. The Sobel z-value for mediation was -6.14 (p < .001).
Table 24  Simple and Multiple Regression Analyses Examining Depression (nlogPHQ-9) as a Mediator When Modelling Prestige (nlogSIPS) using TENSION

<table>
<thead>
<tr>
<th>Path</th>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>Estimate</th>
<th>SE</th>
<th>Standardized</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>TENSION</td>
<td>nlogPHQ-9</td>
<td>0.36</td>
<td>0.03</td>
<td>0.69</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Model: nlogSIPS = TENSION

| C    | TENSION              | nlogSIPS           | -0.11    | 0.03 | -0.25        | <.001   |

Model: nlogSIPS = nlogPHQ-9 + TENSION

| B    | nlogPHQ-9            | nlogSIPS           | -0.26    | 0.08 | -0.31        | .001    |
| C'   | TENSION              | nlogSIPS           | -0.02    | 0.04 | -0.04        | .690    |

Note. The Sobel z-value for mediation was -3.14 (p = .002).
### Table 25  Regression Analysis of Tension*ASRM into Prestige (nlogSIPS)  
(Approach Group, n = 110)

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>Coef. (raw)</th>
<th>SE Coef.</th>
<th>T value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONSTANT</td>
<td>3.71</td>
<td>0.19</td>
<td>19.77</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>TENSION</td>
<td>-0.13</td>
<td>0.06</td>
<td>-2.21</td>
<td>.029</td>
</tr>
<tr>
<td>ASRM</td>
<td>-0.02</td>
<td>0.04</td>
<td>-0.48</td>
<td>.629</td>
</tr>
<tr>
<td>TENSION*ASRM</td>
<td>0.03</td>
<td>0.01</td>
<td>2.24</td>
<td>.027</td>
</tr>
</tbody>
</table>

*Note.* * indicates an interaction term.

### Table 26  Regression Analysis of MSPaM*ASRM into Prestige (nlogSIPS)  
(Approach Group, n = 110)

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>Coef. (raw)</th>
<th>SE Coef.</th>
<th>T-value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONSTANT</td>
<td>3.88</td>
<td>0.27</td>
<td>14.16</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>MSPaM</td>
<td>-0.01</td>
<td>0.00</td>
<td>-1.98</td>
<td>.051</td>
</tr>
<tr>
<td>ASRM</td>
<td>-0.10</td>
<td>0.06</td>
<td>-1.62</td>
<td>.109</td>
</tr>
<tr>
<td>MSPaM*ASRM</td>
<td>0.00</td>
<td>0.00</td>
<td>2.69</td>
<td>.008</td>
</tr>
</tbody>
</table>

*Note.* * indicates an interaction term.
Figure 41. Confidence intervals for depression (PHQ-9) score by spectrum node. PsUni = pseudo-unipolar, SubTh = subthreshold spectrum node.
**Table 27** Simple and Multiple Regression Analyses Examining Prestige approach motivation (MSPaM) as a Mediator When Modelling Prestige (nlogSIPS) using TENSION. (Hypo)manic Subset of Cohort (n=23)

<table>
<thead>
<tr>
<th>Path</th>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>Estimate</th>
<th>SE</th>
<th>Standardized</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>TENSION</td>
<td>MSPaM</td>
<td>5.35</td>
<td>2.19</td>
<td>0.48</td>
<td>.024</td>
</tr>
</tbody>
</table>

Model: nlogSIPS = TENSION

| C    | TENSION | nlogSIPS | 0.08 | 0.06 | 0.28         | .212    |

Model: nlogSIPS = MSPaM + TENSION

| B    | MSPaM | nlogSIPS | 0.01 | 0.01 | 0.48         | .044    |
| C'   | TENSION | nlogSIPS | 0.01 | 0.07 | 0.04         | .844    |

*Note.* The Sobel z-value for mediation was 0.93 (*p* = .355).
### Table 28 Simple and Multiple Regression Analyses Examining Contingent Self-Worth (CSW.av) as a Mediator When Modelling Self-Esteem (SISE) using Prestige (nlogSIPS)

<table>
<thead>
<tr>
<th>Path</th>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>Estimate</th>
<th>SE</th>
<th>Standardized</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>nlogSIPS</td>
<td>CSW.av</td>
<td>-0.15</td>
<td>0.08</td>
<td>-0.13</td>
<td>.062</td>
</tr>
</tbody>
</table>

Model: $\text{SISE} = \text{nlogSIPS}$

| C    | nlogSIPS             | SISE               | 0.50     | 0.10| 0.34         | <.001   |

Model: $\text{SISE} = \text{CSW.av} + \text{nlogSIPS}$

| B    | CSW.av               | SISE               | -0.52    | 0.07| -0.42        | <.001   |
| C'   | nlogSIPS             | SISE               | 0.42     | 0.09| 0.28         | <.001   |

*Note.* The Sobel z-value for CSW.av mediation was -1.81 ($p = .070$).
### Table 29: Simple and Multiple Regression Analyses Examining Contingent Self-Worth (CSW.av) as a Mediator When Modelling TENSION using Self-Esteem (SISE)

<table>
<thead>
<tr>
<th>Path</th>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>Estimate</th>
<th>SE</th>
<th>Standardized</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>SISE</td>
<td>CSW.av</td>
<td>-0.38</td>
<td>0.05</td>
<td>-0.47</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Model: TENSION = SISE

| C    | SISE                 | TENSION            | -0.70    | 0.09| -0.46        | <.001   |

Model: TENSION = CSW.av + SISE

| B    | CSW.av               | TENSION            | 0.49     | 0.12| 0.26         | <.001   |
| C’   | SISE                 | TENSION            | -0.50    | 0.10| -0.33        | <.001   |

*Note.* The Sobel z-value for CSW.av mediation was -3.60 (*p* < .001).
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

Table 30 Simple and Multiple Regression Analyses Examining Social Inclusion Sensitivity (MSIS) as a Mediator and Moderator When Modelling Prestige approach motivation (MSPaM) using TENSION

<table>
<thead>
<tr>
<th>Path</th>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>Estimate</th>
<th>SE</th>
<th>Standardized</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>TENSION</td>
<td>MSIS</td>
<td>1.07</td>
<td>0.18</td>
<td>0.38</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
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</tr>
</tbody>
</table>

Model: MSPaM = TENSION

| C    | TENSION              | MSPaM              | 5.16     | 0.66| 0.47         | <.001   |
|      |                      |                    |          |     |              |         |

Model: MSPaM = MSIS + TENSION

| B    | MSIS                 | MSPaM              | 2.38     | 0.20| 0.61         | <.001   |
| C’   | TENSION              | MSPaM              | 2.76     | 0.57| 0.25         | <.001   |

INT. TENSION* MSIS

|      | TENSION* MSIS        | MSPaM              | -0.22    | 0.10| .024         |         |

Note. The Sobel z-value for mediation was 5.32 (p < .001). The significant p value for TENSION*MSIS (INT. regression) indicated a moderating effect for MSIS in the TENSION into MSPaM relationship.
### Table 31  Simple and Multiple Regression Analyses Examining Contingent Self-Worth (CSW.av) as a Mediator When Modelling Prestige approach motivation (MSPaM) using TENSION

<table>
<thead>
<tr>
<th>Path</th>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>Estimate</th>
<th>SE</th>
<th>Standardized</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>TENSION</td>
<td>CSW.av</td>
<td>0.22</td>
<td>0.03</td>
<td>0.42</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Model: MSPaM = TENSION</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>TENSION</td>
<td>MSPaM</td>
<td>5.16</td>
<td>0.66</td>
<td>0.47</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model: MSPaM = CSW.av + TENSION</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>CSW.av</td>
<td>MSPaM</td>
<td>9.85</td>
<td>1.20</td>
<td>0.47</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C'</td>
<td>TENSION</td>
<td>MSPaM</td>
<td>3.01</td>
<td>0.64</td>
<td>0.27</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

*Note.* The Sobel z-value for mediation was 5.47 (*p* < .001).
Table 32  Simple and Multiple Regression Analyses Examining Contingent Self-Worth (CSW.av) as a Mediator and Moderator When Modelling Depression (nlogPHQ-9) using TENSION

<table>
<thead>
<tr>
<th>Path</th>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>Estimate</th>
<th>SE</th>
<th>Standardized</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>TENSION</td>
<td>CSW.av</td>
<td>0.22</td>
<td>0.03</td>
<td>0.42</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>CSW.av</td>
<td>nlogPHQ-9</td>
<td>0.08</td>
<td>0.06</td>
<td>0.08</td>
<td>.160</td>
</tr>
<tr>
<td>C'</td>
<td>TENSION</td>
<td>nlogPHQ-9</td>
<td>0.35</td>
<td>0.03</td>
<td>0.67</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>INT.</td>
<td>TENSION* CSW.av</td>
<td>nlogPHQ-9</td>
<td>0.07</td>
<td>0.03</td>
<td>.025</td>
<td></td>
</tr>
</tbody>
</table>

Note. The Sobel z-value for CSW.av mediation was 1.31 (p = .190). The significant p value for TENSION*CSW.av (INT. regression) indicated a moderating effect for CSW.av in the TENSION into depression (nlogPHQ-9) relationship.
Table 33  Simple and Multiple Regression Analyses Examining MDQ score as a Mediator When Modelling Self-Esteem (SISE) using Prestige (nlogSIPS)

<table>
<thead>
<tr>
<th>Path</th>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>Estimate</th>
<th>SE</th>
<th>Standardized</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>nlogSIPS</td>
<td>MDQ</td>
<td>-0.73</td>
<td>0.32</td>
<td>-0.15</td>
<td>.024</td>
</tr>
</tbody>
</table>

Model: SISE = nlogSIPS

| C    | nlogSIPS             | SISE               | 0.50     | 0.10| 0.34         | <.001   |

Model: SISE = MDQ + nlogSIPS

| B    | MDQ                  | SISE               | -0.09    | 0.02| -0.29        | <.001   |
| C'   | nlogSIPS             | SISE               | 0.43     | 0.09| 0.29         | <.001   |

*Note.* The Sobel z-value for MDQ mediation was 2.03 (p = .04).
### Table 34  Simple and Multiple Regression Analyses Examining MDQ score as a Mediator When Modelling TENSION using Self-Esteem (SISE)

<table>
<thead>
<tr>
<th>Path</th>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>Estimate</th>
<th>SE</th>
<th>Standardized</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>SISE</td>
<td>MDQ</td>
<td>-1.05</td>
<td>0.21</td>
<td>-0.32</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Model: TENSION = SISE

| C    | SISE                 | TENSION            | -0.70    | 0.09| -0.46        | <.001   |

Model: TENSION = MDQ + SISE

| B    | MDQ                  | TENSION            | 0.10     | 0.03| 0.20         | .001    |
| C’   | SISE                 | TENSION            | -0.60    | 0.03| -0.39        | <.001   |

*Note. The Sobel z-value for MDQ mediation was -2.77 (p = .006).*
### Table 35  Simple and Multiple Regression Analyses Examining MDQ score as a Mediator When Modelling Prestige approach motivation (MSPaM) using TENSION

<table>
<thead>
<tr>
<th>Path</th>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>Estimate</th>
<th>SE</th>
<th>Standardized</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>TENSION</td>
<td>MDQ</td>
<td>0.70</td>
<td>0.14</td>
<td>0.33</td>
<td>&lt;.001</td>
</tr>
<tr>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>5.16</td>
<td>0.66</td>
<td>0.47</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>C</td>
<td>TENSION</td>
<td>MSPaM</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3.92</td>
<td>0.65</td>
<td>0.36</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>C'</td>
<td>TENSION</td>
<td>MSPaM</td>
<td></td>
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<tr>
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</tr>
</tbody>
</table>

Note. The Sobel z-value for MDQ mediation was 3.75 ($p < .001$).
Table 36  Simple and Multiple Regression Analyses Examining MDQ Score as a Mediator When Modelling Depression (nlogPHQ-9) using TENSION

<table>
<thead>
<tr>
<th>Path</th>
<th>Independent variable</th>
<th>Dependent variable</th>
<th>Estimate</th>
<th>SE</th>
<th>Standardized</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>TENSION</td>
<td>MDQ</td>
<td>0.70</td>
<td>0.14</td>
<td>0.33</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Model: nlogPHQ-9 = TENSION

| C    | TENSION             | nlogPHQ-9          | 0.36     | 0.03| 0.69         | <.001   |

Model: nlogPHQ-9 = MDQ + TENSION

| B    | MDQ                 | nlogPHQ-9          | 0.08     | 0.01| 0.34         | <.001   |
| C'   | TENSION             | nlogPHQ-9          | 0.30     | 0.03| 0.58         | <.001   |

*Note.* The Sobel z-value for MDQ mediation was 4.24 (*p* < .001).
# THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

Table 37  Descriptive Statistics for MSPaM/Prestige Ratio

<table>
<thead>
<tr>
<th>Mood State</th>
<th>n</th>
<th>Mean</th>
<th>Standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euthymia (0)</td>
<td>88</td>
<td>21.85</td>
<td>12.35</td>
</tr>
<tr>
<td>(Hypo)mania (1)</td>
<td>22</td>
<td>20.27</td>
<td>12.35</td>
</tr>
<tr>
<td>Depression (2)</td>
<td>65</td>
<td>32.59</td>
<td>12.35</td>
</tr>
<tr>
<td>“Mixed” (3)</td>
<td>9</td>
<td>42.93</td>
<td>12.35</td>
</tr>
<tr>
<td>Mild Depression (4)</td>
<td>38</td>
<td>30.85</td>
<td>12.35</td>
</tr>
</tbody>
</table>

Table 38  Inferential Statistics for ANOVA MSPaM/Prestige Ratio

<table>
<thead>
<tr>
<th>Mood State</th>
<th>Estimate</th>
<th>95% Confidence Interval</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Hypo)mania minus Euthymia</td>
<td>-1.57</td>
<td>[-9.67, 6.52]</td>
<td>.984</td>
</tr>
<tr>
<td>Depression minus Euthymia</td>
<td>10.74</td>
<td>[5.18, 16.29]</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>“Mixed” minus Euthymia</td>
<td>21.08</td>
<td>[9.19, 32.97]</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mild Depression minus Euthymia</td>
<td>9.00</td>
<td>[2.41, 15.60]</td>
<td>.002</td>
</tr>
</tbody>
</table>
Table 39  Relationship between Family History of Bipolar Disorder and Perceived Childhood Relational Trauma (PCRT)

<table>
<thead>
<tr>
<th>PCRT</th>
<th>Family History of Bipolar Disorder</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Negative</td>
<td>Positive</td>
</tr>
<tr>
<td>Negative</td>
<td>96</td>
<td>33</td>
</tr>
<tr>
<td>Expected</td>
<td>83</td>
<td>46</td>
</tr>
<tr>
<td>Positive</td>
<td>20</td>
<td>32</td>
</tr>
<tr>
<td>Expected</td>
<td>33</td>
<td>19</td>
</tr>
<tr>
<td>Total</td>
<td>116</td>
<td>65</td>
</tr>
</tbody>
</table>

Note. PCRT = Perceived Childhood Relational Trauma

$\chi^2 (1, N = 181) = 20.82, \text{ DF } = 1, p < .001.$
Table 40  Relationship Between PCRT and Proband Diagnosis

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>PCRT</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Diagnosis</td>
<td>Negative</td>
<td>Positive</td>
<td>Total</td>
</tr>
<tr>
<td>Nil diagnosis</td>
<td></td>
<td>64</td>
<td>3</td>
<td>67</td>
</tr>
<tr>
<td>Expected</td>
<td></td>
<td>47.50</td>
<td>19.50</td>
<td></td>
</tr>
<tr>
<td>Bipolar disorder</td>
<td></td>
<td>35</td>
<td>38</td>
<td>73</td>
</tr>
<tr>
<td>Expected</td>
<td></td>
<td>51.76</td>
<td>21.24</td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td>35</td>
<td>14</td>
<td>49</td>
</tr>
<tr>
<td>Expected</td>
<td></td>
<td>34.74</td>
<td>14.26</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>134</td>
<td>55</td>
<td>189</td>
</tr>
</tbody>
</table>

Note. PCRT = Perceived Childhood Relational Trauma. The “other diagnosis” group was not included (n=2) to expedite the \( \chi^2 \) analysis: \( \chi^2 \) statistic (2, N =189) = 38.34, \( p <.001 \).
Table 41  Comparison of Diagnostic and Spectrum Allocation for Fidelity

<table>
<thead>
<tr>
<th>Diagnosis/ Spectrum</th>
<th>S1</th>
<th>S2</th>
<th>S3</th>
<th>S4</th>
<th>S5</th>
<th>S6</th>
<th>S7</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td>No diagnosis</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>30</td>
<td>17</td>
<td>0</td>
<td>48</td>
<td>95</td>
</tr>
<tr>
<td>Bipolar disorder</td>
<td>53</td>
<td>24</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>77</td>
</tr>
<tr>
<td>Depression</td>
<td>0</td>
<td>0</td>
<td>17</td>
<td>0</td>
<td>0</td>
<td>37</td>
<td>0</td>
<td>54</td>
</tr>
<tr>
<td>Other diagnosis</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>All</td>
<td>53</td>
<td>24</td>
<td>17</td>
<td>32</td>
<td>17</td>
<td>37</td>
<td>48</td>
<td>228</td>
</tr>
</tbody>
</table>
Table 42  Chi-square Result for Self-esteem by Mood Category (Euthymia, (Hypo)mania, Depression)

<table>
<thead>
<tr>
<th>Self-esteem</th>
<th>Euthymia</th>
<th>(Hypo)mania</th>
<th>Depression</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3</td>
<td>1</td>
<td>30</td>
<td>34</td>
</tr>
<tr>
<td>Expected</td>
<td>17.3</td>
<td>4.37</td>
<td>12.33</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>15</td>
<td>1</td>
<td>10</td>
<td>26</td>
</tr>
<tr>
<td>Expected</td>
<td>13.23</td>
<td>3.35</td>
<td>9.43</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>24</td>
<td>9</td>
<td>12</td>
<td>45</td>
</tr>
<tr>
<td>Expected</td>
<td>22.89</td>
<td>5.79</td>
<td>16.32</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>33</td>
<td>8</td>
<td>8</td>
<td>49</td>
</tr>
<tr>
<td>Expected</td>
<td>24.93</td>
<td>6.30</td>
<td>17.77</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>12</td>
<td>3</td>
<td>2</td>
<td>17</td>
</tr>
<tr>
<td>Expected</td>
<td>8.65</td>
<td>2.19</td>
<td>6.12</td>
<td></td>
</tr>
<tr>
<td>Totals</td>
<td>87</td>
<td>22</td>
<td>62</td>
<td>171</td>
</tr>
</tbody>
</table>

Note. The mixed group has been excluded from this analysis due to low numbers (9). 5 of 15 cells contain less than 5 responses, making the χ² finding contestable.
### Table 43  Descriptive Statistics for Social Inclusion Sensitivity (MSIS) by Mood State

<table>
<thead>
<tr>
<th>Mood State</th>
<th>n</th>
<th>Mean</th>
<th>Standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euthymic</td>
<td>88</td>
<td>22.22</td>
<td>5.12</td>
</tr>
<tr>
<td>(Hypo)manic</td>
<td>22</td>
<td>26.23</td>
<td>5.12</td>
</tr>
<tr>
<td>Depressed</td>
<td>62</td>
<td>25.85</td>
<td>5.12</td>
</tr>
<tr>
<td>“Mixed”</td>
<td>8</td>
<td>28.75</td>
<td>5.12</td>
</tr>
<tr>
<td>Mild depression</td>
<td>37</td>
<td>24.08</td>
<td>5.12</td>
</tr>
</tbody>
</table>

### Table 44  Inferential Statistics for Social Inclusion Sensitivity (MSIS) by Mood State

<table>
<thead>
<tr>
<th>Spectrum</th>
<th>Estimate</th>
<th>95% Confidence Interval</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Hypo)manic -Euthymic</td>
<td>4.01</td>
<td>[0.65, 7.37]</td>
<td>.011</td>
</tr>
<tr>
<td>Depressed-Euthymic</td>
<td>3.63</td>
<td>[1.30, 5.97]</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>“Mixed”-Euthymic</td>
<td>6.53</td>
<td>[1.32, 11.73]</td>
<td>.006</td>
</tr>
<tr>
<td>Mild depression-Euthymic</td>
<td>1.86</td>
<td>[-0.90, 4.62]</td>
<td>.347</td>
</tr>
</tbody>
</table>
### Table 45  Descriptive Statistics for Social Inclusion Sensitivity (MSIS)

<table>
<thead>
<tr>
<th>Spectrum</th>
<th>n</th>
<th>Mean</th>
<th>Standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1</td>
<td>50</td>
<td>26.46</td>
<td>4.56</td>
</tr>
<tr>
<td>S2</td>
<td>23</td>
<td>25.17</td>
<td>5.02</td>
</tr>
<tr>
<td>S3</td>
<td>16</td>
<td>25.94</td>
<td>5.23</td>
</tr>
<tr>
<td>S4</td>
<td>32</td>
<td>24.88</td>
<td>4.86</td>
</tr>
<tr>
<td>S5</td>
<td>17</td>
<td>23.59</td>
<td>3.41</td>
</tr>
<tr>
<td>S6</td>
<td>35</td>
<td>24.34</td>
<td>5.84</td>
</tr>
<tr>
<td>S7</td>
<td>47</td>
<td>20.05</td>
<td>5.41</td>
</tr>
</tbody>
</table>

### Table 46  Inferential Statistics for Social Inclusion Sensitivity (MSIS)

<table>
<thead>
<tr>
<th>Spectrum</th>
<th>Estimate</th>
<th>95% confidence interval</th>
<th>P value</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1 minus S7</td>
<td>4.68</td>
<td>[1.13, 8.23]</td>
<td>.002</td>
<td>0.87</td>
</tr>
<tr>
<td>S2 minus S7</td>
<td>3.84</td>
<td>[-0.40, 8.08]</td>
<td>.10</td>
<td>0.73</td>
</tr>
<tr>
<td>S3 minus S7</td>
<td>4.29</td>
<td>[-0.33, 8.90]</td>
<td>.09</td>
<td>0.82</td>
</tr>
<tr>
<td>S4 minus S7</td>
<td>3.60</td>
<td>[0.05, 7.15]</td>
<td>.04</td>
<td>0.67</td>
</tr>
<tr>
<td>S5 minus S7</td>
<td>2.51</td>
<td>[-1.72, 6.74]</td>
<td>.57</td>
<td>0.48</td>
</tr>
<tr>
<td>S6 minus S7</td>
<td>3.03</td>
<td>[-0.48, 6.53]</td>
<td>.14</td>
<td>0.58</td>
</tr>
</tbody>
</table>

*Note. Controlled for depression (PHQ9) and mood elevation (ASRM).*
**THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY**

Table 47  Descriptive Statistics Prestige Scale (nlogSIPS)

<table>
<thead>
<tr>
<th>Mood category</th>
<th>n</th>
<th>Mean</th>
<th>Standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euthymic (0)</td>
<td>89</td>
<td>3.42</td>
<td>0.71</td>
</tr>
<tr>
<td>(Hypo)manic (1)</td>
<td>22</td>
<td>3.98</td>
<td>0.54</td>
</tr>
<tr>
<td>Depressed (2)</td>
<td>65</td>
<td>2.90</td>
<td>0.83</td>
</tr>
<tr>
<td>“Mixed” (3)</td>
<td>9</td>
<td>2.59</td>
<td>0.92</td>
</tr>
<tr>
<td>Mild depression (4)</td>
<td>38</td>
<td>2.83</td>
<td>0.81</td>
</tr>
</tbody>
</table>

Table 48  Inferential Statistics for Prestige Scale (nlogSIPS) by Mood State

<table>
<thead>
<tr>
<th>Mood state difference</th>
<th>Estimate</th>
<th>95% confidence interval</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Hypo)manic – euthymic</td>
<td>0.56</td>
<td>[0.06, 1.06]</td>
<td>.019</td>
</tr>
<tr>
<td>Depressed – euthymic</td>
<td>-0.52</td>
<td>[-0.86, -0.18]</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Mixed – euthymic</td>
<td>-0.83</td>
<td>[-1.56, -0.10]</td>
<td>.018</td>
</tr>
<tr>
<td>Mild depression – euthymic</td>
<td>-0.59</td>
<td>[-0.99, -0.18]</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>
### Table 49  Descriptive Statistics for Prestige Approach Motivation (MSPaM)

<table>
<thead>
<tr>
<th>Mood category</th>
<th>n</th>
<th>Mean</th>
<th>Standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euthymic (0)</td>
<td>89</td>
<td>69.80</td>
<td>21.65</td>
</tr>
<tr>
<td>Hypomanic (1)</td>
<td>23</td>
<td>81.39</td>
<td>20.56</td>
</tr>
<tr>
<td>Depressed (2)</td>
<td>65</td>
<td>86.13</td>
<td>18.05</td>
</tr>
<tr>
<td>Mixed (3)</td>
<td>9</td>
<td>96.14</td>
<td>18.3</td>
</tr>
<tr>
<td>Mild depression (4)</td>
<td>38</td>
<td>78.74</td>
<td>18.63</td>
</tr>
</tbody>
</table>
### Inferential Statistics for Prestige Approach Motivation (MSPaM)

<table>
<thead>
<tr>
<th>Mood state difference</th>
<th>Estimate</th>
<th>95% confidence interval</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypomanic – euthymic</td>
<td>11.59</td>
<td>[-1.22, 24.41]</td>
<td>.097</td>
</tr>
<tr>
<td>Depressed – euthymic</td>
<td>16.33</td>
<td>[7.39, 25.27]</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Mild depression – euthymic</td>
<td>8.94</td>
<td>[-1.68, 19.56]</td>
<td>.144</td>
</tr>
</tbody>
</table>
Figure 42. Scatterplot of spectrum population in mood state groups on landscape of prestige approach motivation (MSPaM) by prestige (nlogSIPS). Reference lines are drawn at the median for each axis – prestige 3.16, MSPaM 81.00 (jitter applied).
Figure 43. Prestige approach motivation (MSPaM) vs prestige (nlogSIPS) indicated by spectrum group (jitter applied). Reference lines are drawn at the median for each axis – prestige 3.16, MSPaM 81.00 (jitter applied).
### Table 51 Descriptive Statistics of Individuals in Motivated Approach Quadrant

<table>
<thead>
<tr>
<th>Node</th>
<th>Count</th>
<th>Percent</th>
<th>Count</th>
<th>Percent</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1</td>
<td>11</td>
<td>18.97</td>
<td>53</td>
<td>23.25</td>
<td></td>
</tr>
<tr>
<td>S2</td>
<td>9</td>
<td>15.52</td>
<td>24</td>
<td>10.53</td>
<td>Overrepresented</td>
</tr>
<tr>
<td>S3</td>
<td>8</td>
<td>13.79</td>
<td>17</td>
<td>7.46</td>
<td>Overrepresented</td>
</tr>
<tr>
<td>S4</td>
<td>10</td>
<td>17.24</td>
<td>32</td>
<td>14.04</td>
<td></td>
</tr>
<tr>
<td>S5</td>
<td>5</td>
<td>8.62</td>
<td>17</td>
<td>7.46</td>
<td></td>
</tr>
<tr>
<td>S6</td>
<td>5</td>
<td>8.62</td>
<td>37</td>
<td>16.23</td>
<td>Underrepresented</td>
</tr>
<tr>
<td>S7</td>
<td>10</td>
<td>17.24</td>
<td>48</td>
<td>21.05</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>58</td>
<td>228</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* In the cell counts it can be seen that the bipolar and attenuated bipolar (S1-S5) make up the majority of the Motivated Approach cohort (43/58) and that the S7 node has 10 individuals represented. The unipolar (S6) node is underrepresented.
### Table 52  Binary Logistic Regression into Bipolar Disorder (BD) Diagnosis

<table>
<thead>
<tr>
<th>Predictor (reference level, category)</th>
<th>OR</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family History BD (0,1)</td>
<td>2.93</td>
<td>[1.41, 6.10]</td>
<td>.004</td>
</tr>
<tr>
<td>Perceived Childhood Relational Trauma (PCRT) (0,1)</td>
<td>4.31</td>
<td>[1.96, 9.48]</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Prestige approach Motivation (L, H)</td>
<td>4.20</td>
<td>[2.03, 8.69]</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

*Note. L= low MSPaM level; H = high MSPaM level, OR = odds ratio; CI = confidence interval. ab 0 indicates negative and 1 indicates positive.*

### Table 53  Binary Logistic Regression into Perceived Childhood Relational Trauma (PCRT)

<table>
<thead>
<tr>
<th>Predictor (reference level, category)</th>
<th>OR</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family History BD (0,1)</td>
<td>4.65</td>
<td>[2.35, 9.23]</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

*Note. BD = bipolar disorder; OR = odds ratio; CI = confidence interval. a 0 indicates negative and 1 indicates positive.*
TABLE 54  Binary Logistic Regression into Bipolar Disorder (BD) Diagnosis

<table>
<thead>
<tr>
<th>Predictor (reference level, category)</th>
<th>OR</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family History BD (0,1)\textsuperscript{a}</td>
<td>4.55</td>
<td>[2.49, 8.34]</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

*Note. OR = odds ratio; CI = confidence interval. \textsuperscript{a} 0 indicates negative and 1 indicates positive.*

TABLE 55  Binary Logistic Regression into Bipolar Disorder (BD) Diagnosis

<table>
<thead>
<tr>
<th>Predictor (reference level, category)</th>
<th>OR</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family History BD (0,1)\textsuperscript{a}</td>
<td>3.29</td>
<td>[1.65, 6.58]</td>
<td>.001</td>
</tr>
<tr>
<td>Perceived Childhood Relational Trauma (PCRT) (0,1)\textsuperscript{b}</td>
<td>4.02</td>
<td>[1.93, 8.38]</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

*Note. OR = odds ratio; CI = confidence interval. \textsuperscript{ab} 0 indicates negative and 1 indicates positive.*
Table 56  Coefficients for Multiple Regression Analysis into Mood Disorder Questionnaire (MDQ)

<table>
<thead>
<tr>
<th>Predictor category (reference level, category)</th>
<th>Coefficient</th>
<th>SE Coef</th>
<th>T Statistic</th>
<th>DF</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>3.53</td>
<td>0.41</td>
<td>8.53</td>
<td>173</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Family History BD (0, 1)</td>
<td>1.76</td>
<td>0.58</td>
<td>3.05</td>
<td>173</td>
<td>.003</td>
</tr>
<tr>
<td>Childhood Relational Trauma (PCRT) (0, 1)</td>
<td>2.01</td>
<td>0.61</td>
<td>3.32</td>
<td>173</td>
<td>.001</td>
</tr>
<tr>
<td>Prestige approach Motvn (MSPaM-binary) (L, H)</td>
<td>3.32</td>
<td>0.52</td>
<td>6.33</td>
<td>173</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Note. L= low MSPaM level; H = high MSPaM level; SE Coef = standard error of the coefficient; OR = odds ratio; CI = confidence interval. a0 indicates negative and 1 indicates positive; ccategory level interaction.
THE PRESTIGE MODEL OF SPECTRUM BIPOLARITY

Table 57  Coefficients for Multiple Regression Analysis into MDQ with Interacting Term

<table>
<thead>
<tr>
<th>Predictor (reference level, category)</th>
<th>Coefficient</th>
<th>SE Coef</th>
<th>T Statistic</th>
<th>DF</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>3.02</td>
<td>0.44</td>
<td>6.79</td>
<td>173</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Family History BD (0, 1)a</td>
<td>3.52</td>
<td>0.84</td>
<td>4.20</td>
<td>173</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Childhood Relational Trauma (PCRT) (0, 1)b</td>
<td>2.17</td>
<td>0.60</td>
<td>3.63</td>
<td>173</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Prestige approach Motvn (MSPaM-binary) (L, H)</td>
<td>4.37</td>
<td>0.63</td>
<td>6.91</td>
<td>173</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Family Hx (1)*Prestige approach Motivation (H)c</td>
<td>-3.10</td>
<td>1.09</td>
<td>-2.85</td>
<td>173</td>
<td>.005</td>
</tr>
</tbody>
</table>

Note. L= low MSPaM level; H = high MSPaM level; SE Coef = standard error of the coefficient; DF = degrees of freedom. \(^a\)0 indicates negative and 1 indicates positive; \(^b\)category level interaction.
### Table 58  Binary Logistic Regression into Bipolar Disorder (BD) Diagnosis with Interacting Term

<table>
<thead>
<tr>
<th>Predictor (reference level, category)</th>
<th>Coefficient</th>
<th>SE Coef</th>
<th>OR</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>-1.91</td>
<td>0.36</td>
<td>6.78</td>
<td>[2.28, 20.16]</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Childhood Relational Trauma (PCRT) (0, 1)a</td>
<td>1.91</td>
<td>0.56</td>
<td></td>
<td>[2.28, 20.16]</td>
<td>.001</td>
</tr>
<tr>
<td>Prestige approach Motvn (MSPaM-binary) (L, H)</td>
<td>1.48</td>
<td>0.44</td>
<td>4.41</td>
<td>[1.87, 10.37]</td>
<td>.001</td>
</tr>
<tr>
<td>PCRT (1)*Prestige approach Motivation (H)b</td>
<td>0.02</td>
<td>0.76</td>
<td>1.02</td>
<td>[0.23, 4.52]</td>
<td>.978</td>
</tr>
</tbody>
</table>

*Note.* L = low MSPaM level; H = high MSPaM level; SE Coef = standard error of the coefficient; OR = odds ratio; CI = confidence interval. *a* 0 indicates negative and 1 indicates positive; *b* category level interaction.
### Table 59  Correlation Matrix of MSPaM, MSPaM Factors, SISE and Mood Scores

<table>
<thead>
<tr>
<th></th>
<th>MSPaM</th>
<th>NEGEST</th>
<th>POSEST</th>
<th>POSEST</th>
<th>SISE</th>
<th>ASRM</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>-AFF</td>
<td>-ACT</td>
<td>-AFF</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEGESTAFF</td>
<td>0.89***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>POSESTACT</td>
<td>0.87***</td>
<td>0.67***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>POSESTAFF</td>
<td>0.84***</td>
<td>0.61***</td>
<td>0.64***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SISE</td>
<td>-0.24***a</td>
<td>-0.41***a</td>
<td>-0.11a</td>
<td>-0.09a</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASRM</td>
<td>0.24***</td>
<td>0.17</td>
<td>0.25***</td>
<td>0.19**</td>
<td>0.04a</td>
<td></td>
</tr>
<tr>
<td>PHQ-9</td>
<td>0.34***</td>
<td>0.50***</td>
<td>0.18**</td>
<td>0.24***</td>
<td>-0.52***a</td>
<td>0.00</td>
</tr>
</tbody>
</table>

*Note.*  
Spearman correlation – the remainder are Pearson. NEGESTAFF designates the MSPaM factor “negative esteem affect”, POSESTACT designates “positive esteem action” and POSESTAFF “positive esteem affect”.  
*p < .05, ** p < .01, *** p < .001.
### Table 60  MDQ Responses by Self-nominated Diagnosis

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>MDQ negative</th>
<th>MDQ positive</th>
<th>Total</th>
<th>MDQ pos. %</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Diagnosis</td>
<td>100</td>
<td>6</td>
<td>106</td>
<td>5.7</td>
</tr>
<tr>
<td>Bipolar Disorder</td>
<td>14</td>
<td>41</td>
<td>55</td>
<td>74.6</td>
</tr>
<tr>
<td>Depression</td>
<td>48</td>
<td>15</td>
<td>63</td>
<td>23.8</td>
</tr>
<tr>
<td>Other Diagnosis</td>
<td>3</td>
<td>1</td>
<td>4</td>
<td>25.0</td>
</tr>
</tbody>
</table>

### Table 61  Mood State by Spectrum Node

<table>
<thead>
<tr>
<th>Spectrum</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mood</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Euthymic</td>
<td>7</td>
<td>3</td>
<td>4</td>
<td>15</td>
<td>11</td>
<td>12</td>
<td>38</td>
<td>90</td>
</tr>
<tr>
<td>(Hypo)manic</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>13</td>
<td>4</td>
<td>2</td>
<td>0</td>
<td>23</td>
</tr>
<tr>
<td>Depressed</td>
<td>26</td>
<td>11</td>
<td>13</td>
<td>0</td>
<td>0</td>
<td>15</td>
<td>0</td>
<td>65</td>
</tr>
<tr>
<td>“Mixed”</td>
<td>6</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Mild depression</td>
<td>9</td>
<td>7</td>
<td>0</td>
<td>4</td>
<td>2</td>
<td>7</td>
<td>9</td>
<td>38</td>
</tr>
<tr>
<td>Missing</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>All</td>
<td>52</td>
<td>23</td>
<td>17</td>
<td>32</td>
<td>17</td>
<td>37</td>
<td>47</td>
<td>225</td>
</tr>
<tr>
<td></td>
<td>CSW average (CSW.av)</td>
<td>CSW appearance</td>
<td>CSW competition</td>
<td>CSW approval</td>
<td>MSIS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>----------------</td>
<td>----------------------</td>
<td>----------------</td>
<td>-----------------</td>
<td>--------------</td>
<td>------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CSW appearance</td>
<td>0.85</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CSW competition</td>
<td>0.78</td>
<td>0.50</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CSW approval</td>
<td>0.84</td>
<td>0.65</td>
<td>0.41</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MSIS</td>
<td>0.55</td>
<td>0.46</td>
<td>0.44</td>
<td>0.47</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MSPaM</td>
<td>0.58</td>
<td>0.45</td>
<td>0.49</td>
<td>0.49</td>
<td>0.70</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note. Pearson correlations.*

*N = 228; p values for all correlations <.001.*
### Table 63: Coefficients for Multiple Regression Analysis into MSPaM, Focusing on Family History as a Predictor

<table>
<thead>
<tr>
<th>Predictor/Factor Level</th>
<th>Factor Reference Level</th>
<th>Coefficient</th>
<th>Standard Error</th>
<th>T Statistic</th>
<th>DF</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td></td>
<td>16.23</td>
<td>4.80</td>
<td>3.38</td>
<td>209</td>
<td>.001</td>
</tr>
<tr>
<td>Family history BD</td>
<td>0</td>
<td>-2.57</td>
<td>1.15</td>
<td>-2.24</td>
<td>209</td>
<td>.026</td>
</tr>
<tr>
<td>MDQ</td>
<td></td>
<td>0.96</td>
<td>0.29</td>
<td>3.27</td>
<td>209</td>
<td>.001</td>
</tr>
<tr>
<td>PHQ-9</td>
<td></td>
<td>0.16</td>
<td>0.14</td>
<td>1.11</td>
<td>209</td>
<td>.267</td>
</tr>
<tr>
<td>ASRM</td>
<td></td>
<td>0.55</td>
<td>0.35</td>
<td>1.57</td>
<td>209</td>
<td>.118</td>
</tr>
<tr>
<td>MSIS</td>
<td></td>
<td>2.24</td>
<td>0.21</td>
<td>10.85</td>
<td>209</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

*Note.* BD = bipolar mood disorder, MDQ = Mood Disorder Questionnaire, PHQ-9 = Patient Health Questionnaire-9, ASRM = Altman Self-Rating Mania Scale, MSIS = MOPF Social Inclusion Sensitivity Scale. $r^2 = 54.18\%$ ($r^2_{adj} = 53.08\%$).

### Table 64: Coefficients for Multiple Regression Analysis into MSPaM with Respect to the Relative Contributions of Family History and Perceived Childhood Relational Trauma (PCRT)

<table>
<thead>
<tr>
<th>Predictor/Factor Level</th>
<th>Factor Reference Level</th>
<th>Coefficient</th>
<th>Standard Error</th>
<th>T Statistic</th>
<th>DF</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td></td>
<td>82.04</td>
<td>1.66</td>
<td>49.55</td>
<td>188</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Family history BD</td>
<td>0</td>
<td>-4.83</td>
<td>1.64</td>
<td>-2.93</td>
<td>188</td>
<td>.004</td>
</tr>
<tr>
<td>PCRT</td>
<td>0</td>
<td>-4.07</td>
<td>1.72</td>
<td>-2.36</td>
<td>188</td>
<td>.019</td>
</tr>
</tbody>
</table>

*Note.* BD = bipolar mood disorder, PCRT = perceived childhood relational trauma. $r^2 = 9.99\%$ ($r^2_{adj} = 9.03\%$).
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Author/s:
Le Bas, James

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Affect on the prestige landscape: the prestige model of spectrum bipolarity

Date:
2015

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