Sleep, Coping, and Vulnerability to Bipolar Disorder: Modelling Analyses in a Non-Clinical Sample

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Abstract

The aim of this study was to better understand the involvement of sleep and coping in mood/wellbeing outcomes related to bipolar disorder (BD). To enable testing of two novel multivariate models of these relationships, a dimensional approach to BD was employed, and data were collected from a general population sample. Vulnerability to BD was measured on a brief version of the General Behavior Inventory. In the models, vulnerability to BD was both an outcome and a predictor variable. Vulnerability to BD was framed as an outcome of trait-like sleep variables and levels of personality domains and motivational systems. State sleep and coping variables, posited as mediators, were included in analyses that assessed their impact on the models' ultimate outcome variables of mood and wellbeing (state mood and satisfaction with life). To provide a test of the specificity of the role of vulnerability to BD in this model, measures of the five factor model of personality and the behavioural activation and behavioural inhibition systems were also included. Data were collected using an online questionnaire comprising previously validated measures. A total of 608 responses were analysed using correlations, multiple regressions (standard, hierarchical and regression-based mediation analyses), and structural equation modelling. Statistical associations were consistent with previous findings in that hypothesised correlations were observed (between vulnerability to hypomania scores and extraversion; vulnerability to hypomania scores and agreeableness; vulnerability to depression scores and neuroticism; and Morningness with both vulnerability to depression and hypomania scores). In the context of all personality and motivation traits, Neuroticism predicted vulnerability to depression and hypomania scores, and the only motivation variable to predict vulnerability to depression or hypomania scores was BAS Fun Seeking.
Negative correlations between healthy trait-like sleep and depression/hypomania scores were observed. Trait-like sleep variables were found to predict vulnerability to BD (depression and hypomania scores) when forced to share variance with personality and motivation traits, providing support that they are important variables to consider when investigating vulnerability to BD and wellbeing. Structural equation modelling provided evidence that state sleep scores and coping styles acted as partial mediators between (a) trait-like sleep and vulnerability to depression or vulnerability to hypomania and (b) state mood and satisfaction with life. Both coping styles and state sleep directly impacted perceived wellbeing, and state sleep directly impacted coping. It was concluded that state and trait-like sleep are important variables when investigating mood and wellbeing outcomes, and clinicians are encouraged to attend to the variables of sleep quality and coping styles in mood and mental health.
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During the eon (well that’s what it felt like) that was my post-graduate studies I came to realise that many people have been touched by Bipolar Disorder. Whether it was a family member, a friend, a neighbour, or a co-worker, the more times I was asked about my research the more this realisation occurred. The more people spoke about their experiences the more I saw them open up, and sometimes I was a complete stranger. I hope that this tiny piece of research continues to fuel that ‘talk’.

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you for always managing to make me laugh until I can barely talk. Finally, to the baby
growing inside me, I look forward to meeting you.
Declaration

This is to certify that

(i) the thesis comprises only my original work, except where indicated
(ii) due acknowledgment has been made in the text to all other material used
(iii) the thesis is less than 60,000 words in length, exclusive of tables, references and appendices.

Simmone Poulios
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Overview of Thesis

The overarching aim of the present project was to investigate the role of sleep and coping in a non-clinical sample, quantified by degree of vulnerability to bipolar disorder (BD). The first aim was to explore the interaction between known trait correlates (personality and motivation traits) and BD vulnerability. The second aim was to conduct a novel investigation of trait-like sleep as a correlate of vulnerability to BD. The third aim was to investigate if state sleep acted as a mediator in models relating vulnerability to BD to state mood and satisfaction with life. Finally, the fourth aim was to investigate the role of coping within those same models. These aims were addressed using a single cross-sectional self-report study in the general population, with BD understood quantitatively, also referred to as an analogue design. Conceptual models predicting the direction in which variables interact were developed, and for what follows it is useful to tag these particular conceptual models with a name, specifically The BD, Coping, and Sleep models. The BD, Coping, and Sleep models investigated measures of personality, sleep, motivation (behavioural activation and behavioural inhibition systems; BAS and BIS), vulnerability to BD, potential mediating variables of coping and sleep, and mood and wellbeing outcome variables (operationalised as mood and satisfaction with life). It must be clarified here that two models were tested, as there are reasons to believe that BD is two dimensional in nature, with one dimension being depression and the other being hypo/mania. Thus, the first model investigated how vulnerability to depression interacted with sleep (trait and state), coping styles, and mood/wellbeing variables (measured by positive and negative affect and satisfaction with life); and the second model investigated how vulnerability to hypo/mania interacted with these same variables. A novel aspect of the present study was the separation of sleep into trait-like and state components. BD onset is often preceded by
sleep disturbances (Ritter, Marx, Bauer, Leopold, & Pfennig, 2011) and approximately 70% of individuals with BD also suffer from significant clinical disturbances with sleep (Harvey, Schmidt, Scarnà, Semler, & Goodwin, 2005). Sleep studies have also shown that recent changes in sleep impact on mood (Fortunato & Harsh, 2006). Another focus of the BD, Coping, and Sleep models was the involvement of coping styles and their potential involvement in the relationship between vulnerability to BD, state sleep and mood and wellbeing outcome variables (state mood and satisfaction with life).

Chapter One begins with a brief description of the two conceptual models that frame the project. It then describes BD, from early conceptualisations, to current diagnostic criteria, developmental perspectives of BD, course of BD, prevalence, and biological pathways. The chapter then describes comorbidities, and psychological treatments.

Chapter Two reviews literature investigating relationships between BD and trait correlates, specifically personality and motivational systems (i.e., BIS/BAS). The chapter begins with a brief description of the General Behavior Inventory (GBI), a validated measure of vulnerability to BD. Then definitions of both trait and personality are provided. Chapter Two then reviews studies investigating personality and BIS/BAS variables' associations with BD.

Chapter Three begins with a description of normal sleep. Neurohormone regulation is briefly discussed, followed by circadian rhythm entrainment and a broad range of findings from sleep deprivation studies in healthy controls. The chapter then describes sleep disorders, focusing on insomnia. Then findings regarding sleep in individuals with BD and those vulnerable to BD are reviewed. Sleep disturbances documented across the course of BD are also discussed. Following this, sleep
deprivation as a treatment for BD depression is described. Finally, the trait variable of Morningness and findings related to chronotype observed in BD are discussed.

Chapter Four introduces the notion of coping styles. The chapter continues with an overview of research investigating coping styles in individuals with BD. This includes comparative studies addressing the coping styles of individuals with BD to healthy controls, individuals with unipolar depression (UD), and to unaffected relatives of individuals with BD. A description of research that has investigated BD, coping and personality traits follows. Management of episode prodromes in people with BD is then introduced.

Chapter Five presents the two BD, Coping, and Sleep models in detail. The aims are presented, then the chapter describes the measures used for each variable and also introduces the outcome variables that were used to measure the impact of the predictors involved in the models (specifically mood and satisfaction with life). The chapter finishes by presenting the hypotheses of the present project.

Chapter Six describes the project methodology. Sampling and recruitment decisions are described, and choice of measurement instruments defended. The study generated a large amount of data, supporting multiple approaches to data analysis. The chapter concludes with a description of the analytic approach.

Chapter Seven presents the results of the present project including data cleaning and preliminary analysis of the psychometric properties of all scales. The chapter describes correlation analyses, multiple regression analyses, partial mediation analyses and finally the Structural Equation Modelling (SEM) results. The chapter closes with a summary table reviewing findings in relation to the study's specified aims and hypotheses.
The study's findings are discussed in Chapter Eight. The chapter begins with a restatement of the aims of the project. Then in three separate sections (personality and motivation, sleep, and coping) findings are discussed in relation to the project's hypotheses with links to previous research. The implications of the findings and suggestions for future research are also discussed. Limitations of the present project are then described. The chapter ends with the final conclusions that: state and trait-like sleep are important variables when investigating mood and wellbeing outcomes; and the present project emphasises the importance of attending to coping styles and sleep quality with respect to mood and wellbeing.
1.0 Bipolar Disorder

1.1 Overview of Chapter 1

This chapter begins with a description of BD and an introduction to the two models investigated in the present project. Conceptualisations of BD from early reports to present day are introduced, highlighting that, although the current diagnostic system adopts a useful categorical approach, a spectrum of BD may be more valid. This leads into a discussion of current diagnostic criteria for BD and sub-types. The chapter then describes developmental perspectives, course of BD, prevalence, comorbidities, biological pathways, and psychological treatments.

1.2 The Present Study

BD is a complex disorder of mood (Goodwin & Jamison, 2007). Lifetime prevalence rates around the world indicate that BD affects approximately 0.1% (observed in India) to 4.4% (in the United States) of the population (Merikangas et al., 2011). BD affects males and females equally (Mitchell, Slade, & Andrews, 2004).

Many individuals with BD also suffer from sleep disturbances, both during episodes and when there are no residual symptoms (euthymia), and the presence of sleep disturbances increases the risk of manic or depressive episode relapses (Murray & Harvey, 2010; Sylvia et al., 2012). The relationship between sleep and BD has and continues to fuel research into the causal links between sleep disturbance and mood symptoms. It is likely that how an individual copes with stressors, such as elevated or decreased mood, impacts on how one recovers from episodes (Lam & Wong, 2005).

The present project sought to investigate the role of sleep in vulnerability to BD. A novel feature of the present project was a distinction between state sleep (the quality of sleep experienced in the last month) and trait-like sleep (overall quality of sleep for most of one's life). Trait-like sleep is conceptualised to predict vulnerability to BD.
(vulnerability to depression or vulnerability to hypo/mania) and state sleep is used as an outcome of vulnerability to BD. The present project also sought to investigate how coping influences this interaction. A second novel feature of the project is the investigation of how general coping styles and state sleep may interact and impact on mood/wellbeing outcome variables.

The overarching aim of the present project was to investigate two parallel models named the *BD, Coping, and Sleep models*. The ultimate models that were tested divided the Vulnerability to BD variable into vulnerability to BD-Depression and vulnerability to BD-Hypomania. Figure 1 represents the conceptual framework for the present project. This framework also included background investigations that preceded SEM investigations. These background investigations were the correlation and regression analyses that tested the influence of personality and motivational system traits. The following chapters will offer a comprehensive review of the literature into each of the variables and relationships that make up the models. The focus of the conceptual models is on the interaction between vulnerability to BD (measured as both vulnerability to depression and vulnerability to hypo/mania), sleep, and coping, in relation to the normal range outcome variables of mood and satisfaction with life.
1.3 Conceptualisations of BD

The first descriptions of BD date back to the Classical period in Greek history, between the fourth and fifth century B.C.E. (Goodwin & Jamison, 2007). Broad definitions of mania and melancholia were articulated by philosophers such as Hippocrates and Aristotle, and later in the second century C.E. by Arataeus (Angst & Marneros, 2001; Goodwin & Jamison, 2007). Classical terminologies defined the condition of melancholia as the presence of despondency, irritability, restlessness and an aversion to food (Goodwin & Jamison, 2007). Soranus described mania as a condition including continued wakefulness, fluctuating mood states ("anger and merriment") and an impaired ability to reason (Goodwin & Jamison, 2007). Following the Dark Ages there was a resurgence of interest in classifying mental illness. In the late Middle Ages, Andres Piquer of Spain described how the king at the time suffered from
“melancholic-manic affect” (as cited in Goodwin & Jamison, 2007), describing it as one illness.

In the nineteenth century Jean-Pierre Falret described clinical observations of what today would be considered BD. Falret described the temporal relationship between mania and melancholia as one of circular insanity [la folie circulaire], that is, a single illness. A few years later (in 1854) Baillarger referred to the same illness as double insanity [la folie a double forme]. Between 1850 and 1854 Falret detailed the course of the illness he observed in patients with this form of ‘insanity’. In 1854 he gave a lecture detailing the circular insanity, the lecture was transcribed and almost 130 years later it was translated to English. Excerpts from that lecture are below. We begin with the clinical description of mania:

…the manic state [l’etat maniaque]…is characterised by simple exaltation of thought and feeling, which at first is recognised merely as a happy moment when the spirits are high, where everything seems easy, and it feels natural to be always smiling. At this point the patient presents only an abundance of activity in all the faculties; he appears to be changed for the better, to the astonishment of those around him, were it not that already one can see the dawn of subtle emotional aberrations, and were it not that this behaviour is beginning to appear strange and even disorganised. Progressively and rapidly the condition worsens: the profusion of ideas is prodigious, the feelings are exalted, great affection is expressed for people towards the patient had previously felt indifferent, and hatred flares against those persons who have before been loved the most. Their movements are rapid and unceasing. It is at this point that if the patients are left to themselves,
they turn over their furniture, change apartments, dig up their garden, become mischievous, malicious, and play all sorts of tricks, make plans which they impulsively carry out, compose and write prose and verse; and this prodigious activity, flowing forth in all directions, is present at night as well as during the daytime... (Sedler, 1983, p. 1130)

Falret eloquently describes the affect that is often observed, the change in interpersonal style and the behaviours that ensue. He alludes to the impulsive behaviour that is often observed in those experiencing a manic episode such as the over-spending or inappropriate amorous acts that are often later regretted. During this lecture he also described the switch; the phase when one is coming out of one’s manic episode and beginning to enter a state of depression, or melancholia:

At the point where the agitation ceases, there occurs a state that is difficult to characterize: it consists of the final residue of excitement and also of the depression, which is just beginning...Finally to appreciate their actual condition, one must look for what is missing rather than to what is manifest. Then one sees that the patients do not speak, or do much of anything, as one would expect if they were in a normal state...It is a state in which reason has a difficult time maintaining a balance between the fading excitement and the depression that is beginning, although it is sometimes possible to observe the patient’s efforts to succeed in this struggle... (Sedler, 1983, p. 1131)

Again Falret has captured the struggle that patients often experience, detailing some of the prodromes, symptoms first recognised at the onset of a manic or depressive
episode, that occur before the onset of the depressive episode, which can be read in the passage below.

The state of depression [l'état de depression] also develops by degrees, especially in circular insanity with long phases, but we do not deny that in certain exceptional cases the onset may be sudden… the patient begins to withdraw and now speak only rarely. Sometimes they express remorse over their previous condition, of which they may retain some memory. Others appear humble. Soon these symptoms become more severe; the patients withdraw remaining all alone and motionless. If in their earlier state they were demanding now they are meek, and their humility may go so far as for them to refuse treatment in the belief that they do not deserve it…The patient has a feeling of general malaise, and the limbs are torporous. Appetite is decreased, and the patient eats little; digestion is equally slow, and defecation laborious. Sleep is better than during the phase of excitement, but it is neither regular nor prolonged…(Sedler, 1983, p. 1131)

As with his description of mania, Falret’s description of depression highlights behaviours seen before full-blown symptoms are observed. Two points are noteworthy, first is his observation that everything slows down, including behaviour, cognitions and biological processes. Second, his observation of sleep patterns alludes to comorbid sleep issues. Falret went on to distinguish between the depression (or melancholia) observed in circular insanity and 'true forms of melancholia' (presently associated with UD). Falret also noted that individuals with BD sometimes develop certain fixations that centre on themes of guilt, paranoia of being poisoned, ruin, and humility. Finally, Falret made some epidemiological observations, specifically that this illness was observed in
twice as many females as males, generally persisted throughout their life, and had a poor prognosis (Sedler, 1983).

Arguably the most influential of the descriptions of BD was provided by the German psychiatrist, Emil Kraepelin, who in 1899, described the same illness as Falret but called it manic-depressive insanity. Kraepelin developed a nosology dividing mental disorders into two categories: manic-depressive illness and schizophrenia, then known as dementia praecox. He documented the course of the illness, described subtypes now referred to as mixed states and cyclothymia, observed the length of episode cycles, observed that poorer prognostic outcomes were associated with comorbid substance abuse issues, documented the importance of genetics and heritability in the manifestation of the illness, and believed that the conditions or subtypes occurred on a spectrum, instead of existing as separate illnesses (Goodwin & Jamison, 2007).

Following on from Kraepelin, in the late 1940's and early 1950's, Karl Kleist and his protégés, Leonhard and Neele, were the first to employ the terms unipolar and bipolar (as cited in Angst & Gamma, 2002). They proposed, as Falret had approximately 100 years earlier, that the mania and melancholia that occur in BD were not the same as either pure mania or pure melancholia (Angst & Gamma, 2002). So, not only were the mania and melancholia different to pure forms of either, but they occurred in one and the same illness.

This view that BD occurs on a spectrum is arguably the dominant one among current clinicians and researchers, and BD is said to be at the extreme end of an illness spectrum (Akiskal, 1996; Akiskal et al., 2000; Angst, Gamma, & Lewinsohn, 2002; Angst & Marneros, 2001; Bauer, Simon, Ludman, & Unützer, 2005; Cassano et al., 2004; Ghaemi, Ko, & Goodwin, 2002; Haslam, Holland, & Kuppens, 2012; Marneros, 2001; Merikangas et al., 2007; Phelps, Angst, Katzow, & Sadler, 2008; Walsh,
The other disorders of the bipolar spectrum include Bipolar II Disorder, Cyclothymia, Dysthymia, and Schizoaffective Disorder (Angst & Marneros, 2001). Researchers also suggest that the population has traits of BD, ranging from depressive, hyperthymic and cyclothymic temperaments to clinically significant traits warranting diagnosis (Marneros, 2001). Figure 2, taken from Angst (2007), depicts the current dimensional conceptualisation of the BD spectrum, relating temperament to major disorders along a spectrum of severity.

<table>
<thead>
<tr>
<th>Spectrum of Severity</th>
<th>Psychotic major mood disorders (mood congruent or incongruent)</th>
<th>Non psychotic major mood disorders</th>
<th>Minor mood disorders (sub-threshold)</th>
<th>Affective personality disorders*</th>
<th>Temperament (normal)</th>
<th>Symptoms (normal)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major</td>
<td>MDD (D)</td>
<td>MDD (D)</td>
<td>Dysthymia</td>
<td>Depressive personality disorder</td>
<td>Depressive temperament</td>
<td>(dsx)</td>
</tr>
<tr>
<td>Minor</td>
<td>BP-II (Dm)</td>
<td>BP-II (Dm)</td>
<td>RBD Minor depression (d)</td>
<td>Borderline/cycloid personality disorder</td>
<td>(mdsx)</td>
<td>(mdsx)</td>
</tr>
<tr>
<td></td>
<td>BP-I (MD)</td>
<td>BP-I (MD)</td>
<td>Minor BP Cyclothymia (md)</td>
<td>Cyclothymic temperament</td>
<td>(msx)</td>
<td>(msx)</td>
</tr>
<tr>
<td></td>
<td>(Md)</td>
<td>(Md)</td>
<td>(H)</td>
<td>Hyperthymic temperament</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mania (M)</td>
<td>Mania (M)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


BP-I = Bipolar depression type I; BP-II = Bipolar depression type II; D = Depression; d = Sub-threshold depression; Dm = Major depression and minor mania; dsx = Depressive symptoms; H = Hypomania; Md = Mania and minor depression; mdsx = Manic and depressive symptoms; msx = Manic symptoms; RBD = Recurrent brief depression. * = a term specifically used by Angst and colleagues and does not appear in the DSM-5.
A feature of Angst's conceptualisation is that personality disorders are a complicating factor when classifying psychiatric disorders, as the exact nature of how they relate to the manifestation of disorder remains unresolved. Angst clustered the psychiatric disorders, personality disorders, temperamental styles (often seen in the non-clinical sample) and pure symptoms into categories along a spectrum, ranging from Major Depressive Disorder (MDD) to Mania (M), and then decreasing in severity under each point in the spectrum.

If BD is perceived to occur on a dimensional spectrum then large community samples could be used to investigate proneness or vulnerability to BD, from those who score low on mania- or depression-proneness to those who score high on both. Furthermore, taking on this theoretical perspective means that research data and findings are not limited to 'pure' disorders (Krueger & Piasecki, 2002), and research designs are not influenced by medication effects or lifestyle consequences of the disorder. Krueger and Piasecki (2002) recommend the use of SEM analysis to investigate dimensional psychopathology.

1.4 Diagnostic Criteria for BD

In contrast to the spectrum view of BD, current diagnostic systems continue to take a categorical approach, as seen in the Diagnostic and Statistical Manual of Mental Disorders (5th ed., DSM-5, American Psychiatric Association, 2013) and International Classification of Diseases (World Health Organization, 1994). A hierarchical spectrum model has been purported to better represent individuals with a mental health diagnosis by viewing variation in pathology as dimensional and continuous, and hierarchically ordering the continuous variation between pathology (Krueger & Piasecki, 2002). Tensions between optimal research description and diagnostic classification are beyond
the current scope: for a detailed discussion of the limitation of a categorical diagnostic system see Cuthbert and Insel (2013), Angst (2007) or Goldberg (2000).

Although the present project employed a dimensional approach to BD, much of the reviewed literature employs categorical diagnoses. It is therefore necessary to describe the categorical BD construct as defined by current diagnostic systems. In the contemporary DSM-5 (American Psychiatric Association, 2013), Bipolar and Related Disorders have been separated from Depressive Disorders (American Psychiatric Association, 2013). The following section briefly describes the diagnostic criteria for Bipolar Disorder I (BD I), Bipolar Disorder II (BD II), and Cyclothymic Disorder as defined in the DSM-5 (American Psychiatric Association, 2013).

1.4.1 BD I.

The current diagnostic criteria posit that a single manic episode is necessary and sufficient for a diagnosis of BD I (American Psychiatric Association, 2013). However, individuals with BD I typically experience major depressive episodes during their lives (American Psychiatric Association, 2013). Table 1, taken directly from the DSM-5, lists the diagnostic criteria for a Manic Episode. Episodes can be classed into mild, moderate, or severe, and specifiers include with anxious distress, mixed features, rapid cycling, melancholic features, atypical features, mood-congruent psychotic features, mood-incongruent psychotic features, catatonia, peripartum onset or with seasonal pattern (American Psychiatric Association, 2013).
Table 1

**DSM-5 Manic Episode Criteria (American Psychiatric Association, 2013, p. 124)**

| A. | A distinct period of abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased goal-directed activity or energy, lasting at least 1 week and present most of the day, nearly every day (or any duration if hospitalization is necessary). |
| B. | During the period of mood disturbance and increased energy or activity, three (or more) of the following symptoms (four if the mood is only irritable) are present to a significant degree and represent a noticeable change from usual behavior: |
|   | 1. Inflated self-esteem or grandiosity. |
|   | 2. Decreased need for sleep (e.g., feels rested after only 3 hours of sleep). |
|   | 3. More talkative than usual or pressure to keep talking. |
|   | 4. Flights of ideas or subjective experience that thoughts are racing. |
|   | 5. Distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli), as reported or observed. |
|   | 6. Increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation (i.e., purposeless non-goal-directed activity). |
|   | 7. Excessive involvement in activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments). |
| C. | The mood disturbance is sufficiently severe to cause marked impairment in social, or occupational functioning or to necessitate hospitalization to prevent harm to self or others, or there are psychotic features. |
| D. | The episode is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication, other treatment) or to another medical condition. |

**Note.** A full manic episode that emerges during antidepressant treatment (e.g., medication, electroconvulsive therapy) but persists at a fully syndromal level beyond the physiological effect of that treatment is sufficient evidence for a manic episode and therefore, a bipolar I diagnosis. **Note.** Criterion A-D constitute a manic episode. At least one lifetime manic episode is required for the diagnosis of a bipolar I disorder.

1.4.2 BD II.

BD II is diagnosed on the basis of one or more Major Depressive Episodes as well as at least one Hypomanic Episode. Tables 2 and 3, taken directly from the DSM-5, list diagnostic criteria for a Major Depressive Episode and a Hypomanic Episode, respectively. A Hypomanic Episode refers to a period of elevated mood, with symptoms akin to a Manic Episode but less severe. For example, hospitalisation or the presence of psychosis are exclusionary criteria for a diagnosis of a Hypomanic episode (American Psychiatric Association, 2013).
Table 2


<table>
<thead>
<tr>
<th>Major Depressive Episode</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.</td>
</tr>
<tr>
<td>Note: Do not include symptoms that are clearly attributable to another medical condition.</td>
</tr>
<tr>
<td>1. Depressed mood most of the day, nearly every day, as indicated by either subjective rapport (e.g., feels sad, empty, or hopeless) or observation made by others (e.g., appears tearful). (Note: In children and adolescents, can be irritable mood.)</td>
</tr>
<tr>
<td>2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observations).</td>
</tr>
<tr>
<td>3. Significant weight loss when not dieting or weight gain (e.g., change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (Note: In children, consider failure to make expected weight gain.)</td>
</tr>
<tr>
<td>4. Insomnia or hypersomnia nearly every day.</td>
</tr>
<tr>
<td>5. Psychomotor agitation or retardation nearly every day (observable by others; not merely subjective feelings of restlessness or being slowed down).</td>
</tr>
<tr>
<td>6. Fatigue or loss of energy nearly every day.</td>
</tr>
<tr>
<td>7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).</td>
</tr>
<tr>
<td>8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).</td>
</tr>
<tr>
<td>9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.</td>
</tr>
<tr>
<td>B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.</td>
</tr>
<tr>
<td>C. The episode is not attributable to the physiological effects of a substance or another medical condition.</td>
</tr>
<tr>
<td>Note: Criterion A-C constitute a major depressive episode. Major depressive episodes are common in bipolar I disorder but are not required for the diagnosis of bipolar I disorder.</td>
</tr>
<tr>
<td>Note: Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in Criterion A, which may resemble a depressive episode. Although such symptoms may be understandable or considered appropriate to the loss, the presence of a major depressive episode in addition to the normal response to a significant loss should also be carefully considered. This decision inevitably requires the exercise of clinical judgment based on the individual's history and the cultural norms for the expression of distress in the context of loss.</td>
</tr>
</tbody>
</table>
Table 3

*DSM-5 Criteria for a Hypomanic Episode (American Psychiatric Association, 2013, p. 132)*

**Hypomanic Episode**

A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased activity or energy, lasting at least 4 consecutive days and present most of the day, nearly every day.

B. During the period of mood disturbance and increased energy and activity, three (or more) of the following symptoms (four if the mood is only irritable) have persisted, represent a noticeable change from usual behavior, and have been present to a significant degree:

1. Inflated self-esteem or grandiosity.
2. Decreased need for sleep (e.g., feels rested after only 3 hours of sleep).
3. More talkative than usual or pressure to keep talking.
4. Flight of ideas or subjective experience that thoughts are racing.
5. Distractibility (i.e., attention too easily drawn too unimportant or irrelevant external stimuli), as reported or observed.
6. Increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation.
7. Excessive involvement in activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments).

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

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

E. The episode is not severe enough to cause marked impairment in social or occupational functioning or to necessitate hospitalization. If there are psychotic features, the episode is, by definition, manic.

F. The episode is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication, other treatment).

*Note:* A full hypomanic episode that emerges during antidepressant treatment (e.g., medication, electroconvulsive therapy) but persists at a fully syndromal level beyond the physiological effect of that treatment is sufficient evidence for a hypomanic episode diagnosis. However, caution is indicated so that one or two symptoms (particularly increased irritability, edginess, or agitation following antidepressant use) are not taken as sufficient for diagnosis of a hypomanic episode, nor necessarily indicative of a bipolar diathesis.

### 1.4.3 Cyclothymic Disorder.

Cyclothymic disorder is diagnosed when an adult has experienced at least a two-year period where multiple hypomanic and depressive symptoms occur, and the individual has not been symptom free for more than two months. To diagnose cyclothymic disorder in children and adolescents, symptoms must have been experienced for a period of one year. During any of this time, symptoms must not have met criteria for a Major Depressive Episode, Manic or Mixed Episode.
Diagnosis of any of the BD's described above must also include the following requirements: symptoms must cause clinical impairment or distress in important areas of functioning (American Psychiatric Association, 2013) and cannot be better explained by delusional disorder, schizoaffective disorder, schizophrenia, schizophreniform disorder, other schizophrenia spectrum or psychotic disorder, nor can they be attributable to another medical condition or physiological effects of a substance (American Psychiatric Association, 2013).

### 1.4.4 Changes from DSM-IV-TR to DSM-5.

The changes to the diagnostic criteria for BD I between the DSM-IV-TR and the DSM-5 include the addition of a further criterion for mania and hypomania. Specifically, aside from an elevation in mood, a change in activity and energy levels must be observed. The term "mixed episode" is now "with mixed features", recognising that symptoms of mania and hypomania occur in depressive episodes, and symptoms of depression occur in hypomania and mania (American Psychiatric Association, 2013). New specifiers for both BD I and BD II include "with mood-congruent psychotic features" and "mood incongruent psychotic features", and an "anxious distress" specifier which is indicative of higher risk and poorer prognosis (American Psychiatric Association, 2013). Criteria for each specifier are provided in the DSM-5 (American Psychiatric Association, 2013).

### 1.5 Development of BD

This section reviews research on BD from a developmental perspective. To more accurately diagnose BD it is important to be aware of the antecedents that have been documented to occur prior to the onset of BD. Retrospective studies note that participants recall first signs of BD when they were as young as 15 years (Lish, Dime-Meenan, Whybrow, & Price, 1994). Often, an individual will have lived with BD for
quite some time before an accurate diagnosis is made. This typically occurs because the first episode is often a depressive one, and it is not until a manic episode follows that a diagnosis of BD is made: research has identified that 47% of individuals who were eventually diagnosed with BD, were initially diagnosed with depression (Lish et al., 1994). Furthermore, between 20 and 40% of individuals who are first diagnosed with a depressive episode are later diagnosed with BD (Hauser et al., 2007).

Previous research has also identified antecedents that may actually be early symptoms of BD (Duffy, 2010; Duffy, Alda, Crawford, Milin, & Grof, 2007; Henin et al., 2007). A longitudinal study by Duffy, Alda, Hajek, Sherry, and Grof (2010) monitored children of BD parents over a period up to 15 years. Their sample consisted of 207 of these "high-risk" individuals and 87 controls. Over the course of the study only 21 of the 207 were diagnosed with BD (I, II, or schizoaffective subtypes) during their mid-adolescence. Duffy et al. observed a clinical profile that was the same for 15 of the 21 subjects. The clinical sequence typically included a non-mood episode (anxiety, Attention Deficit Hyperactive Disorder, or sleep disorder) followed by a minor mood or adjustment disorder, then a major depressive episode and finally an episode of mania or hypomania. In 90% of their sample ($n = 19$) the clinical presentation of disorders went from a non-mood diagnosis, to a minor mood or adjustment disorder, and finally to a major mood disorder diagnosis. They saw that the majority of subjects with an eventual BD diagnosis first experienced a depressive episode, as this was the case in 16 of the 21 individuals. This finding is consistent with longitudinal studies reporting that anxiety disorders, mood disturbances and sleep difficulties precede the onset of BD and that mania does not occur prior to puberty (Akiskal et al., 1985; Duffy, 2009; Hammen, Burge, Burney, & Adrian, 1990; Henin et al., 2007; Hillegers et al., 2005; Shaw, Egeland, Endicott, Allen, & Hostetter, 2005).
Developmental studies of people whose parents have a BD diagnosis have also observed a sequential clinical presentation over time. Akiskal et al. (1985) observed adolescent offspring of parents with BD and found that although some presented with hypomanic symptoms, those who initially presented with non-affective symptoms later met diagnostic criteria for dysthymia or cyclothymic disorder. Akiskal et al. also observed that psychotic symptoms in mania did not present until after puberty. Lish et al. (1994) observed that most of the individuals who were subsequently given a BD diagnosis, initially presented with a depressive episode.

Duffy (2010) suggested that diagnostic systems should take into consideration the early course or antecedents to a full-blown BD diagnosis. When children of bipolar parents present to therapy, their parent’s illness is not taken into consideration when formulation and treatment plans are made. For these children, attempts to treat the prodrome (anxiety or depressive symptoms) with conventional treatment, such as play therapy or antidepressants, has been shown to be either unhelpful or worsen the course of the illness (Duffy, 2010). Research suggests that longitudinal clinical course and familial risk need also be taken into consideration when monitoring for BD and, thus, a clinical staging model would be more appropriate in the diagnosis of BD (Duffy, 2010).

1.6 Course of BD

Longitudinal studies have generally found that the average individual with a BD diagnosis will experience multiple affective episodes over their lifetime and will experience more depressive symptoms than manic or hypomanic symptoms (Judd et al., 2002). Individuals with BD who suffer from residual affective symptoms between episodes will relapse into another major affective episode faster than an individual with BD who does not experience residual affective symptoms (Judd et al., 2008). The vast majority of individuals with BD relapse within four years of their first episode (Tohen,
Waternaux, & Tsuang, 1990). One study suggested some 49% of individuals relapse within two years, and with majority of relapse (70%) being a depressive episode (Perlis et al., 2006).

The average age of diagnosis is 22.2 years (Goodwin & Jamison, 2007); however, the true age of onset is likely to be much earlier because early BD symptoms are non-specific. Individuals may suffer with undiagnosed BD symptoms for many years (Ghaemi, Sachs, Chiou, Pandurangi, & Goodwin, 1999). Some individuals have gone up to 10 years, and may see multiple professionals, before symptoms are correctly diagnosed (Hirschfeld, Lewis, & Vornik, 2003). As previously mentioned, researchers have identified early antecedents to BD, specifically presence of anxiety and depressive episodes observed in childhood and adolescence (Akiskal et al., 1985; Duffy, 2009; Hammen et al., 1990; Henin et al., 2007; Hillegers et al., 2005; Shaw et al., 2005). Furthermore, research has found that a high percentage of individuals first diagnosed with BD-NOS or cyclothymia later meet diagnostic criteria for BD I or BD II (Alloy et al., 2012; Axelson et al., 2011; Birmaher et al., 2009; Martinez & Fristad, 2013).

Factors impacting the course of BD include life events (Johnson et al., 2008), support networks (Johnson, Winett, Meyer, Greenhouse, & Miller, 1999), response to medication, medication compliance, response to psychosocial treatment (Huxley, Parikh, & Baldessarini, 2000), internal locus of control of health (Barnes et al., 2011; Poole, Simpson, & Smith, 2012), and the presence of comorbidities (Ostacher et al., 2006, see below).

1.7 Prevalence

Approximately 2.5% of the Australian population may warrant a diagnosis of BD (Goldney, Fisher, Dal Grande, Taylor, & Hawthorne, 2005). Worldwide, prevalence rates average 0.84% (for a review see Ferrari, Baxter, & Whiteford, 2011). When
considering the spectrum of BD disorders, the prevalence rates total 4.4 % (Merikangas et al., 2007). BD affects females and males equally (Ferrari et al., 2011; Goodwin & Jamison, 2007; Morgan, Mitchell, & Jablensky, 2005), and is also equally spread across social classes (Ferrari et al., 2011; Goodwin & Jamison, 2007). Recently, Fassassi, Vandeleur, Aubry, Castelao, and Preisig (2014) examined prevalence rates using the DSM-5 criteria and found that lifetime prevalence rates remained approximately the same as when DSM-IV-TR criteria were previously employed, with 1 % for a BD I diagnosis and 0.8 % for a BD II diagnosis.

Alloy et al. (2012) used scores from the General Behavior Inventory (GBI; Depue, Krauss, Spoont, & Arbisi, 1989) to identify diagnosis progression along the BD spectrum in high-risk non-patient college students \((n = 201)\) and controls \((n = 208)\). They monitored participants every four months (using questionnaires and semi-structured interviews) for approximately four and a half years; during that time, of the 57 individuals initially diagnosed with cyclothymia or BD NOS, 10.5 % progressed to a diagnosis of BD I, and 42.1 % progressed to a diagnosis of BD II. Similarly, of the 144 participants initially diagnosed with BD II, 17.4 % progressed to a BD I diagnosis. Of the 208 in the control group, only four experienced a hypomanic episode, and none met criteria for a BD diagnosis. They found that an earlier age of onset BD spectrum disorders predicted a higher likelihood of a conversion to BD I suggesting that the more severe BD psychopathology was linked to an earlier presentation of BD symptoms.

1.8 Comorbidities

Comorbidity is the norm in diagnosed BD populations. Disorders commonly comorbid with BD include anxiety (Albert, Rosso, Maina, & Bogetto, 2008; Kauer-Sant'Anna et al., 2007; Simon et al., 2004), substance abuse (Bauer, Altshuler, et al., 2005; Goodwin & Jamison, 2007), obsessive-compulsive disorder (Altindag, Yanik, &
Nebioglu, 2006; Maina, Albert, Pessina, & Bogetto, 2007), and social phobia (Kessler, Stang, Wittchen, Stein, & Walters, 1999). A study focusing on pre-pubertal and early adolescent BD participants found high comorbidity with Disruptive disorders (97 %), Oppositional Defiant Disorder (79 %), Conduct disorder (12 %), and anxiety disorders (23 %; Tillman et al., 2003).

1.9 Genetics and Neurophysiology

There is a strong genetic component to BD aetiology. Heritability has been found to be as high as 85 % in twin studies (McGuffin et al., 2003). Children and adolescents who have first-degree relatives with BD have a 5 to 10 % risk of developing BD (Maier, Höfgen, Zobel, & Rietschel, 2005). The specific genes mediating heritability are unknown, but BD has been linked to differences in genes involved with circadian rhythmicity (Kripke, Nievergelt, Joo, Shekhtman, & Kelsoe, 2009), cortical development (Aubry, Schwald, Ballmann, & Karege, 2009), the metabolism of calcium (Ross, Hughes, Kish, & Warsh, 2006), and cell survival (Aubry et al., 2009). Earlier studies identified several genes on chromosomes 2q, 3q, 4q 8q, 6q and 17q that may be involved in the development of BD (Dick et al., 2003; Roybal et al., 2007).

1.9.1 Brain structure.

Several studies have sought to identify brain structure abnormalities of those with clinical BD or trait vulnerability to BD. Strakowski et al. (2012) suggested that a possible neurological explanation of BD is that white matter connectivity between limbic brain structures (particularly the amygdala) and the ventral prefrontal cortex are disrupted. Studies have not only found that over time neurophysiological changes take place, and appear to worsen the more burdened one has been by their illness (Fusar-Poli, Howes, Bechdolf, & Borgwardt, 2012; Hajek et al., 2012; Hajek et al., 2013), but that neurological changes are observable from the presentation of the first manic
episode, as early as adolescence (Gogtay et al., 2007). Neurological studies have identified abnormal histopathology associated with a BD diagnosis in the regions of the amygdala (Almeida, Versace, Hassel, Kupfer, & Phillips, 2010; Altshuler et al., 2005; Blumberg et al., 2005; Kalmar et al., 2009; Usher, Leucht, Falkai, & Scherk, 2010), ventral striatum (Caseras, Lawrence, Murphy, Wise, & Phillips, 2013; Liu et al., 2012), anterior cingulate cortex (Liu et al., 2012; Matsuo et al., 2009), and the lateral prefrontal cortical system (Altshuler et al., 2008).

Usher et al. (2010) conducted a meta-analysis of studies that had performed magnetic resonance imaging (MRI) of the amygdala of patients diagnosed with BD and found that overall there was no difference in amygdala volume between scans of healthy controls and BD patients. However, they did find a positive correlation between amygdala volume and mean age in BD participants, concluding that increase in size of the amygdala over time is related to the course of the illness (Usher et al., 2010).

Hajek et al. (2013) conducted two studies at two separate centres (one in Canada and one in the Czech Republic). The first study examined the difference between the brain structures of 36 individuals with BD who also had a family history of BD (at least one parent having been diagnosed with BD), 50 unaffected individuals with a family history of BD, 19 young BD participants (with no family history of BD aged between 15 and 30 years), and 49 controls (no family history of BD and no BD symptoms). The groups were comparable on handedness, age, sex, and brain volume; however, the young BD participants (mean age 26.5 years) were older than controls (mean age 20.6 years). Modulated voxel-based morphometry was employed to compare grey matter volumes between groups. Results suggested that 10 brain regions differed between the control group and unaffected descendants of BD individuals. When the brains of control subjects were compared to individuals with a BD diagnosis (and a positive family
history), the number of regions that differed decreased to six. The volume of gray matter in the right inferior frontal gyrus (rIFG) also decreased with illness burden. The volume of the rIFG was also larger for both unaffected and affected BD offspring.

Hajek et al.’s (2013) second study examined the effects of long-term illness burden and pharmacological treatment by lithium, by comparing the brain structures of three different groups: those who had either never used lithium or had been using lithium short term (less than 3 months in total); those who had been taking lithium long term (at least 24 months); and controls matched on gender and age. Identified patterns of volume changes were similar to the first study. Individuals with short-term lithium treatment had smaller rIFG volumes than controls; however, individuals treated long-term with lithium showed no difference in the volume of their rIFG when compared to controls. The authors concluded that the volume of the rIFG was a likely indication of illness burden and that lithium either corrects or protects the brain from these changes over time (Hajek et al., 2013). This finding suggests that treatment can protect against the neurological changes that occur as a consequence of illness burden in BD.

1.9.2 Brain function.

Research has also investigated the brain function of individuals with BD or unaffected first-degree relatives and healthy controls (e.g., Jogia, Dima, Kumari, & Frangou, 2012; Kanske, Heissler, Schönfelder, Forneck, & Wessa, 2013; Kim et al., 2012; Otten & Meeter, 2015; Singh et al., 2014). These studies have employed functional MRI (fMRI), which measures neural activity and blood flow to brain regions whilst the participant is either resting or asked to complete a task (Andellini, Cannatà, Gazzellini, Bernardi, & Napolitano, 2015). The administered tasks typically involved paradigms of memory function (episodic or working), emotional processing and executive functioning, as well as tests that involve both emotional and cognitive
processing, including processes involved in reward (Piguet, Fodoulian, Aubry, Vuilleumier, & Houenou, 2015).

A meta-analysis of fMRI studies in BD adults, indicated that during emotional processing basal ganglia and the medial temporal lobe were observed to be overactive, whilst the inferior frontal cortex (frontal limbic system) was underactive (Chi-Hua, Suckling, Lennox, Ooi, & Bullmore, 2011). Cognitive processing was associated with underactivity in the inferior frontal cortex, as was a state of mania; and the amygdala was overactive during euthymia (Chi-Hua et al., 2011).

Similarly, Piguet, Fodoulian, Aubry, Vuilleumier, and Houenou (2015) reviewed 29 papers that had employed the fMRI technique to investigate brain function in BD and/or unaffected first-degree relatives to healthy controls (Piguet et al., 2015). Altered activation levels were observed in unaffected first-degree relatives when compared to controls. The areas associated with these activity changes have been associated with the pathophysiology of BD—specifically the medial prefrontal cortex, limbic areas (amygdala), parietal lobes and the IFG.

Piguet et al. compared research findings that related to emotional processing, executive functioning, reward, during periods of rest, and research that had observed the brain functioning of children and adolescents (siblings or offspring of people with BD). They concluded that, in individuals with BD and unaffected relatives, when it concerned emotional processing, the modulation of the amygdala and medial prefrontal cortex was different to that of healthy controls. They suggested that the interaction of emotional and cognitive processes could involve altered activation levels in areas associated with attention, specifically the IFG and parietal cortex. With regard to executive functioning, there were observed differences in the posterior cingulate cortex, basal ganglia and the ventrolateral and precuneus prefrontal cortex—areas already associated with BD and
cognitive processing. Response to reward was observed by higher activity levels in the medial orbitofrontal cortex in individuals with BD compared to controls, and unaffected relatives displayed higher activity levels in these areas in response to reward, punishment and rule reversal (Piguet et al., 2015). Overall findings regarding brain function during a resting phase observed lower connectivity between the anterior default mode-prefrontal network (associated with social cognition and theory of mind) and the fronto-occipital network (associated with visual processing and perception). Piguet et al. suggested that the connectivity of basal ganglia or limbic regions and frontal cortex varied non-specifically in unaffected relatives. Their overall findings concerning studies that had employed children or siblings of individuals with BD were that, like in the adult studies, similar regions were involved, specifically the amygdala (parahippocampal gyrus), pre and subgenual anterior cingulate cortex, and the ventrolateral inferior frontal gyrus and ventrolateral prefrontal cortex. However, they also noted that the way in which the brain was activated related to the emotion displayed in the testing procedure. Piguet et al. concluded that a possible vulnerability trait marker of BD is the involvement of the amygdala and, more generally, the limbic system.

1.9.3 Neurotransmitters and BD.

Overall, researchers proposed that the greater mood lability seen in BD is due to global abnormalities in the brain circuitry related to emotion regulation (de Almeida & Phillips, 2013). In addition to brain structure differences, neurohormones are also implicated in the presentation of BD. Hormones implicated in the core symptoms of BD include dopamine (Cousins, Butts, & Young, 2009; Lee et al., 2013), serotonin (Chou et al., 2010), and norepinephrine (Wiste, Arango, Ellis, Mann, & Underwood, 2008).
1.10 Psychotherapeutic Treatment for BD

There are presently four well known psychological treatment approaches in the management of BD. The first approach that will be described is Family Focussed Treatment (FFT: Miklowitz, 2004; Miklowitz et al., 2013). The second psychological treatment that will be described is Cognitive Behaviour Therapy (CBT) for BD (Lam, Hayward, Watkins, Wright, & Sham, 2005; Lam et al., 2003), an individual therapy. The third approach that will be described is Interpersonal and Social Rhythm Therapy (IPSRT; Frank, Swartz, & Kupfer, 2000). Finally, there is also evidence that manualised group psychoeducation treatment (Bauer, McBride, Chase, Sachs, & Shea, 1998; Bond & Anderson, 2015; Zaretsky, Rizvi, & Parikh, 2007) also reduces relapse, and so will be described at the end of this section.

FFT is based on the notion that the family environment can increase the cycles of BD by the way in which family members react to the symptomatic individual (Miklowitz, 2014). FFT involves 21 educational sessions in an outpatient program with five components: family assessment; educating the consumer about symptoms, mood, aetiology, relapse risk, and protection; communication enhancement between family and consumer; skills training in problem solving; and termination (Miklowitz, 2004). FFT delayed the recurrence of BD episodes (Miklowitz, George, Richards, Simoneau, & Suddath, 2003; Rea et al., 2003), decreased symptom severity (Miklowitz et al., 2003), and reduced the number of hospitalisations (Rea et al., 2003). A follow up study by Miklowitz, George, Richards, Simoneau, and Suddath (2008) observed quicker recovery rates from depressive episodes and less severe depressive episodes two years post-treatment. Miklowitz (2014) stated that there are sub-groups of patients who do not fare well following FFT, and they are usually individuals who do not accept a BD diagnosis, or those who do not have any contact with family members.
CBT for BD is based on a diathesis-stress model which aims to empower participants by encouraging the use of medication whilst participating in psychotherapy (Lam et al., 2005; Lam, Jones, Hayward, & Bright, 1999). Psychotherapy involves educating the participant on prodromes (by examining previous triggers from past episodes), behavioural techniques (for example, monitoring mood, food intake and sleep, and learning to plan and relax), and dealing with unhelpful automatic thoughts in order to prevent relapse (Lam et al., 1999). Treatment generally consists of approximately 20 sessions, divided into three stages. The first stage focuses on education on BD and treatments (both pharmacological and psychological), the second focuses on cognitive approaches, and the final stage focuses on consolidating the skills and behavioural changes required for minimisation of relapse (Lam et al., 1999). A one-year follow-up study found that individuals diagnosed with BD who participated in CBT for BD and were compliant with their medications, fared significantly better than individuals who managed with medication alone (Lam et al., 2003). Also, those that participated in CBT treatment reported, and demonstrated, significantly better coping when managing manic and depressive prodromes (Lam et al., 2003). A two-year follow-up study demonstrated that individuals who participated in the CBT for BD study continued to have fewer days in bipolar-related episodes, reported equal or better mood ratings, social functioning, and coping with prodromes (Lam et al., 2005). However, findings indicated that the strongest effects were observed six months post-treatment as improvements two years post-treatment were weaker in strength; hence the need for maintenance or booster sessions (Lam et al., 2005).

IPSRT is based on a model of interpersonal psychotherapy combined with training in self-regulation of sleep-wake cycles and daily routines (Miklowitz, 2014). IPSRT (Frank et al., 2000) involves four main phases—initial, intermediate,
preventative, and termination— and ideally lasts approximately two years (Frank et al., 2005). The initial phase involves a detailed history, psychoeducation about BD and information about the social rhythm model. The intermediate phase involves weekly contact with the therapist where both parties mutually develop strategies to cope with symptoms and regulate daily rhythms, particularly focusing on decreasing activities that may dysregulate circadian rhythms. The preventative phase involves monthly contact with the therapist and is designed to prevent relapse. Termination is a gradual process where the therapist reviews the patient’s successes, vulnerabilities, and strategies for when stressful life events will occur. Research investigating the efficacy of IPSRT two years after treatment found that increased time between affective episodes and more regularity in social rhythms (Frank et al., 2005). Furthermore IPSRT improved individuals’ occupational functioning significantly more than psychoeducation and support alone (Frank et al., 2008).

Group psychoeducation sessions aim to improve awareness of illness through discussions about beliefs and attitudes, recognising early signs of relapse, the importance of treatment adherence, encouraging regularity of lifestyle, behavioural interventions and the assignment of homework between sessions (Colom et al., 2003; Colom et al., 2005). Group psychoeducation reduced relapse rate and hospitalisations, and also individuals who participated in this mode of therapy had stable serum-lithium levels (Colom et al., 2003; Colom et al., 2005). It is a cost-effective addition to pharmacotherapy in individuals who are in remission (Parikh et al., 2012). Furthermore, brief psychoeducation has been found to be as effective as FFT (Miklowitz et al., 2014).

1.11 Summary of Chapter 1

A clinical condition matching BD has been documented over the last two and a half centuries. This chapter reviewed literature indicating that BD is a highly heritable
disorder of mood. The literature suggests that, in some cases, symptoms are observed as early as childhood and can persist throughout the lifetime. The majority of current researchers and clinicians assert that BD occurs on a spectrum with BD considered the extreme end of the illness spectrum. BD can also occur in conjunction with other disorders, most often anxiety and substance use disorders. BD can be treated and managed. Neuroanatomy and neurohormone regulation differences observed in BD have shed some light on the mechanisms involved in the onset of BD, and medications (namely lithium) may protect or correct brain changes from illness burden. Psychotherapies, including FFT, CBT, IPSRT and group psychoeducation, may also be effective in decreasing the BD illness burden.
2.0 Relationship between Personality, Motivational Systems and Vulnerability to BD

2.1 Overview of Chapter 2

This chapter introduces traits associated with vulnerability to BD. The chapter will begin with a brief discussion of the General Behavior Inventory (GBI), the tool most widely used to measure BD vulnerability. A general definition of the term *trait* will be offered, then the constructs of personality and BIS/BAS will be introduced. Finally, studies investigating the associations between personality and BIS/BAS with BD will be reviewed.

2.2 The General Behaviour Inventory as a Specific, Two-Dimensional Measure of Vulnerability to BD

The General Behavior Inventory (GBI; Depue et al., 1981) was primarily developed to identify individuals in the general population who are at risk of bipolar or unipolar mood disorders (Depue et al., 1989). It has high prognostic power and specificity, sufficient sensitivity, and does an adequate job of selecting individuals who are at risk of affective disorders in a nonclinical population for the purpose of research (Depue et al., 1989; Klein, Dickstein, Taylor, & Harding, 1989). Furthermore, the GBI captures the two dimensions (bipolarity) of vulnerability to BD (specifically, depression-proneness or trait-depression and mania-proneness or trait-mania; Youngstrom, Murray, Johnson, & Findling, 2013). Both states of BD, mania and depression, are viewed as two contrasting biopsychosocial states, not opposite mood poles, that measure the BD phenotype (Youngstrom et al., 2013). However, a limitation of the GBI is that, because of its length (73 densely-worded items), completing it is burdensome on the participant, particularly when participants are required to complete a battery of questionnaires (Youngstrom, Frazier, Demeter, Calabrese, & Findling, 2008;
Youngstrom et al., 2013). The present project created a short form of the GBI (sGBI, described in the Method section; Poulios, Murray, & Bullock, 2010) and its psychometric properties were investigated prior to use in hypothesis testing.

2.3 Personality Traits

The first aim of the present study was to explore the trait correlates of vulnerability to BD, specifically personality and motivational traits. The concept of trait emerged from the covariance produced between patterns of scales (or as measured by stable and enduring behaviour that is assessed by rating, question items, or scales or other observable measures) with, or separate to, one another (Watson, Clark, & Harkness, 1994). This covariance forms the structure of personality from which the individual trait emerges (Watson et al., 1994). Personality is often described as the elements which make up an individual, that is, the building blocks that make them think and act the way they do (Miserandino, 2012). Relevant constructs include cognition, genetics, intrapsychic aspects, neurobiology, self and identity, and—most important to the present thesis—traits (Miserandino, 2012).

2.4 The Relationship between Personality and Psychopathology

As described by Widiger (2011), personality may relate to psychopathology in three ways. The first framework regards personality as bi-directionally related to psychopathology, called a pathoplastic relationship (Widiger, 2011). For example, the higher a person scores on Neuroticism, the more vulnerable they are to receiving a depression diagnosis in the future. The second framework posits that personality and psychopathology share a common cause (etiology), for example, Neuroticism and depression may be due to the same entity. Finally, the third framework is that personality causes psychopathology, this relationship is also bidirectional (Widiger, 2011), and an example would be elevated Neuroticism could be a causal explanation for
the onset of a depressive episode. Discussing these three different theoretical approaches to psychopathology and personality in detail is beyond the scope of the present project. However, it is important to introduce these conceptual frameworks as they provide different methods of investigating vulnerability to BD.

2.4.1 Personality and BD.

Personality factors may predict vulnerability to BD (Christensen & Kessing, 2006; Goodwin & Jamison, 2007; Lozano & Johnson, 2001; Murray, Goldstone, & Cunningham, 2007; Quilty, Pelletier, DeYoung, & Bagby, 2013; Solomon, Shea, Leon, & Mueller, 1996). Identifying a cluster of personality traits associated with BD would aid therapists and clinicians in diagnosis and subsequent treatment (Quilty et al., 2013). A complexity that then arises is lack of consensus about the structure of personality (Carver & Connor-Smith, 2010). Some theorists have proposed two factors (Jenkins, 1950), others three factors (Eysenck, 1967), five factors (Digman, 1990; Goldberg, 1990; McCrae & Costa, 1992; Zuckerman, Kuhlman, Thornquist, & Kiers, 1991), and even six factors (Jackson, Ashton, & Tomes, 1996; Jackson, Paunonen, Fraboni, & Goffin, 1996). However, the most widely used model is the Five-Factor Model (FFM), which the present study will focus on.

The FFM is commonly measured by the NEO-PI-R which measures the traits Neuroticism (N), Extraversion (E), Openness to Experience (O), Agreeableness (A), and Conscientiousness (C; McCrae & Costa, 1991). The N scale taps the degree of vulnerability to psychological distress, and consists of six facets: hostility, anxiety, depression, impulsivity, self-consciousness, and vulnerability. The E scale measures the intensity and amount of energy one invests in the social world, and consists of six facets: activity, assertiveness, excitement seeking, gregariousness, positive emotions and warmth. The O scale measures the degree to which one seeks and appreciates
experience and is measured six facets: *actions, aesthetics, fantasy, feelings, ideas* and *values*. The A scale measures the interactions one prefers, and consists of six facets: *altruism, compliance, modesty, straight-forwardness, tender-mindedness* and *trust*. Finally, the C scale measures level of organisation, control, motivation and persistence in attaining goals, and also consists of six facets: *achievement striving, competence, deliberation, dutifulness, order* and *self-discipline* (McCrae & Costa, 1992).

Irrespective of gender and age, particular personality profiles are associated with recurrence of depressive symptoms (Steunenberg, Braam, Beekman, Deeg, & Kerkho, 2009), can predict trait-depression in student samples (Lozano & Johnson, 2001; Murray et al., 2007) and those diagnosed with BD (Kim, Joo, Kim, Lim, & Kim, 2011), and can predict trait-mania in psychiatric patients (Quilty, Sellbom, Tackett, & Bagby, 2009). Research comparing personality between controls and individuals with BD found that, even during remission, individuals with BD had higher N scores than the never-ill controls (Solomon et al., 1996). Personality traits can also predict the severity of BD, as noted by Koszewska and Rybakowski (2008) who observed higher N scores in individuals with BD who experience mixed states. The following paragraphs will describe, in more depth, specific studies that influenced the present study and subsequent hypotheses.

Earlier work comparing personality correlates of UD and BD suggest that those with BD experience both negative and positive emotions more intensely than UD participants. Bagby et al. (1996) used the NEO-PI-R and the NEO-PI to examine differences in personality between recovered UD participants (*n* = 74), euthymic BD participants (*n* = 34), and the combined group (*n* = 108). Analyses found that BD patients scored higher on O, with the biggest difference between the two groups coming from the *feelings* facet. They also found that BD participants were more able to
experience positive affect then UD patients, as the facet *positive emotions* predicted patient group. However, when investigating the correlations between personality and diagnosis (BD, UD or combined group), no significant correlations were found between any of the personality scales or facets and the scores obtained by the BD group. It was only when the BD group scores were combined with the UD group scores did significant relationships appear, namely between: E and the combined group (a negative weak correlation of -.23); the N facet of anxiety and the combined group (a positive weak correlation of .21); the N facet of self-conscientiousness and the combined group (a positive moderate correlation of .32); the E facet of excitement seeking and the combined group (a negative weak correlation of -.20); and between the O facet of actions and the combined group (a negative weak correlation of -.25).

Continuing Bagby et al.’s (1996) work, Lozano and Johnson (2001) employed a short version of the NEO-PI-R (the NEO-Five Factor Inventory; NEO-FFI; Costa & McCrae, 1992), to investigate manic and depressive symptoms of BD. Participants were 39 individuals diagnosed with BD I, using the Structured Clinical Interview for DSM-IV (SCID-I; First, Spitzer, Gibbon, & Williams, 2012). Using regression analyses, Lozano and Johnson (2001) found that N predicted depressive symptoms, and that high C (specifically, achievement striving) predicted symptoms of mania.

Röttig, Röttig, Brieger, and Marneros (2007) investigated the difference in temperament and personality between BD patients with and without a history of mixed episodes. They measured personality with the NEO-FFI and temperament using the Temperament Evaluation of Memphis, Pisa, Paris and San Diego Auto-questionnaire (TEMPS-A; Akiskal et al., 2005). Five temperaments (anxious, cyclothymic, depressive, hyperthymic, and irritable) are investigated in the subscales of the TEMPS-A. Although no significant differences in personality emerged, participants who
experienced mixed episodes scored significantly higher on the depressive, cyclothymic, anxious and irritable subscales (Röttig et al., 2007). This study did not have a control group, thereby preventing the comparison of reported personality traits of BD and controls (Röttig et al., 2007).

Murray et al. (2007) investigated FFM correlates of trait vulnerability to BD (as trait-depression, trait mania or trait-bipolarity) measured on the GBI (Depue et al., 1989) in a student sample \((n = 176)\). They found positive, large and statistically significant relationships between: N and trait-depression \((r = .70)\); and N and trait-mania \((r = .51)\). They also found significant relationships between: E and trait-depression (positive in direction and weak in size: \(r = .17)\); A and trait-depression (negative in direction and weak in size: \(r = -.24)\); A and trait-mania (negative in direction and moderate in size: \(r = -.36)\); C and trait-depression (negative in direction and moderate in size: \(r = -.40)\); and C and trait-mania (negative in direction and moderate in size: \(r = -.36)\). When they investigated personality as a predictor of predisposition to BD they found that N, E, and A predicted bipolar vulnerability.

Murray et al. (2007) analysed the impact of the personality traits on two models of BD. The first was a two-dimensional model looking at the relationship of personality traits with trait-mania and trait-depression. The second was a one-dimensional model combining trait-mania and trait-depression into one factor, trait-bipolarity. The authors concluded that the one-dimensional model was sufficient to describe the interaction between the five factors of personality and bipolar vulnerability; however, the two-dimensional model provided more specificity about how each personality trait affected the two traits of bipolarity. In the two-dimensional model, the tendency for mania or depression were not independent of each other, but rather the tendency for depression was part of the manic aetiology (Murray et al., 2007). Overall, N directly affected
predisposition to depression, E predicted predisposition to mania, and A negatively related to mania. Murray et al. also concluded that N did not have a direct effect on mania vulnerability, but rather influenced it indirectly through vulnerability to depression. However, it should be noted that the sample was not screened for past mental health issues. Thus, the authors could only assume that the incidence of mental health in their sample was similar to that observed in the general public. They concluded that a two-dimensional model was more useful in understanding the shared dimensions of mania and depression in BD (Murray et al., 2007).

In an attempt to extend Murray et al.’s (2007) one- and two-dimensional analyses of personality in BD, Quilty et al. (2009) analysed responses of 370 psychiatric patients who completed the NEO-PI-R and the Minnesota Multiphasic Personality Inventory - 2 (MMPI-2; Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 2001). Scores from the Clinical Scale 9 (hypomania) were used to measure Mania, and scores from both Clinical Scale 2 (low positive emotions) and Clinical Scale 7 (dysfunctional negative emotions) were used to measure Depression in the two-dimensional model (containing both trait-depression and trait-mania). The sums of the three clinical scales described above were used to measure Bipolarity in the one-dimensional model. Correlation analyses revealed significant positive relationships between N and Depression (strong in size: \( r = .60 \)); N and Mania (weak in size: \( r = .24 \)); E and Mania (weak in size: \( r = .18 \)); and O and Mania (weak in size: \( r = .19 \)). Significant negative relationships existed between E and Depression (moderate in size: \( r = -.46 \)); O and Depression (weak in size: \( r = -.18 \)); A and Depression (weak in size: \( r = -.13 \)); C and Depression (moderate in size: \( r = -.40 \)); A and Mania (moderate in size: \( r = -.49 \)); and C and Mania (weak in size: \( r = -.21 \)). Quilty et al. (2009) found that N and A (negative) predicted a one-dimensional model of Bipolarity. In the two-dimensional model, Mania
was predicted by N, E and (negative) A, whereas Depression was predicted by N and (negative) E. Like Murray et al. (2007), Quilty et al. (2009) concluded that a two-dimensional model of BD better explained how personality traits interacted with BD symptoms; however, their findings differed in that Quilty et al. found that N directly predicted Hypo/mania scores.

Further work investigated the difference between UD \( (n = 139) \) and BD \( (n = 136) \) with respect to a hierarchical personality structure of the FFM (Quilty et al., 2013). Their aim was not only to identify if mood disorders (UD versus BD) produced different hierarchical personality clusters, but also if severity of symptoms (manic versus depressive) differed. To date, this has been the sole study to analyse the aspect level of personality traits in BD and UD. Aspects are accurate characterisations of each personality domain, that discriminate between the five personality domains of the FFM (DeYoung, Quilty, & Peterson, 2007): volatility and withdrawal for N, enthusiasm and assertiveness for E, intellect and openness for O, compassion and politeness for A, and industriousness and orderliness for C. With respect to mood disorder diagnosis, regression analyses revealed that, unlike previous studies (Murray et al., 2007; Quilty et al., 2009), N did not predict mood disorder diagnosis; however, significant prediction of BD or UD mood disorder diagnosis was made at the aspect level by increased volatility and decreased withdrawal, furthermore the facet of impulsiveness was a significant marker between a BD and UD diagnosis (Quilty et al., 2013). All personality domains, except C, significantly contributed to the prediction of mood disorder diagnosis. Components of C (namely industriousness and deliberation) did, however, contribute to mood disorder diagnosis. As previously mentioned E contributed at all levels from the domain, to the aspect of enthusiasm and the facets of assertiveness, activity, and positive emotions. O and A also contributed to the prediction of mood disorder diagnosis.
diagnosis, as did the facet of compassion (under the domain of A). Simultaneous prediction of mood disorder diagnosis by all five domains produced a significant model, and higher levels of E predicted a BD diagnosis (Quilty et al., 2013).

Quilty et al. (2013) also investigated personality and symptom severity. Positive predictors of depressive symptoms were N, withdrawal (N domain), anxiety (N facet), depression (N facet), vulnerability (N facet), modesty (A facet), and order (C facet). Significant negative predictors of depressive symptoms included E, enthusiasm (E aspect), positive emotions (E facet), intellect (O aspect), actions (O facet), A, compassion (A aspect), trust (A facet), modesty (A facet), C, industriousness (C aspect), order (C facet), and self-discipline (C facet; Quilty et al., 2013). Simultaneous prediction of depression severity by all five domains produced a significant model, with N and E emerging as significant predictors. Unlike depression severity, only three lower facets and one aspect predicted mania. Mania severity was positively predicted by volatility (N aspect), angry hostility (N facet), and activity (E facet), and was negatively predicted by deliberation (C facet). Simultaneous prediction of mania severity by all five domains did not produce a significant model. However, as this study excluded individuals with extreme mania, it is not known how the full range of mania would have altered these findings. Quilty et al.’s (2013) study highlighted the importance of assessing all levels of personality, from the domain down to the facet level.

A related study by Kim et al. (2011) examined the five-factor model of personality in BD, specifically in individuals who had been hospitalised for different types of BD episodes (depressive, manic or mixed). In the cross-sectional study, individuals with a diagnosis of BD I \( (N = 83) \) completed the NEO-PI-R. Regression analyses indicated that N positively, and E and O negatively, related to number of hospitalisations due to a depressive episode; and that C positively related to
hospitalisations for mixed episode. Unlike Lozano and Johnson (2001), no personality traits related to hospitalisations for manic episodes; suggesting that admission to hospital for a manic episode was independent of personality. Although personality is generally accepted as stable in adulthood (Costa, Bagby, Herbst, & McCrae, 2005), Kim et al. (2011) acknowledged the necessity of a longitudinal study to investigate personality before, during and after hospitalisation and treatment, to assess the pathoplastic effect of psychopathology on personality. Despite this limitation, they concluded that the combination of N (positive), E (negative), and O (negative) increased the likelihood of an individual with BD having depressive morbidity. Kim et al. also compared individuals who experienced an affective switch from mania to depression without euthymia (ASWE) and non-ASWE participants. Their hypothesis, that ASWE participants would be more emotionally dysregulated and thus more prone to a depressive state than non-ASWE participants, was initially supported as they found higher N scores in the ASWE group. However, this particular finding did not remain significant after a Bonferroni correction. Thus, unlike the two previous studies described (Quilty et al., 2013; Quilty et al., 2009), Kim et al. did not find significant relationships between the FFM of personality and mania.

In summary, previous research has consistently found N to be the strongest correlate and predictor of both depression and mania scores. As mentioned above, previous research has also identified weak to moderate relationships between the other personality scales of E (negative), C (negative), and A (negative). Studies have shown that in addition to N, the personality scales of A and E have also been found to predict mania. Not only have personality variables been found to relate and predict symptoms and scores related to BD, but they have also been found to predict diagnosis, severity of symptoms, and hospitalisations. As personality structure has been a focus of research
investigating the phenomenology of BD, the present project also sought to investigate these previously determined associations.

2.5 BIS/BAS and BD

The behavioural activation system (BAS; Hofmann & Meyer, 2006) and the behavioural inhibition system (BIS; Meyer, Johnson, & Carver, 1999) are neurobiological systems of motivation (Alloy et al., 2008; Elliot & Thrash, 2002; Gray, 1970). Stimulation of the BAS has been associated with increases in goal-oriented behaviour (Harmon-Jones et al., 2002; Johnson et al., 2000), positive affect (Giovanelli, Hoerger, Johnson, & Gruber, 2013), impulsivity (Alloy et al., 2009), and sociability (Windsor, Anstey, Butterworth, & Rodgers, 2008), behaviours that are abnormal in manic prodromes or episodes (Johnson, Winters, & Meyer, 2006). BIS activation is associated with behavioural inhibition and withdrawal, and behaviours linked to depression (Alloy et al., 2008; Meyer, Johnson, & Winters, 2001). Thus BD may be associated with increased peaks and troughs of BAS (Urošević, Abramson, Harmon-Jones, & Alloy, 2008).

The most commonly used measure of BIS and BAS is the 20-item self-report BIS/BAS scales developed by Carver and White (1994). The BIS/BAS scales comprise four subscales, one measuring BIS and three measuring different aspects of BAS. The BIS scale was designed to measure sensitivity to punishment through behavioural reactions. The BAS scale includes three subscales: BAS Drive, BAS Fun Seeking, and BAS Reward Responsiveness. BAS Drive measures persistence in the pursuit of goals; BAS Fun Seeking measures desires for novel rewards and willingness to impulsively get involved in an event that may be rewarding; and BAS Reward Responsiveness scale measures positive affect in response to reward (Carver & White, 1994).
Scores on hypomania scales are significantly related to BAS and BIS subscales. Specifically, BAS Fun Seeking and BAS Reward Responsiveness uniquely predicted hypomania risk scores, as measured by the Hypomanic Personality Scale (Jones, Shams, & Liversidge, 2007). Meyer et al. (1999) were the first to investigate the relationship between BIS/BAS sensitivity and risk to BD in a general population sample, classified as high risk to BD \( (n = 63) \) and low risk to BD \( (n = 294) \), based on their scores on the GBI. Significant positive, moderate correlations between active hypomanic symptoms (as measured by the Internal States Scale [ISS; Bauer et al., 1991]) and all BAS subscales (as well as the BAS Total score) were found in those at risk of BD \( (r \text{ ranging from .33 to .49}) \). In the same sample of participants at risk of BD, depression symptoms (as measured by the ISS) positively correlated with BIS \( (r = .32) \) and negatively correlated with each of the BAS subscales (including BAS Total), and the strength of these relationships varied from moderate to strong \( (r \text{ ranging from -.39 to -.53}) \). In the low-risk group, there were no significant correlations between depression symptoms and any of the BIS/BAS scales. With regard to hypomania scores in the low-risk group, significant positive correlations were found with BAS Total \( (r = .20) \), BAS Fun Seeking \( (r = .20) \), and BAS Drive \( (r = .16) \); however, these relationships were weak.

Meyer et al. then controlled for current mania and depressive symptoms. In the high-risk group there were significant positive, correlations between all BAS subscales and hypo/mania scores derived from the GBI \( (r = .29 \text{ for BAS Reward Responsiveness}; \ r = .35 \text{ for BAS Drive}; \ r = \text{BAS Fun Seeking}; \text{ and } r = .42 \text{ for BAS Total}) \). BIS and hypo/mania scores from the GBI did not produce any significant correlations for both the high-risk and low-risk groups. No significant correlations existed between any of the BIS/BAS scales and GBI-depression scores in the high-risk group. In the low-risk group, the sole significant relationship for GBI hypo/mania scores was with BAS Fun
Seeking (positive in size and weak in strength: \( r = .26 \)). Also in the low-risk group, only BIS \( (r = .17) \) and BAS Fun Seeking \( (r = .12) \) positively correlated with GBI-depression scores (Meyer et al., 1999) when symptom severity was controlled for, however as noted, these relationships were weak in strength.

Through regression analyses Meyer et al. demonstrated that BAS Total was a significant predictor of current mania symptoms for the high-risk group, but upon more detailed examination, BAS Fun Seeking was the only BAS subscale that significantly predicted current mania symptoms. Further, BIS positively and BAS Reward Responsiveness negatively predicted current depression symptoms. Meyer et al. also reported that for the low-risk group, similar to the results of the high-risk group, BAS Total, especially BAS Fun Seeking, positively predicted mania symptoms, and concurrent depression was positively predicted by BIS and negatively predicted by BAS Reward Responsiveness; however, effect sizes were much smaller. The authors concluded that manic and depressive symptoms could be explained by trait differences in sensitivity to stimuli, as are measured on the BIS/BAS scales. These findings suggest that elevated levels of BAS may indicate vulnerability to BD, as BAS remained a predictor of lifetime mania vulnerability when controlling for current affect. Thus, BIS/BAS subscales can be viewed as motivation traits associated with vulnerability to BD.

Extending the work of Meyer et al. (1999), Meyer, Johnson, and Winters (2001) examined the relationship between BIS and BAS sensitivity, using the BIS/BAS scales, in 59 individuals diagnosed with BD I. Their analysis of cross-sectional data revealed that only BIS positively correlated \( (r = .45) \) with depression symptom severity (as measured by the Modified Hamilton Revised Scale for Depression; MHRSD). Unlike Meyer et al. (1999), neither total score nor any BAS subscale correlated with depressive
or mania symptom severity (as measured by the Bech-Rafaelson Mania Scale, BRMS; Meyer et al., 2001). Meyer et al. attributed this finding to a difference in methodology where clinicians rated symptoms, as opposed to the self-report data used in their earlier work. Depressive symptoms fluctuated over time and, when analyses also included follow-up symptom severity scores, BIS levels also fluctuated. Thus, BIS was likely not an operable vulnerability factor, but instead functioned as state-dependent current depressive symptomatology (Meyer et al., 2001). Similarly, BAS scales did not correlate with concurrent manic symptomatology in the BD I sample, despite a significant correlation between increased manic symptoms over time and BAS Total ($r = .35$) and Reward Responsiveness ($r = .35$) subscale scores. The researchers argued that BAS subscales may be more stable over time than the BIS scale, consistent with conceptualising high BAS as a vulnerability factor for mania (Meyer et al., 2001). Meyer et al. stated that as the BIS/BAS scales were initially developed using a student sample, a possible reason for their null findings was the sample used (clinical population and not undergraduate students). The researchers also pointed out that there is content overlap in the items measuring mania and those measuring BAS Fun Seeking (Meyer et al., 2001), and this could explain why previous work (Meyer et al., 1999) found such strong correlations with BAS Fun Seeking in a sample of at-risk students.

Alloy et al. (2012) extended Meyer et al.’s (1999) work by using GBI scores to identify high-risk secondary school students and monitoring them over a period of four and a half years. Findings specifically concerned with BIS/BAS sensitivity in BD were as follows: high scores on the BAS Total were predictive of future BD II diagnosis with the BAS Fun Seeking scale as the only significant predictor of conversion to a BD II diagnosis. Similarly, BAS Fun Seeking was the only BAS scale to significantly predict progression to a BD I diagnosis; however, when family history was included as a
Sleep, Coping, and Vulnerability to Bipolar Disorder

covariate, BAS no longer significantly predicted progression to BD I. When the interaction between BAS Total and BIS was also considered in regression analyses (that predicted progression to BD I), the interaction was significant. Results indicated that an individual was more likely to progress to a BD I diagnosis when they had high GBI scores and high BAS and BIS sensitivity scores. Like Meyer et al. (1999) they concluded that a vulnerability to the bipolar spectrum is high BAS sensitivity, particularly BAS Fun Seeking (Alloy et al., 2012). The authors also proposed that a combination of high BAS and high BIS sensitivity might be predictive of a more severe form of BD that is symptomatic at an earlier age (Alloy et al., 2012).

In summary, research has generally found that BAS Fun Seeking both correlates and predicts BD symptom severity and progression along the BD spectrum, from low risk to BD to a BD I diagnosis. Research has also found that BIS correlates with and predicts depression scores. Although the majority of research described above did not use a clinical sample, the motivation traits of BIS and BAS (particularly BAS Fun Seeking) have consistently been associated with an increase in vulnerability to BD and thus, will also be investigated in the present project.

### 2.5.1 BIS/BAS, emotion regulation and impulsivity.

Research using undergraduate samples has also investigated difficulties regulating emotion and impulse control as correlates of vulnerability to BD. Markarian, Pickett, Deveson, and Kanona (2013) investigated models that included interactions between BIS/BAS sensitivity, emotion regulation difficulties, and sleep quality with depression, anxiety, and stress. They collected survey data from 459 undergraduate students ($n = 96$ males) and employed the BIS/BAS scale (higher scores indicative of greater sensitivity to stimuli), the Difficulties in Emotion Regulation Scale (DERS, high scores indicative of more difficulty with emotion regulation), the Pittsburgh Sleep
Quality Index (PSQI, scores greater than or equal to five indicative of poor sleep quality) and the Depression, Anxiety and Stress Scale (DASS, higher scores indicative of more severe symptoms). BAS Fun Seeking and BAS Drive did not correlate with any other variables, and were removed from further analyses. However, BAS Reward Responsiveness significantly correlated with DERS, PSQI and DASS-depression scores ($r = -.14$), and all correlations were negative in direction. That is, higher BAS sensitivity was associated with less difficulty regulating emotions, better sleep quality, and less severe depressive symptomatology. BIS significantly and positively correlated with DERS, PSQI, DASS-depression, DASS-anxiety and DASS-stress scores, thus BIS sensitivity is associated with difficulty regulating emotions, poorer sleep quality and higher symptom severity (for anxiety, stress, and depression).

Furthermore, when their proposed model was analysed according to sleep quality (good versus poor), the model for poor sleep quality contained stronger path loadings than the good sleep quality model, and as direct pathways between DERS scores and depression and anxiety were non-invariant, the authors suggested that sleep quality played a moderating role between the model relating emotion regulation difficulties and depression and anxiety symptoms. Markarian et al. (2013) concluded that BIS and BAS Reward Responsiveness indirectly affected depression, anxiety and stress scores via emotion regulation difficulty and were moderated by sleep quality. Thus, a more severe state of mental ill-health (depression, anxiety and stress symptoms) may occur when one is more sensitive to aversive stimuli (high BIS), less sensitive to reward (low BAS), has greater difficulty regulating emotion and also suffers from poor quality sleep. Although this study focussed on depression, it is one of the few studies investigating BIS/BAS and emotion in the context of sleep quality. Furthermore, although the study did not focus on BD, findings could suggest that difficulties
regulating emotion increase vulnerability to mood problems, and this vulnerability could be moderated by sleep.

BAS Fun Seeking has been used to assess impulsivity (Giovanelli et al., 2013). Impulsivity has commonly been defined as responding to a stimulus or event without regard for the consequences or the opportunity for reflection, suggesting the involvement of both attentional and pre-attentional functioning (planning and decision making), and physiological processes involved in action initiation (motor inhibition; Swann, 2010). Giovanelli et al. (2013) conducted two cross-sectional studies using college samples, and one of the studies (N = 823) examined the interactions between current or future symptoms of mania scores and measures of personality on the Hypomanic Personality Scale (HPS; Eckblad & Chapman, 1986). The HPS is a self-report scale developed to gauge the risk of a manic episode occurring in the future. One of the measures used to assess impulsivity was the BAS Fun Seeking subscale of the BIS/BAS scale (Carver & White, 1994). As previously mentioned, BAS Fun Seeking measures the drive to pursue rewarding new experiences without regard for the consequences. Correlation analyses revealed significant positive, weak- to moderate-sized relationships between BAS Fun Seeking and scores on the HPS. Giovanelli and colleagues asserted that an important factor in assessing risk of mania is impulsivity, as mania is defined by impulsive behaviours (Giovanelli et al., 2013).

2.6 Summary of Chapter 2

This chapter began with a brief description of the GBI, a tool that measures vulnerability to BD using items that capture BD symptoms. The GBI is also able to produce scores which can be used along a spectrum of vulnerability. Two known correlates of vulnerability to BD were described—the personality traits of the FFM and motivation traits, specifically the BIS/BAS motivational systems. The chapter defined
personality, the term trait, and putative relationships between personality and psychopathology. Empirical research reviewed suggests that personality, as conceptualised using the FFM, indicated that N correlates highly with symptoms and vulnerability to BD; however, N is a measure of general psychological distress, and so proves to be a non-specific marker of vulnerability to BD. Findings regarding BIS/BAS have been mixed. Emerging research indicates that the subscale of BAS Fun Seeking is a consistent correlate of vulnerability to BD.
3.0 Sleep

3.1 Overview of Chapter 3

This chapter begins by introducing normal sleep including a description of the stages of sleep, sleep across the lifespan, neurochemical regulation during the different phases of sleep, and findings from sleep deprivation studies. The chapter then introduces the distinction between state- and trait-like sleep, and its importance to the present study. The second part of this chapter will then describe the prevalence and possible mechanisms involved in the disruption of sleep in BD. Sleep deprivation as a possible treatment of BD depression will be briefly described. Finally, a review of research investigating chronotype preference in BD is presented.

3.2 Normal Sleep

All animal species that have been studied to date enter a sleep-like state, suggesting sleep plays an evolutionarily important function (Peigneux, Urbain, & Schmitz, 2012). The exact function of sleep remains unknown (Krystal, Thakur, & Roth, 2008); however, it is believed to be restorative (Benington & Craig Heller, 1995; Maquet, 1995). Sleep may strengthen neurohormone regulation (Benington & Craig Heller, 1995), somatic growth, and immune function (Maquet, 1995). Many definitions of sleep have been put forward; however, the current project adopts the following definition: sleep is a reversible behavioural state during which voluntary movements and response to stimulation are significantly reduced (Fuller, Gooley, & Saper, 2006).

Sleep is governed by a homeostatic process with the sleep drive increasing the longer an individual remains awake, and decreasing during sleep (Fuller et al., 2006). Sleep architecture refers to the organisation and structure of sleep (Colten & Altevogt, 2006). Once asleep, there are four stages of sleep: three Non-Rapid Eye Movement (NREM) sleep stages and REM sleep. It should also be noted here that sleep takes on a
cyclical nature and latency to REM—the time taken to complete a cycle—is measured by the time it takes an individual to pass through all of the NREM sleep stages and begin REM sleep (Dijk & Lazar, 2012). The different stages of sleep are classified on the basis of electroencephalogram (EEG) data, which measures the electrical activity of post-synaptic potentials of neurons in the brain; an electrooculography (EOG), which records voltage changes produced by eye movement; and an electromyogram (EMG), which measures muscle activity (Iber, Ancoli-Israel, Chesson, & Quan, 2007).

A typical adult night’s sleep is on average eight hours long, and during this time four to five NREM-REM cycles occur (Dijk & Lazar, 2012). Although dreaming occurs primarily in REM, dreaming also occurs during all stages of sleep, and the deeper sleep one experiences (going through all NREM stages to REM), the more vivid the dreams become (De Koninck, 2012).

3.2.1 Physiological observations during wakefulness.

During wakefulness, EEG patterns are desynchronised, with high frequency beta (β) waves ranging between 14 and 40 hertz (Hz), a characteristically low amplitude of voltage ranging between 10 and 30 µV, and EMG recordings displaying high muscle tonus (Peigneux et al., 2012). When in a relaxed state, on the verge of sleep, EEG patterns display synchronous alpha (α) waves, ranging between 8 and 12 Hz, which are larger in amplitude ranging between 20 and 40 µV (Peigneux et al., 2012).

3.2.2 Physiological observations during NREM sleep.

Although the average sleeper will report that it took them 15 to 30 minutes to fall asleep (Majer et al., 2007), the average sleeper will take approximately 10 to 15 minutes to begin sleeping once the lights are out (Carskadon & Dement, 1996). As one progresses through lighter non-REM sleep stages (N1 and N2) to deep sleep (N3), also known as Slow Wave Sleep (SWS), breathing becomes more regular, there is a decrease
in muscle tonus, and EEG patterns increase in amplitude (Peigneux et al., 2012). Following the work of Peigneux (2012), the three sub-stages of NREM sleep are described below.

Stage N1 of NREM is the state between wakefulness and sleep. EEG patterns display theta (θ) waves, 4 to 8 Hz in frequency, which are larger in size, observed in the 50 to 100 µV range. Vertex sharp waves (V waves) are observed during this state and as the name suggests are sharp waves that last approximately 0.5 seconds (Iber et al., 2007). Physiological recordings indicate slow ocular movements and a decline in muscle activity. Individuals entering this state may not respond, but if woken may say that they had not been asleep at all (Peigneux et al., 2012). A healthy sleeper will stay in stage N1 for one to seven minutes at the start of the night (Carskadon & Dement, 1996).

The second stage of NREM (N2) commences a few minutes after stage N1 (Carskadon & Dement, 1996). During N2, EEG waves are in the sigma (σ) range with a frequency between 11 and 16 Hz. The size of the waves ranges from 50 to 150 µV. The EEG pattern also displays K-complexes, which are approximately 0.5 seconds in duration and appear as negative sharp waves preceding a positive component (Peigneux et al., 2012), and sleep spindles which are observed as a train of unique waves greater than or equal to 0.5 seconds (Iber et al., 2007). The eyes stop moving, the heart rate slows, and as the body prepares for deep sleep, core body temperature decreases (Peigneux et al., 2012).

N3 sleep or SWS is scored when EEG waves are at their slowest (also referred to as delta, δ), ranging between 0.5 to 2 Hz and greater than 75 µV in amplitude (Iber et al., 2007). N3 is experienced as the deepest sleep stage. If someone were to wake from this stage they might be confused or disorientated. There is a higher threshold for
arousal during N3 and typically it takes longer to wake up from this stage when compared to waking up from stages N1 or N2 (Peigneux et al., 2012).

### 3.2.3 Physiological observations during REM sleep.

REM sleep is characterised by the presence of desynchronised EEG patterns, similar to wakefulness. EEG waves range between 15 and 30 Hz and less than 50 µV in amplitude. There is no muscle movement (atonia). Eye movement, in contrast, is rapid and respiration is irregular (Peigneux et al., 2012). In healthy sleepers, REM occurs between 70 and 100 minutes after the N1 (Dijk & Lazar, 2012). Generally, initial REM episodes are the shortest, and as the night progresses, REM episodes increase in length, and NREM decreases in length (Feinberg & Floyd, 1979).

### 3.2.4 Sleep stages from childhood to adulthood.

It is important to understand that even in ‘healthy’ individuals sleep changes across the lifespan, and so before sleep changes across the lifespan of BD can be examined, this section will describe ‘normal’ changes. Ohayon, Carskadon, Guilleminault, and Vitello (2004) conducted a meta-analysis of human sleep from childhood to adulthood. Their paper reviewed 65 objectively recorded studies that investigated age-related changes across the lifespan. Figure 3, taken from Ohayon et al. (2004), depicts the changes in sleep architecture across the lifespan. As the present project focuses on BD, and since the earliest symptoms appear to manifest in late childhood/adolescence, the following section will describe the development of normal sleep from late childhood to late adulthood.

Findings regarding sleep architecture between childhood (5 years and older) to early adolescence include: the percentage of Stage 2 sleep and REM increases with age; sleep efficiency and latency remained unchanged; and there is no change in total sleep
time from the age of 5 years—differences observed by some studies were explained by environmental factors (i.e. school times; Ohayon et al., 2004).

A major change in sleep occurs with puberty, in the form of a delay of onset of night sleep and subsequent sleeping in, known as a phase delay (Carskadon, 1990; Frey, Balu, Greusing, Rothen, & Cajochen, 2009). Of the few studies that look at sleep during the transition from childhood to adolescence, the majority employed subjective tools (Lee & Rosen, 2012). Of those that use polysomnography (multi-modal technique that incorporates physiological and electrophysiological measures to record sleep, mainly using EMG, EOG and EEG recordings; Peigneux et al., 2012) and actigraphy (a device worn like a watch which measures and stores motion via an accelerometer) measures, changes in sleep patterns appear age-related and occur before the onset of puberty.
Feinberg et al. (2006) found a significant decrease in delta power density, a brain maturation indicator, seen over time from the age of 12 years. Further, the delta power density values were lower for females, but the rate at which delta power decreased was similar between boys and girls. Cambell, Higgins, Trinidad, Richardson, and Feinberg (2007) observed that between the ages of 12 and 14 years, sleep decreased, on average, 17 minutes. However, other studies did not observe a decrease in sleep need during preadolescence (Carskadon, Acebo, & Jenni, 2004; Carskadon, Acebo, & Seifer, 2001). By 16 years of age delta power density levelled out between males and females (Feinberg & Campbell, 2010).

Sleep delay may continue in early adolescence. Laberge et al. (2001) conducted a longitudinal study of sleep in early adolescents aged between 10 and 13 years. They found that nocturnal sleep decreased, bed time is delayed, and the difference between weekend (beginning on Friday night) and weekday sleep routines is increased. They found that 10-year olds slept, on average, one hour more on weekends, whereas 13-year olds slept, on average, two hours longer on weekends. School time appeared to act as a zeitgeber, an environmental cue that signals time (Bear, Connors, & Paradiso, 2007). Laberge et al. (2001) observed that girls showed more time in bed at an earlier chronological age than boys, but explained this as due to the earlier maturation of females. These findings have been confirmed by more recent work (Sadeh, Dahl, Shahar, & Rosenblat-Stein, 2009); specifically, that when pubertal stage was taken into consideration, sleep timing was delayed, and sleep pattern was disrupted from weekday sleep to weekend sleep.

Changes to the duration of SWS and REM continue with aging. SWS (delta sleep) continues to decline with age, and eventually is replaced with light sleep (N1 and
N2; Ohayon et al., 2004). As adults get older, the percentage of time taken to enter stages N1 and N2 increases, while the percentage of time spent in REM sleep decreases (Ohayon et al., 2004). Sleep efficiency from the age of approximately 60 years begins to decline (Ohayon et al., 2004). Sleep becomes more discontinuous at night and the need for daytime sleep increases (Dijk, Groeger, Stanley, & Deacon, 2010). In healthy older adults, however, the total time spent in REM and total sleep time changes little (Ohayon et al., 2004). No longitudinal studies have examined sleep in healthy elderly participants. Furthermore, is it difficult to identify a true 'normal' of the development and changes in sleep in the elderly due to their experiences of different developmental (e.g., menopause) and life transitions (e.g., caring for children or retirement; Lee & Rosen, 2012).

3.2.5 Circadian rhythm entrainment.

Across species, the timing of the active phase of the circadian system is believed to occur during times where engagement in the earth's environment is most optimal (Moore-Ede, 1986). The hypothalamic suprachiasmatic nucleus (SCN) regulates the circadian rhythms of the human body (Reppert & Weaver, 2002). Circadian rhythms refer to the bodily functions and behaviours that occur in a day, such as body temperature, hormone levels, metabolic rate, urine production, blood flow and (most importantly for BD) sleep-wake cycles (Bear et al., 2007). The free-running human circadian period is approximately 24.2 hours in length (Czeisler et al., 1999). The human circadian period can be entrained, meaning that environmental cues, zeitgebers, can adjust the endogenous systems that regulate the period and phase of the circadian cycle (Caldelas, 2005).

The most dominant of zeitgebers is light (Borisenkov, 2011; Takasu et al., 2006; Wright et al., 2013). Timing of light pulses has been found to advance or delay
circadian rhythms, dependent on when they are administered in relation to core body temperature nadir (lowest point; Dijk & Lazar, 2012). If light pulses are administered at night prior to the core body temperature nadir, a phase delay of the circadian period will occur; if administered in the morning or afternoon, subsequent to a core body temperature nadir, the circadian phase will advance (Dijk & Lazar, 2012). However, the circadian rhythm (or period) can also be entrained by non-photic events. These events include social cues (Elmore, Betrus, & Burr, 1994; Mistlberger & Skene, 2004), exercise (Yamanaka et al., 2006), meal times (Kräuchi, Cajochen, Werth, & Wirz-Justice, 2002; Mendoza, 2007), and sleep schedule (Cajochen, Jewett, & Dijk, 2003). The circadian system is regulated by a series of feedback loops (Bechtel, 2010). Behaviour is the most important feedback, as it directly (Mistlberger, Antle, Glass, & Miller, 2000) and indirectly (by light exposure) influences the circadian timing system, allowing light and behavioural based interactions to change the timing of sleep. As the mechanisms behind sleep are not the focus of the present project, the reader is directed to Dijk and Lazar (2012) for more information.

3.2.6 Neurohormone regulation of wakefulness and sleep.

Although neurohormone regulation was not a focus of the present project, it is useful to briefly characterise these mechanisms in the regulation of sleep. Multiple neurochemicals and cells have been identified in the regulation of normal sleep (see Table 4). Furthermore, it has been suggested that the irregular secretion of certain neurohormones may contribute to the manifestation of full blown BD. Drug studies have found improved outcomes for BD individuals who are administered psychotherapeutic drugs that involve serotonergic transmission (Smeraldi, Benedetti, Barbini, Campori, & Colombo, 1999), dopaminergic transmission (Andreazza & Kim, 2012; Cousins et al., 2009), and the administration of melatonin (Livianos, Sierra,
The findings regarding altered neurohormone regulation in BD will also be described through this section.

Table 4

**Neurohormone Activity During Wakefulness, NREM and REM**

<table>
<thead>
<tr>
<th>Neurohormone Activity</th>
<th>Wakefulness</th>
<th>NREM</th>
<th>REM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetylcholine</td>
<td>High</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Increased Activation</td>
<td>Active</td>
<td>Active</td>
</tr>
<tr>
<td>Glutamatergic cells</td>
<td>High</td>
<td>Low</td>
<td></td>
</tr>
<tr>
<td>Histamine</td>
<td>Active</td>
<td>Inactive</td>
<td>Inactive</td>
</tr>
<tr>
<td>Hypocretin/ Orexin</td>
<td>Active</td>
<td>Inactive</td>
<td></td>
</tr>
<tr>
<td>Melatonin</td>
<td>Suppressed</td>
<td>Active</td>
<td>Active</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>Active</td>
<td>Slowing activity</td>
<td>Inactive</td>
</tr>
<tr>
<td>Serotonin</td>
<td>Maximum activity</td>
<td>Decreased activity</td>
<td>Inactive</td>
</tr>
</tbody>
</table>

*Note. Information presented above has been taken from Peigneux (2012)*

Cells that produce acetylcholine are located in the basal forebrain and the brainstem, specifically in the anterior hypothalamus and the pedunculopontine tegmentum (Datta & MacLean, 2007; Peigneux et al., 2012). As shown in Table 4, acetylcholine production is high during REM and wakefulness, and diminishes during NREM. Acetylcholine is largely involved in processes related to wakefulness by activating specific systems in the brain and behaviours, like attention (Bartus, Dean, Beer, & Lippa, 1982), learning (de Castro et al., 2009; Thiel, Huston, & Schwarting, 1998), and sensory processing (Oldford & Castro-Alamancos, 2003). Interestingly, the
administration of acetylcholine elicits muscle atonia (Gall, Poremba, & Blumberg 2007; Gillin, Sitaram, & Mendelson, 1982) and is involved in the suppression of motor activity during REM sleep (Peigneux et al., 2012).

Dopamine producing cells are located in the ventral tegmental area and the substantia nigra and dopamine concentrations are significantly increased during wakefulness (Lena et al., 2005). Dopamine is involved in motor control (Rosenzweig, Breedlove, & Leiman, 2002), and may play a central role in BD (Cousins et al., 2009). Dopaminergic drug administration improves mood in BD participants (Benedetti, Campori, Barbini, Fulgosi, & Colombo, 2001). Stimulation of dopamine activity results in brain effects similar to those observed in BD (Cousins et al., 2009). Furthermore, the dopamine precursor L-dopa (van Praag & Korf, 1975) and other dopamine agonists also affect mania (Colonna, Petit, & Lepine, 1979; Post, 1978; Silverstone, 1984).

Datta and MacLean (2007) suggested that glutamatergic cells are involved in maintenance of a wakeful state. These cells are located in the mesencephalic reticular formation (Peigneux et al., 2012). Glutamatergic cell activity is highest during wakefulness. Like the role of glutamatergic cells, histamine-producing cells are also involved in the maintenance and promotion of wakefulness (John, Wu, Boehmer, & Siegel, 2004; Ramesh, Thakkar, Strecker, Basheer, & McCarley, 2004). The loss of consciousness observed during sleep may be caused by inactivation of histamine-synthesising cells (John et al., 2004).

Hypocretinergic (also known as orexinergetic) neurons are located between the posterior hypothalamus and the lateral hypothalamus (Gerashchenko & Shiromani, 2004). They promote wakefulness (Gerashchenko & Shiromani, 2004), and human narcolepsy has been linked to the loss of these cells (Thannickal, Siegel, Nienhuis, & Moore, 2003). Lesions to the lateral hypothalamus are associated with narcoleptic
behaviours in rats seen as sleep onset REM sleep periods, increased NREM and REM sleep at night, fragmented sleep, and blunting of the diurnal rhythm (Gerashchenko & Shiromani, 2004).

Melatonin is produced in the pineal gland (Riemann & Nissen, 2012). Melatonin peaks during the night, increases in the hours leading to sleep, and is suppressed during wakefulness (Riemann & Nissen, 2012). Melatonin brings on sleep, increases the duration of time spent sleeping (Mendelson, 1997; Rajaratnam, Middleton, Stone, Arendt, & Dijk, 2004), and entrains circadian rhythms in blind people so that their circadian rhythm is no longer free-running (Sack, Brandes, Kendall, & Lewy, 2000). The circadian timing system may be adjusted by administration of melatonin (Fuller et al., 2006). Melatonin may also be abnormally secreted in individuals with euthymic BD (Lewy et al., 1985; Nurnberger et al., 2000). Lewy et al. (1985) observed that, during the night, melatonin levels in euthymic BD individuals fell twice as much as the levels observed in matched healthy controls. Several years later, Nurnberger et al. (2000) also found that melatonin was abnormally secreted in euthymic BD I participants. Similarly, Robillard et al. (2013) compared melatonin levels of BD participants to those of participants with UD, and found that individuals with BD had lower concentrations of melatonin than UD participants did, and the timing of secretion was delayed in BD participants. Livianos et al. (2012) found that administration of melatonin improved the sleep quality and quantity of 14 euthymic BD individuals who also suffered from insomnia. Despite differences in melatonin secretion, there is no evidence that the volume of the pineal gland (responsible for melatonin secretion) in BD is significantly different in size compared to healthy controls (Sarrazin et al., 2011).

Norepinephrine is produced mostly in the locus coeruleus of the pons (Peigneux et al., 2012). Norepinephrine cells fire regularly during wakefulness, slow down during
early NREM phases (Foote, Bloom, & Astonjones, 1983), and cease firing during REM (Datta & MacLean, 2007). Activation of cells that produce norepinephrine are linked to behavioural arousal and cortical activation (Peigneux et al., 2012). Similar to the effect of acetylcholine suppressing muscle tone, the inhibition of these cells is also linked to loss of muscle tone (John et al., 2004). Post mortem analyses comparing the locus coeruleus of six individuals who had suicided whilst in a depressive episode of BD to that of healthy control brains suggested that both serotonin and norepinephrine activity was lower in the brains of individuals with BD (Wiste et al., 2008).

Serotonin production occurs in the raphe nuclei of the brainstem (Peigneux et al., 2012). Like norepinephrine, serotonergic cells are at their maximum activity during wakefulness, show decreased activity during NREM sleep, and are inactive during REM (Lydic, McCarley, & Hobson, 1983). The exact role that serotonergic cells play in sleep/wake cycles remains unclear (Datta & MacLean, 2007). Serotonin may play various roles, including the regulation of muscle tone and arousal (John et al., 2004; Wu et al., 2004). Research investigating genes has also identified a serotonin transporter gene involved and suggests possible mediation of this gene between harm avoidance and BD (Lu et al., 2012). Additionally, a serotonin transporter was regulated differently in euthymic BD participants when compared to healthy controls (Chou et al., 2010).

Cortisol is involved in the inhibition of central nervous system neurons, inhibits the immune system and mobilises energy stores (Bear et al., 2007). Although cortisol has not been implicated in the regulation of sleep, altered levels have been observed following total sleep deprivation in healthy adults (Klumpers et al., 2015; Wright et al., 2015). In healthy adults, sleep deprivation lowers cortisol levels (Klumpers et al., 2015). It has been suggested that life events trigger changes in cortisol levels, and these changes may directly impact sleep (Levenson, Nusslock, & Frank, 2013). Levenson et
al. proposed a conceptual model that explained the role of life events in the disruption of sleep. Their model included factors such as coping, BAS reward systems, light exposure, emotional and physiological arousal and the impact of these life events on the hormone cortisol. They suggested the following possible avenues that impact cortisol release: that changes in social rhythms or an increase in light exposure could disrupt sleep by increasing cortisol levels; that striving or attaining reward could impact sleep; and the stressful or threatening life events disrupt sleep by directly increasing cortisol levels.

In summary, multiple neurohormones are involved in the regulation of wakefulness and sleep. The regulation of serotonin, norepinephrine, dopamine, and melatonin has also been observed to be altered in BD. Although exploration of life events and hormone regulation are beyond the scope of the present project it is important to understand the underlying mechanisms possibly involved in the regulation of sleep in BD.

3.2.7 Sleep as a State and Trait.

Sleep has state (temporary) and trait-like (more enduring) features. Sleep is typically understood and researched as a state phenomenon, but a range of evidence suggests that there are also stable individual differences in sleep (Van Dongen, Vitellaro, & Dinges, 2005). State features refer to the type of sleep one has experienced in the short-term, for example the past night or week, or even month; whereas trait-like features of sleep, refers to the type of sleep one has experienced for most of their life, such as sleep timing preference (such as morningness/eveningness; Dagys et al., 2012; Mansour et al., 2005), the typical duration of sleep (Auyeung et al., 2013; Dewald-Kaufmann, Oort, BÖGels, & Meijer, 2013; Lemola, Ledermann, & Friedman, 2013), and sleep quality (how well one deals with disturbed or restricted sleep; Buysse et al.,
There is no shortage of self-report measures to quantify state sleep. Some notable instruments include the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), which is a self-report questionnaire assessing sleep quality, and the Epworth Sleepiness Scale (Johns, 1991), a short self-report measure that measures daytime sleepiness. In contrast, there are no self-report questionnaires that enquire about trait-like sleep variables.

Aside from self-report scales that measure chronotype (morningness/eveningness), such as the Composite Scale of Morningness (CSM; Smith, Reilly, & Midkiff, 1989), there are no self-report measures that enquire about facets of sleep over a long period of time, representing stable, trait-like individual differences in sleep. As recent studies have begun to look at trait vulnerability to sleep focussing on how healthy individuals respond to sleep restriction or total sleep deprivation (Rupp, Wesensten, & Balkin, 2012), the present study sought to investigate trait-like sleep variables and state sleep qualities in a general-population sample whose vulnerability to BD was also measured using self-report questionnaires.

A scale that enquires about the facets of sleep behaviour is the Adult Sleep Wake Scale (ASWS; Fortunato et al., 2008); however, this scale is state-framed. The ASWS is a 25-item scale that measures five facets of sleep behaviours, specifically the degree of difficulty an individual has when going to bed (GTB), falling asleep (FA), maintaining sleep (staying asleep: SA), reinitiating sleep (RS) and returning to wakefulness (waking up: WU). With the authors’ permission, the ASWS was adapted as a trait-like sleep measure; the psychometric properties of the modified ASWS are described in the Method section. The ASWS was chosen as the best suited measure to
modify to a trait-framed questionnaire because it enquires about behaviours around the different aspects of sleep. Although the present study modified an existing sleep measure, it was beyond the scope of the present study to investigate the validity and psychometric properties of the modified ASWS (however, internal consistency was tested and can be viewed in the Results section). The modified ASWS was used purely as a measure to capture trait-like sleep.

3.2.8 Relationship between sleep, cognitive and emotional functioning, and coping in healthy samples.

In healthy individuals, sleep deprivation impairs multiple facets of cognitive functioning, including attention (Mander et al., 2008; Shao et al., 2008), alertness (Lo et al., 2012; Wehrens, Hampton, Kerkhofs, & Skene, 2012), vigilance (Martella, Marotta, Fuentes, & Casagrande, 2014), decision making (Glass et al., 2011; McKenna, Dicjinson, Orff, & Drummond, 2007), problem solving (Killgore et al., 2008; Kjellberg, 1975; Sadeh, Keinan, & Daon, 2004), and emotional intelligence related to effective coping (Killgore et al., 2008).

Sleep deprivation, in non-clinical samples, also impacts perception of self and the choices one makes (Killgore et al., 2008). Killgore et al. found that sleep deprivation decreased perceived self-regard, assertiveness, independence, self-actualisation, capacity for empathy, quality of interpersonal relationships and impulse control in 26 healthy military volunteers (mean age = 25.4 years; Killgore et al., 2008). Thus, not only are the sleep-deprived likely to make poorer decisions, they are more likely to alienate themselves from others by their actions.

Sleep deprivation is strongly linked with mood changes (Dagys et al., 2012; Franzen, Siegle, & Buysse, 2008; Minkel et al., 2012; Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010). Sleep deprivation has been linked with adverse impact of affect
in both adolescents and adults (Talbot et al., 2010). Talbot et al. examined the effects of sleep deprivation on mood in adolescents (10 to 13 year olds), mid-adolescents (13 to 16 year olds) and adults (30 to 60 year olds). Two nights of sleep deprivation significantly reduced reported positive mood across the age groups compared to the rested condition (two nights of healthy sleep). Anxiety as a consequence of catastrophising was significantly higher across the age groups when compared to the rested condition. When asked to rate the likelihood of a catastrophe occurring, scores were higher in the sleep-deprived condition. Talbot et al. (2010) also observed that adolescents appraised a threatening worry as more threatening in the sleep-deprived condition than when rested, but this pattern was not observed in adults or mid-adolescents.

Similarly, Dagys et al. (2012) investigated the effects of sleep deprivation on mood in 47 healthy adolescents (mean age = 13.06 years) by comparing their mood following a rested night and a sleep deprived condition. They found that sleep deprivation was associated with lower positive affect. Furthermore, a subset of participants \( (n = 24) \) were identified as extreme morning or evening types, and preference for eveningness in healthy adolescents was associated with emotional vulnerability as they produced even lower positive affect scores than participants who had a preference for activity in the morning.

In line with research findings of Talbot et al. (2010) and Dagys et al. (2012), sleep deprivation is associated with higher levels of negative affect in young healthy adults (mean age = 24.4 years; Franzen et al., 2008). Franzen et al. compared measures of mood between participants who had been assigned to a sleep-deprived condition \( (n = 15) \) to those that were assigned to a non-sleep deprived condition \( (n = 14) \), following a baseline condition (one night of healthy sleep). Sleep deprived participants reported
lower positive affect, higher negative affect and higher negative mood than the non-sleep-deprived participants. They also observed that physiological mood measures were impacted by sleep deprivation. Participants in the sleep-deprived condition showed reduced positive affect and increased negative affect through pupil diameter changes (based on brain activity) in response to pictures (Franzen et al., 2008). Franzen et al. suggested that careful examination of the impact of sleep deprivation on factors such as affect and cognition could lead to predictors of trait vulnerabilities to negative effects of sleep loss.

When in a distressed state, sleep-deprived individuals also made poorer coping style decisions (Sadeh et al., 2004). Sadeh and colleagues analysed the coping styles (self-report scores from a coping questionnaire) and sleep using actigraphy and sleep logs in 36 healthy tertiary students during a stressful academic week (being evaluated for admission into a graduate program) and a non-stressful academic week (two to three months prior to the stressful week). They investigated the relationship between emotion-focussed coping (the regulation of emotional responses to the problem), problem-focussed coping (changing or managing the distress causing problem) and disengagement (disengaging from thoughts, emotions and threatening stimuli), and sleep. Analyses revealed that coping style acted as a moderator of changes observed in sleep. During the high stress week, individuals who scored higher on emotion-focussed coping had less sleep and reported poorer quality sleep, and those low on emotion-focussed coping reported better quality sleep and slept for longer periods than they did during the low-stress week. Irrespective of stress, individuals who scored high on problem-focussed coping slept for longer periods. There were no significant findings for disengagement. Sadeh et al. suggested that ineffective coping may result in shortened sleep length, and concluded that individuals who score higher on emotion-focussed
coping are more likely to be aroused and thus have more difficulty sleeping. This is consistent with research observations attempting to explain the occurrence of insomnia (see section 3.3.1; Harvey, McGlinchey, & Gruber, 2010), and also suggests that sleep may have a moderating effect on coping.

Sleep deprivation is also linked to higher self-reported symptoms of affective psychopathology (Kahn-Greene, Killgore, Kamimori, Balkin, & Killgore, 2007). Kahn-Greene et al. (2007) compared scores on the Personality Assessment Inventory (a widely used clinical tool assessing psychopathology; Morey, 2007) of 25 healthy participants (mean age = 25.36 years) at baseline (normal night's sleep) and following sleep deprivation (56 hours awake). They found significant differences between the two sleep conditions (normal and sleep deprivation) on the clinical scales of anxiety (health concerns), depression (affective and cognitive), paranoia (persecution and resentment), and somatic complaints (health concerns). This is consistent with a review by Morin and Ware (1996) who also highlighted the overlap between sleep disturbances and psychopathology, stating that between 50 and 80 % of individuals who suffer from a psychiatric illness also complain of sleep disturbances early in their illness. Results from a sleep deprivation study suggested that there is a trait-like resilience from individual to individual that determines how well one copes with either sleep restriction or total sleep deprivation (Rupp et al., 2012).

3.3 Sleep Disorders

Although the present project investigates quantitative measures of sleep quality, it is useful to briefly describe categorical pathologies of sleep. Most taxonomies recognise two broad categories of sleep disorders: dyssomnias and parasomnias. Dyssomnias refer to disorders of the intrinsic mechanisms of sleep, including diagnoses of insomnia, narcolepsy, circadian rhythm sleep disorders, hypersomnia, and breathing-
related sleep disorders (Edinger & Morin, 2012). Parasomnias, in contrast, are disorders that involve events or behaviours that occur during sleep (Edinger & Morin, 2012), such as sleep terrors, nightmares, and sleep-walking (Anderson & Bradley, 2013; Edinger & Morin, 2012). Describing each sleep disorder is beyond the scope of the present project; however, because there is considerable comorbidity between insomnia and a BD diagnosis (Gruber, Eidelman, & Harvey, 2008; Harvey et al., 2005) it will be briefly described here.

3.3.1 Insomnia.

The term *insomnia* is used to define difficulties getting to sleep (initial insomnia), staying asleep (middle insomnia) or waking up earlier than desired (late insomnia; Gehrman, Findley, & Perils, 2012), and not feeling as though sleep was restful or restorative (Harvey et al., 2010). Individuals who suffer from insomnia have difficulty functioning during the day (Riemann et al., 2010), significant difficulties with memory (Backhaus et al., 2006; Fortier-Brochu, Beaulieu-Bonneau, Ivers, & Morin, 2012), and are more at risk of developing a psychiatric disorder (Harvey, 2008a). The prevalence of a clinical diagnosis of insomnia is approximately 6% (Ohayon, 2002).

A prominent explanation for insomnia implicates hyper-arousal (Kaplan, Talbot, & Harvey, 2009; Riemann et al., 2010); however, there are competing hypotheses as to how exactly hyper-arousal contributes to the occurrence of insomnia. The first possible explanation for insomnia implicates a bi-directional vicious cycle involving emotions, that is, when the individual suppresses their emotions during the day, a rebound effect occurs at night and the individual can no longer suppress their emotions (Harvey et al., 2010). Alternatively, the individual could be hyper-aroused during the day, and may get more aroused as the day progresses, so that when it comes to falling asleep they are unable to deactivate the arousal, fuelling insomnia (Harvey et al., 2010). Therapeutic
approaches in the treatment of insomnia involve relaxation, sleep hygiene, stimulus control, sleep compression and restriction, and cognitive behaviour therapy for insomnia (CBT-I; Lichstein, Vander Wal, & Dillon, 2012), with CBT-I being the most prominent intervention (Ebben & Narizhnaya, 2012).

3.4 Sleep and BD

Another vulnerability to BD appears to be circadian variability (Jones, Hare, & Evershed, 2005; Ritter et al., 2011). Of all the circadian rhythms, the most affected in BD is the sleep-wake cycle (Harvey, Mullin, & Hinshaw, 2006). There is a variety of different types of evidence to show that sleep is involved in BD. Sleep disturbances are both trait-like and potentially causal in nature, as they are considered early markers of BD (Ritter et al., 2015) and occur between affective episodes (Harvey et al., 2005), and state-like as they have been observed to be prodromes to both depressive and manic BD episodes (Jackson, Cavanagh, & Scott, 2003; Sierra, Livianos, Arques, Castelló, & Rojo, 2007; Sylvia et al., 2012) and are associated with an impact on mood the next day (Talbot et al., 2012). Sleep disturbances in BD are also indicative of poor prognosis (Asarnow, Soehner, & Harvey, 2014; Kaplan, Gruber, Eidelman, Talbot, & Harvey, 2011). This section will summarise literature linking BD to sleep problems.

3.4.1 Sleep problems in diagnosed BD samples.

Approximately 70% of individuals with BD experience significant clinical disturbances with sleep (Harvey et al., 2005). The sleep observed in BD has been found to more closely resemble the sleep observed in insomnia (Harvey, 2008b). It has also been observed that approximately 25% of individuals also experience hypersomnia between episodes (Kaplan et al., 2011). Sleep disturbance is the most common prodrome reported prior to a BD manic episode and the sixth most common prodrome reported prior to a BD depressive episode (Jackson et al., 2003).
Disturbed sleep is a feature across all phases of BD (Harvey, Talbot, & Gershon, 2009). Sleep dysregulation may be a core feature of BD (Jones, Tai, Evershed, Knowles, & Bentall, 2006), and has been referred to as an endophenotype, a biological or vulnerability marker, of BD (Lenox, Gould, & Manji, 2002). Sleep disturbances appear to have a causal relationship with BD as they have been documented to precede the onset of a BD diagnosis (Ritter et al., 2015; Ritter et al., 2011). Ritter et al. (2015) conducted a 10-year investigation of sleep in a sample that was initially free of diagnosis ($N = 1943$). They found that BD incidence was predicted by ‘trouble falling asleep’ and ‘early morning awakening’. This study was the first to longitudinally document that poor sleep quality was a risk factor for BD onset at a later time point, and provides causal evidence that sleep disturbances occur before the onset of BD.

Furthermore, when experiencing depression and mania mood symptoms, adolescents have reported sleep impairments to duration of sleep and disturbed morning routines (Lunsford-Avery, Judd, Axelson, & Miklowitz, 2012). The sleep impairments observed in adolescents continue on in adulthood.

Harvey et al. (2005) compared the sleep of euthymic BD patients to that of participants with insomnia and participants with good sleep. They found that, aside from criterion D (“that the disturbance does not occur exclusively during the course of another mental disorder” American Psychiatric Association, 2000, p. 604, which is no longer a diagnostic criterion for Insomnia Disorder in the current DSM-5), 55% of the BD group ($n = 20$) met diagnostic criteria for Insomnia and 70% of the BD group had a clinically significant problem with sleep (Harvey et al., 2005). Like Harvey et al., Brill et al. (2011) found that, of the 116 participants in a euthymic state of BD, 23.6% also suffered from a clinically significant sleep disturbance. Although the percentages seem high in both studies, the sample sizes were small, and so larger studies are needed to
examine the prevalence of sleep disorders in people with a BD diagnosis. Similarly, Jones, Hare, and Evershed (2005) conducted a cross-sectional investigation of mood and a 7-day actigraphy recording comparing the sleep of 19 adults with sub-syndromal BD symptoms and 19 matched controls. They found that BD participants experiencing minor levels of sub-syndromal symptoms continued to experience disrupted circadian activity (Jones et al., 2005).

Sleep disturbances may predict BD episodes as they have been observed to precede BD episodes. Recent research found an association between sleep disturbance and later recurrence of a mood episode in euthymic bipolar patients (Sylvia et al., 2012). Sylvia and colleagues (2012) found that 73 of 483 individuals diagnosed with BD also suffered from at least a mild sleep disturbance. In that sample of identified sleep disturbed individuals there was no pattern or association in demographic variables such as age, race, gender, marital status or in specific mental health variables, like substance abuse disorder, rapid cycling BD or comorbid anxiety diagnosis (Sylvia et al., 2012). Sylvia et al. also investigated if medications commonly used in the treatment of BD were associated with sleep disturbance and found that, although the commonly prescribed BD pharmacotherapies (lithium, lamotrigine, valproate, or atypical antipsychotics) did not have an association, anticonvulsants, which are commonly prescribed in addition to antidepressants, were significantly associated with sleep disturbances. However, the majority of individuals who were on a form of anticonvulsant medication were taking gabapentin. They concluded that sleep disturbance is a prodrome to bipolar episodes (Sylvia et al., 2012).

Additionally, the architecture and timing of sleep appears abnormal in BD (Eidelman, Talbot, Gruber, Hairston, & Harvey, 2010; Salvatore et al., 2008; Talbot, Hairston, Eidelrnan, Gruber, & Harvey, 2009). Salvatore et al. (2008) compared the
daily activity of 32 healthy control individuals to that of 36 adults diagnosed with BD I whilst syndromal and whilst euthymic, and found significant differences in the amount of nocturnal sleep between individuals with BD I and controls; between daytime activity (less daytime activity in BD I participants), and significant differences in the timing of the circadian system (acrophase advance—meaning that the circadian rhythm of activity peaks at an earlier time in the day). They concluded that there might be a trait difference in the circadian activity of recovered, clinically well, BD I patients when compared to healthy controls (Salvatore et al., 2008). Furthermore, people who have BD also have decreased sleep efficiency compared to participants diagnosed with UD (Riemann, Voderholzer, & Berger, 2002). Riemann et al.’s (2002) review found: that time spent in SWS was significantly decreased; that time in REM sleep was significantly more intense; and that early morning awakenings significantly increased in participants with BD.

Mood also impacts on the architecture of sleep in BD (Talbot et al., 2009). Talbot et al. compared the sleep onset latency (SOL) and REM measures (latency and density) between 28 inter-episode BD participants and 28 healthy controls and mood induction (by playing happy or sad music). They recorded participants' sleep for two baseline nights and then compared the average readings to a night's sleep following a happy mood induction and one following a sad mood induction. There were no differences between the groups for the baseline nights. For the happy mood induction night the following differences were observed: the BD participants reported taking longer to fall asleep than the controls; polysomnography recordings showed higher REM density for the BD group than the controls; and the control participants reported falling asleep quicker than they did on baseline nights. For sleep following sad mood induction the following was observed: both groups reported shorter SOL when
compared to the baseline nights, indicating that they fell asleep more quickly than on the sad-mood induction and non-mood conditions; both groups also displayed higher REM density on the sad mood induction night than the baseline nights; and polysomnography readings for the BD group showed that they experienced significantly higher REM density than controls and that they spent less time in the first REM period during the sad mood induction when compared to baseline. Talbot et al. concluded that, even when in a euthymic state, mood influences the sleep of individuals with BD.

Eidelman, Talbot, Gruber, Hairston et al. (2010) conducted a similar investigation of the effect of mood on sleep architecture in BD. They examined if sleep architecture predicted BD symptoms at a later time point comparing the sleep of 22 individuals with BD (I or II) to 22 non-clinical controls. Participants completed questionnaires to measure mood and impairment and had their sleep recorded via polysomnography, and repeated the questionnaires after three months via a telephone interview. At baseline, the BD group had significantly higher REM density. At the three-month follow-up, the sleep architecture observed in the BD participants indicated the following: a significant positive correlation existed between the duration of the first REM period observed at baseline and both manic symptoms and reported impairment at the three-month follow up; REM density at baseline correlated positively with reported impairment and depressive symptoms at the three-month follow up; and SWS recorded at baseline was negatively correlated with impairment and manic symptoms at the three-month follow up (Eidelman, Talbot, Gruber, Hairston, et al., 2010). The only significant finding concerning sleep architecture in the control group was that REM density at baseline negatively correlated with impairment at the three-month follow up (Eidelman, Talbot, Gruber, Hairston, et al., 2010). Eidelman and colleagues concluded that sleep architecture could predict future BD symptoms and that the differences in the way REM
and NREM function, in individuals with BD, may act as an illness-maintaining mechanism.

People with BD also report poor quality sleep. Rocha et al. (2013) compared reported sleep quality of 104 healthy controls with that of 105 euthymic BD participants. Responses from the PSQI (Buysse et al., 1989) were compared and 21.2% of the healthy control group reported poor sleep quality, compared to 82.9% of the BD sample. Furthermore, when the subscales were analysed, there were significant differences between the two groups on the subscales of perceived sleep quality, sleep efficiency, daytime functioning, and sleep latency, as well as the overall PSQI total score, showing that the BD group displayed more impaired sleep (Rocha et al., 2013). Of the BD group, 78% reported using sleep medication, as opposed to none of the control group. The significant differences in sleep quality between healthy participants and those with BD strengthens the contention that sleep acts a state and trait-like marker of BD (Rocha et al., 2013).

The onset of manic or depressive symptoms appears to be directly associated to the amount of sleep individuals have, with a decrease in sleep related to shift towards a mania or hypomania and an increase in sleep related to a shift towards depression (Bauer et al., 2006). Total sleep duration is also linked to illness severity in BD. Gruber et al. (2011) analysed the total sleep time and sleep variability of 196 individuals who were euthymic at the beginning of the study. They found that decreased total sleep time was associated with more severe manic symptoms, and increased sleep variability was associated with more severe manic and depressive symptoms over a 12-month period.

Disturbed sleep has been associated with increased BD symptoms. Eidelman, Talbot, Gruber, and Harvey (2010) investigated sleep events and their impact on symptom or episode (either depressive or manic) recurrence amongst 21 individuals
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with inter-episode BD. Bivariate correlations investigated the relationships between sleep efficiency, total wake time and total sleep time with manic or depressive symptoms and revealed some significant correlations. Significant correlations existed between: lifetime depressive episodes and variability of total wake time in bed; lifetime depressive episodes and variability of sleep efficiency when in bed; mania symptoms positively correlated with average sleep efficiency scores; and depressive symptoms positively correlated with variability in the time it took participants to fall asleep (Eidelman, Talbot, Gruber, & Harvey, 2010). Furthermore, when sleep items were removed from the mania and depression symptoms scales, significant positive correlations remained between mania symptoms and the average sleep efficiency scores, and between depressive symptoms and variability in falling asleep time (Eidelman, Talbot, Gruber, & Harvey, 2010). The only significant negative correlation was between lifetime depressive episodes and average sleep efficiency scores. Eidelman, Talbot, Gruber, and Harvey concluded that sleep disturbance is a trait marker of BD. This observation indicates that individuals with manic symptoms are more likely to fall asleep quicker once in bed, and are more likely to get out of bed. Furthermore, individuals who are experiencing depressive symptoms appear to take longer to fall asleep, and are more likely to stay in bed once awake in the morning (Eidelman, Talbot, Gruber, & Harvey, 2010).

Talbot et al. (2012) examined the relationship between sleep and mood in BD, but compared it to that of sleep in individuals with insomnia. They analysed the sleep and mood diaries of 49 individuals diagnosed with BD, 34 diagnosed with Insomnia, and 52 healthy controls, over a period of approximately one week. Analyses revealed that, overall, the insomnia group reported worse sleep than the BD and healthy control groups; the control group reported lower negative mood in the subsequent morning than
the BD and insomnia groups; there was no difference between the BD and insomnia groups for either morning or evening negative mood; and, there was no difference for reported positive mood between the three participant groups. Results analysing the effect of total sleep time and diagnostic group on subsequent day mood revealed that total wake time predicted positive and negative mood reports the subsequent morning in all three groups. Diagnostic status (BD, insomnia or healthy control) also predicted both positive and negative mood on subsequent mornings. More complex analyses showed that diagnosis was not a moderating factor in the prediction of subsequent positive mood in the morning, but being in the BD diagnostic group acted as a moderating factor on the relationship between total wake time and subsequent negative mood in the morning (Talbot et al., 2012).

Talbot et al. (2012) also analysed the interaction in the opposite direction; that is, the effect of mood on subsequent sleep patterns. Positive mood in the evening was not associated with total wake time on the next day; however, diagnostic status continued to predict wake time the next day, and further analyses revealed that only insomnia acted as a moderator in this relationship. Negative mood was associated with increased subsequent total wake time, and both diagnosed group (BD and insomnia), and negative mood predicted subsequent total wake time. More in-depth analyses revealed that both insomnia and BD acted as moderators on the interaction between total wake time and negative mood. Although Talbot et al. (2012) acknowledged that sample sizes could have been larger, and that one week may not have been long enough to observe the effects of positive mood, they concluded that sleep and negative mood are bi-directionally related in BD (as well as insomnia), and suggested that treatments employed to treat insomnia should also be considered in the treatment of BD, specifically sleep restriction and stimulus control.
In summary, sleep is connected to BD in multiple ways. Research has consistently suggested that there are trait-like and state associations between sleep and BD. The trait-like evidence stems from the documented sleep differences in healthy adolescents who later go on to develop BD (Ritter et al., 2015), and the studies showing that sleep difficulties persist in the absence of BD symptoms and episodes (Brill et al., 2011; Harvey et al., 2005). State associations come from established associations between sleep disturbance and mood changes in BD (Talbot et al., 2012), evidence that the amount of sleep impacts affect in BD the next day (Bauer et al., 2006) and over a period of 12 months (Gruber et al., 2011), and that sleep difficulties act as prodromes to BD episodes (Sylvia et al., 2012). Other important findings showing the link between sleep and BD include: those that show that sleep difficulties affect the quality (Rocha et al., 2013) and architecture of sleep (Riemann et al., 2002); that the sleep of individuals with BD who also suffer from sleep disturbances resembles the sleep of those diagnosed with insomnia (Harvey, 2008b); and often individuals with BD also take sleep medication (Rocha et al., 2013).

3.4.2 Sleep deprivation as a treatment for BD depression.

Further evidence for the causal relationship between sleep and BD is that sleep deprivation has an antidepressant effect in the treatment of depression in BD (for a review see Benedetti & Colombo, 2011). The mere act of going to bed and waking up at regular times has been found to improve sleep in individuals with BD who also suffer from insomnia (Kaplan & Harvey, 2013). When treating depressive episodes in BD with sleep deprivation alone, only 5 to 10% of individuals sustain their remission (Colombo et al., 2000; Smeraldi et al., 1999). Lithium administration sustained and enhanced the therapeutic effect of sleep deprivation in BD (Colombo et al., 2000). Total sleep deprivation in combination with light therapy also improved mood in individuals
who were drug responsive (70 % improved) and drug-resistant (44 % improved; Benedetti et al., 2005). Sleep deprivation is not recommended in rapid-cycling BD (Benedetti & Colombo, 2011). Switch rates into mania or hypomania in non-rapid cycling BD participants following sleep deprivation therapy have been found to be comparable to drug treatment for depression (4.85 % and 5.83 % respectively; Colombo, Benedetti, Barbini, Campori, & Smeraldi, 1999).

3.4.3 Morningness/Eveningness and BD.

Morningness/Eveningness (M/E) measures have been used to indirectly investigate the pathogenesis of BD (Wood et al., 2009). Scores from M/E questionnaires are considered estimates of internal circadian phase (Mansour et al., 2005; Wood et al., 2009), or what could be referred to as trait-like sleep preference or chronotype. Individuals who score high on measures of Morningness, otherwise known as morning types or larks, prefer to go to bed and wake up earlier in the day, and also report good mood during the day. In contrast, individuals who score high on Eveningness (known as Evening types) prefer to go to bed and wake up later in the day, and mood and alertness improves later in the day than morning types (Kerkhof, 1998).

Mansour et al. (2005) investigated the relationship between M/E and BD, using the CSM (Smith et al., 1989), in healthy controls (n = 349), adults with BD (n = 75), and adults with schizophrenia or schizoaffective disorder (n = 81). Results revealed significant correlations between CSM scores and age. After age was corrected for, significant differences existed between CSM scores of BD patients and those of control and schizoaffective/schizophrenia participants. Their novel finding was that CSM scores positively correlated with age of onset of BD (r = .24) and the length of the most acute depressive episode (r = -.40). They concluded that the findings related to age of
onset and severity of episode were idiosyncratic to BD only, as this interaction did not occur in participants with a diagnosis of schizophrenia or schizoaffective disorder.

Wood et al. (2009) compared scores from the CSM between adults with BD (I and II, \( n = 134 \) and \( n = 56 \), respectively) and community controls (\( n = 128 \)). There were no significant differences between the BD I and BD II groups; however, individuals with a BD diagnosis had significantly lower scores on the CSM (indicative of more evening types) than the control sample. Wood et al. also identified a significant weak, negative correlation of \( r = -0.28 \) between CSM scores and scores from the Beck Depression Inventory (higher scores indicative of more severe depressive mood ratings; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961); and a significant positive and weak correlation of \( r = 0.22 \) between CSM scores and Global Assessment of Function (GAF) scores, indicating that individuals with higher depression scores were more likely to be evening types, and the more evening type an individual, the more severe their symptoms were. They also observed that individuals taking mood stabilisers, antipsychotics, antidepressants, or stimulants scored significantly lower on the CSM than individuals not taking these medications, whilst controlling for age-related effects on CSM scores.

Wood et al. also tested a subset of participants (\( n = 52 \)) two years later and found that test scores were stable over time. This finding aligns with that of Ahn et al. (2008) who found that, after controlling for age, individuals with a diagnosis of BD (\( n = 92 \)) significantly preferred activity in the evenings, than did healthy controls (\( n = 95 \); and Ahn et al., 2008, also employed the CSM). The BD participants also displayed a significant delay in sleep time. These findings indeed indicate that there is trait circadian preference for activity in individuals with BD.

Similarly, Bullock, Corlass-Brown, and Murray (2014) examined chronotype in a university sample (\( N = 213 \)) who were assessed along the BD spectrum using GBI
scores. Findings aligned with the clinical sample studies of Mansour et al. (2005), Wood et al. (2009), and Ahn et al. (2008), in that an increase in vulnerability to BD was associated with an increase in preference for evening activity (as noted by the significant, but weak, correlation of $r = .18$ between reverse-coded Morningness scores and GBI-Depression scores). The researchers suggested that, as they used a non-clinical sample who were assessed along a spectrum of BD, their findings added to the concept of a trait vulnerability to BD, observed through chronotype.

There have been mixed findings with regard to chronotype preference and BD. Auger et al. (2013), in contrast to works by Ahn et al. (2008) and Mansour et al. (2005), compared chronotype preference in inpatient BD ($n = 98$), to MDD ($n = 98$) and controls ($n = 59$), but did not find a significant difference in chronotype preference between the two diagnostic groups. One of the aims of the present research was to investigate the relationship between sleep and vulnerability to BD. To this end, based on findings by Wood et al. (2009), Ahn et al. (2008), Mansour (2005) and Bullock et al. (2014), the chronotype of individuals along the BD spectrum was examined.

3.5 Summary of Chapter 3

Literature reviewed in Chapter 3 provides strong evidence for links between BD and sleep. This chapter described normal sleep, including physiological observations during wakefulness and the different stages of sleep. The chapter then summarised normative sleep changes across the lifespan, with the main changes being that both SWS and REM decrease with age, and N1 and N2 increase with age. Entrainment of the circadian system was then described, with the strongest zeitgeber being light. The neurohormones involved in normal sleep were discussed, as were findings linking the regulation of the neurohormones of dopamine, serotonin, norepinephrine and cortisol to BD. The notion of trait-like and state sleep was discussed, and the absence of a trait-like
measure of sleep noted. The impact of sleep disturbance on healthy individuals was also briefly discussed in terms of the effects on cognitive functioning, perception of self and interpersonal skills, mood changes, poorer coping choices, and an increase in self-reported symptoms of affective psychopathology. Sleep disorders were briefly mentioned with insomnia being a focus as there is considerable overlap between symptoms of insomnia and a diagnosis of BD.

Then, findings regarding sleep and BD were described. Sleep disturbances in BD occur as early as adolescence and continue into adulthood, with 70% of individuals with BD also suffering from sleep disturbances. Not only do individuals with BD suffer from sleep disturbances (even during periods of euthymia), but sleep disturbance is a prodrome to BD symptoms and episodes, and sleep disturbances predate BD diagnosis. Differences in the sleep architecture of individuals with BD were described with overall findings indicating a difference in REM density, SWS, sleep efficiency, and total wake time. The chapter provided a brief description of some studies finding sleep deprivation (in combination with other therapies such as light and medications) to improve mood in BD depression, showing that not only is sleep of individuals with BD different, but that sleep can be manipulated to improve mood in those with BD. The chapter concluded by describing chronotype, specifically research investigating Morningness/Eveningness preference in individuals with BD. The studies revealed mixed findings, with some observing a clear evening chronotype in BD, and others not finding any significant differences between BD, UD and healthy controls. Overall, this chapter presented multiple works all strongly indicating that there are both state differences and trait-like differences in the sleep of BD.
4.0 Coping

4.1 Overview of Chapter 4

This chapter will begin by defining and describing *coping*. It will then describe coping styles observed in individuals diagnosed with BD. This chapter then provides a brief description of findings regarding coping styles, response to BD treatment, and research investigating the interaction of personality factors and coping styles in individuals with BD. Finally the chapter closes with discussion of prodromes in manic and depressive episodes of BD.

4.2 Coping

Coping is an important clinical focus in psychotherapy. Unlike personality and temperament, it can be targeted by therapeutic intervention (Lam et al., 2005; Moon et al., 2014; Parikh et al., 2013; Stafford, 2013). New therapeutic approaches and avenues could be opened up if researchers can establish the role of coping styles in emotional consequences in BD. The present project investigated the role of coping in models that included vulnerability to BD (with one model including proneness to depression, specifically sGBI-Depression scores, and the second model including proneness to hypo/mania, specifically sGBI-Hypomania scores), sleep, and mood/wellbeing variables. To date there are no studies examining the interaction between sleep and coping in a BD population, and so to firstly investigate if coping and sleep interact to impact outcome in the general population, the present project used a non-clinical sample. The following sections will briefly describe coping in general and then coping styles and strategies employed by individuals with BD, however first the rationale for employing a general population (analogue study) will be described.

As described in section 1.3, the main assumption that the present project assumes is that BD occurs along a spectrum, that it is dimensional in nature. If BD
were to occur or not occur (categorical in nature) in an individual then no previous research studies would employ members of the general public, related but unaffected relatives, or even ‘high risk’ relatives. However, BD research has used such samples (e.g. Duffy & Carlson, 2013; Green et al., 2011; Piguet et al., 2015; Savitz, van der Merwe, & Ramesar, 2008; Wessa, Kollmann, Linke, Schönfelder, & Kanske, 2015). Additionally, measures have also been developed that capture the range of severity, from symptoms (observed in children and adolescents) to the full-blown manifestation of BD (in adulthood), namely the GBI (Depue et al., 1989) and the 7 Up 7 Down Inventory (Youngstrom et al., 2013). Furthermore, findings from BD studies that have used analogue samples have observed similar findings to those that have used clinical samples (e.g. Bullock et al., 2014; Lozano & Johnson, 2001; Murray et al., 2007; Piguet et al., 2015). Indeed a limitation of analogue studies is that validation of treatment programs, or models that attempt to explain a clinical condition, cannot be validated until a clinical sample is used (Abramowitz et al., 2014). A benefit of employing analogue studies is that researchers are able to investigate if research questions (or models, such as the present project) have any basis in the real world before costly (including financial, access to resources, and time) research methods involving clinical samples are begun. Finally, other benefits concern sample size, as larger samples can be collected, and severity progression, as analogue studies are helpful in investigating the progression of severity of a clinical condition (Abramowitz et al., 2014).

The literature on coping is heterogenous, mainly because of the differing theoretical perspectives on the construct (Beutler, Moos, & Lane, 2003). The present thesis defines coping as the behavioural and cognitive efforts of the individual to deal with or reduce a particularly stressful event or situation (Carver & Connor-Smith, 2010; Lam & Wong, 2005). The way a person manages a stressful event can either increase or
decrease the impact the event has in the short- and long-term (Carver & Connor-Smith, 2010; Skinner & Lei, 1980). The ultimate drive in the coping literature is the attempt to understand why some people manage better than others (Folkman & Moskowitz, 2004). The ability to cope with stress determines the likelihood of a BD diagnosis being made or symptoms worsening (Grassi-Oliveira, Daruy-Filho, & Brietzke, 2010). Furthermore, coping may be investigated as a (as of yet unestablished) mediator in the effectiveness of therapeutic interventions (Grassi-Oliveira et al., 2010). Meaning that, the type of coping styles employed may change the effectiveness of a therapeutic intervention. For example, if an individual is over-using an unhelpful coping strategy such as denial or substance abuse, this could hinder any progress that may be made if that same individual were open to positive change or were able to fully absorb the therapeutic content because they were not under the influence of psychotropic substances.

The definition and measurement of coping differs across theorists. Some researchers view coping styles as state-dependent, changing more easily than defence mechanisms and, thus, being a better platform to focus shorter-term therapy (Kramer, 2010). Others posit that coping can also be described as a trait-like response, depending on the situation and stressor (Beutler et al., 2003). Coping can be further classified as adaptive or maladaptive (Fletcher, Parker, & Manicavasagar, 2013). Adaptive coping is often associated with behaviours such as taking active steps to decrease or remove the stressor, planning, and seeking support (emotional and instrumental; Carver, Scheier, & Weintraub, 1989); whereas maladaptive coping is associated with behaviours such as denial, disengagement (mental and behavioural), alcohol use (Carver et al., 1989), and rumination (Nolen-Hoeksema, 1991).

A popular coping scale that measures both adaptive and maladaptive coping styles is the Brief COPE (Carver, 1997). It is a 28-item scale that asks participants how
they respond when they confront difficult or stressful events. Items examine 14 different coping styles such as active coping, acceptance, using instrumental support, denial, behavioural disengagement and religion. Another advantage of using the Brief COPE is that it can be reduced to three or four factors (Kapsou, Panayiotou, Kokkinos, & Demetriou, 2010; Sadeh et al., 2004). This coping scale was employed in the present project.

4.3 Coping and BD

Several studies have investigated the coping styles of individuals diagnosed with BD (Fletcher et al., 2013; Goossens, Knopvert-van der Klein, & van Achterberg, 2008; Green et al., 2011; Kramer, Drapeau, Khazaal, & Bodenmann, 2009; Pavlickova et al., 2013; Thomas, Knowles, Tai, & Bentall, 2007). Compared to UD, BD individuals exhibit higher levels of maladaptive coping, such as rumination and avoidance (Green et al., 2011), potentially leading to higher relapse rates (Lam, Wong, & Sham, 2001). In contrast, adaptive coping responses, such as active coping, may improve outcomes in BD (Greenhouse, Meyer, & Johnson, 2000). Individuals with BD tend to employ more maladaptive coping styles than controls (Fletcher et al., 2013; Goossens et al., 2008; Green et al., 2011; Kramer et al., 2009), UD patients (Fletcher et al., 2013), or unaffected relatives (Green et al., 2011).

4.3.1 Coping in BD versus healthy controls.

Goossens et al. (2008) conducted a cross-sectional study investigating the coping styles of male \( n = 55 \) and female \( n = 102 \) BD outpatients compared to male \( n = 1,493 \) and female \( n = 712 \) healthy control participants. Participants provided details of demographic variables of age, sex, education, living situation, diagnosis, number of depressive and manic episodes, medication use, number of hospitalisations, treatment, and relapse prevention techniques. Participants also completed the Utrecht Coping List
(Schreurs, Van de Willige, & Tellegen, 1988), a 47-item self-report questionnaire that asks respondents to rate how often they employ one of seven coping styles when confronted with an unpleasant event. The seven subscales are Active Approach, Avoidance, Expression of Emotions, Palliative Reaction, Passive Reaction Pattern, Reassuring Thoughts, and Seeking Social Support. Male outpatients endorsed coping styles of Avoidance, Seeking Social Support, and Passive Reaction Pattern significantly more than the male control group, whereas the male control group endorsed Active Approach and Expression of Emotions significantly more than male outpatients. Female outpatients endorsed Avoidance and Passive Reaction Pattern significantly more than female control subjects, and female control subjects endorsed employing Active Approach and Reassuring Thoughts significantly more than female outpatient participants.

Four of the demographic variables measured by Goossens and colleagues moderated the type of coping styles used. Firstly, there was a significant difference in active coping style between outpatients who used the relapse prevention technique of an action plan and those who did not. Surprisingly, outpatients who had participated in a group psychotherapy course employed more passive coping styles than those who did not participate in group psychotherapy. Moreover, outpatients who previously received cognitive behaviour therapy used significantly more passive and avoidant coping styles than those who had not undergone CBT. And finally, the demographic variable of education affected the type of coping style employed—individuals who had tertiary education significantly endorsed the use of active coping styles more than individuals with a primary education.

Goossens et al. (2008) further compared coping styles between outpatients who had 0–4, 5–9, and 10 or more hypo/manic and depressive episodes. When the number of
hypo/manic episodes and coping styles were compared, the only coping style that was significantly different was that of passive reaction pattern, which was increasingly used as the number of hypo/manic episodes increased. When the number of depressive episodes and coping styles were analysed, again passive reaction pattern was used more as the number of depressive episodes increased. This increase was significant between the three groups. The active approach coping style was used less as participants reported more depressive episodes; however, statistical significance only occurred between the 0-4 episode group and the greater than 10 episode group. These findings suggest that more passive and less active coping styles are employed as the number of BD episodes experienced increases, suggesting the more episodes someone with BD experiences, the less control they attempt to assert over the course of their illness.

Kramer et al. (2009) compared the coping styles of 30 BD I inpatients and 30 matched controls. They employed the Coping Inventory for Stressful Situations (CISS; Endler & Parker, 1988), which measures three dimensions of coping (emotion-orientated, task-orientated and avoidance), and the observer-rated scale, Coping Action Patterns (CAP; Perry, Drapeau, Dunkley, & Blake, 2005), which measures coping actions (affective, behavioural and cognitive). Analyses revealed that BD inpatients employed affective and behavioural aspects of support seeking, and opposition (CAP) more than the matched control group, and employed self-reliance (behavioural aspect) and task-oriented coping less than the matched control group. Overall coping functioning (a score derived from the CAP) was lower in BD patients. The coping styles of support seeking, opposition and distraction were noted as high frequency coping styles in BD. Kramer and colleagues (2009) concluded that individuals with BD employed more maladaptive coping strategies.
Using the Response Style Questionnaire (RSQ; Nolen-Hoeksema, 1991), Thomas et al. (2007) examined response styles to depressed mood in 73 BD patients (14 experiencing a depressive episode, 30 experiencing a manic episode, and 29 in remission) and 44 controls. The only significant differences between the three BD subgroups and controls were for active coping, rumination, and risk-taking (Thomas et al., 2007). More detailed analyses found: a non-significant trend for manic BD participants to endorse active coping more than depressed BD participants, participants in remission, and controls; remitted BD patients ruminated more than controls, manic participants and depressed BD participants; and manic BD participants reported more risk-taking behaviours than controls, remitted BD participants, and depressed BD participants.

In summary, when comparing the coping styles of individuals with BD to matched healthy controls the following were noted. Female outpatients used more passive and avoidant coping styles than healthy controls (who employed more active coping strategies). Education had a positive effect on the type of coping styles employed. Having a relapse prevention plan in place improves outcome. The number of episodes experienced appears to influence the coping styles employed, with more maladaptive styles (passive) employed as number of BD episodes increases, and surprisingly, participation in either group psychotherapy or CBT therapies both saw a rise in passive coping styles.

4.3.2 Comparing coping in BD, healthy controls and UD.

Fletcher et al. (2013) conducted a cross-sectional study employing online questionnaires. They compared coping styles and responses of 94 BD I, 114 BD II, 109 UD, and 100 healthy control participants. They used a wide range of coping measures, including the Brief COPE (Carver, 1997), the Responses to Positive Affect
Questionnaire (17 items measuring Emotion-Focussed rumination, Self-Focussed rumination and Dampening in response to positive affect; Feldman, Joormann, & Johnson, 2008), the RSQ (48-items that measure Rumination, Adaptive coping and Risk-Taking in response to negative affect; Nolen-Hoeksema, 1991), the Coping Inventory for Prodromes of Mania (CIPM; 23-items measuring Denial or Blame, Seeking Professional Help, Stimulation Reduction and Problem-directed Coping in response to manic prodromes; Wong & Lam, 1999) and the Cognitive Emotion Regulation Questionnaire (CERQ; 36-items measuring nine cognitive strategies used in response to a threatening or stressful event to regulate emotion; Garnefski, Kraaij, & Spinhoven, 2001).

Healthy participants reported using more adaptive coping strategies, such as positive reframing (Brief COPE), planning (Brief COPE), active coping (Brief COPE), instrumental support (Brief COPE), emotional support (Brief COPE), positive refocussing (CERQ), positive reappraisal (CERQ), planning (CERQ), and adaptive coping (RSQ) and less maladaptive coping strategies than each of the patient groups (Fletcher et al., 2013). There were significant differences between the patient groups on both style of coping and coping responses with respect to positive and negative affect. In the context of negative affect, both BD groups reported a higher use of the Risk Taking strategy (as measured by the RSQ) than UD participants; and in the context of positive affect, both BD groups were significantly more likely to employ Self-focussed and Emotion-focussed rumination than the UD participants (Fletcher et al., 2013). When coping with negative mood, the BD II group endorsed items for Emotional Support and Instrumental Support less often than the BD I participants. With regard to coping styles and manic prodromes, BD I participants were more likely than BD II to employ
adaptive coping styles, specifically the strategies of Seeking Professional Help, Stimulation Reduction and Problem Directed Coping.

The researchers then controlled for treatment status, current medications, and mood. When controlling for medication use, analyses indicated that BD I respondents no longer significantly differed to UD respondents on Emotion-focussed and Self-focussed rumination, but BD II participants still significantly differed on these two scales. Risk-taking was no longer significantly different between the three groups. Medication effects did not influence results regarding Emotional Support (Brief COPE) and the three subscales from the CIPM (Problem-directed coping, Seeking Professional Help and Stimulation Reduction) between BD I and II respondents, as they remained significantly different between the two groups. When controlling for treatment status, both BD groups and UD group differed significantly again on Emotion-focussed and Self-focussed rumination and Risk Taking. Similarly, when controlling for treatment status, the BD subgroups continued to differ on Seeking Professional Help, Stimulation Reduction, Problem Directed Coping and Emotional Support (BD II scored lower than BD I), but Instrumental Support emerged as a difference between BD I and BD II groups (BD II scored lower). The BD II group consistently employed more maladaptive coping styles when medication effects were controlled. Fletcher et al. (2013) suggested that treatments should be modified for BD II patients to take into account their more maladaptive coping style (when using BD tailored therapies). They specifically suggested incorporating adaptive strategies (such as seeking instrumental and emotional support) and making BD II patients aware of their coping style to facilitate personal insight by challenging hypomania-relevant appraisals (Fletcher et al., 2013).

In summary, Fletcher et al. (2013) found that individuals with either BD I or II endorse more maladaptive coping strategies (increased risk taking, self-focussed and
emotion-focused rumination) when compared to healthy controls and individuals with UD. When medication was controlled for, the coping styles employed by individuals with BD I no longer significantly differed from those with UD; however, when treatment status was controlled for, the BD I and BD II groups again differed from the UD group on the coping strategies of risk-taking, self-focused and emotion-focused rumination. Furthermore, the BD II group employed more maladaptive coping styles than the BD I group, suggesting that treatment needs to be tailored to focus on shifting coping styles from maladaptive to adaptive coping styles.

### 4.3.3 Comparing coping in BD, healthy controls and unaffected relatives.

Green et al. (2011) examined self-report cognitive strategies employed in response to a threat or stressful life event by individuals with BD I (n = 105), unaffected relatives of individuals with BD (n = 124), individuals considered to be vulnerable to BD, and healthy controls (n = 63). Participants completed the Depression Anxiety Stress Scale (DASS; Lovibond & Lovibond, 1996), the Hypomanic Personality Scale (HPS; Eckblad & Chapman, 1986), and the Cognitive Emotion Regulation Questionnaire (CERQ; Garnefski et al., 2001). The CERQ consists of 36 items enquiring about emotion regulating cognitive strategies that make up the nine subscales of Self-Blame, Other-Blame, Rumination, Catastrophising, Putting into Perspective, Positive Refocussing, Positive Appraisal, Acceptance, and Refocus on Planning. The three groups differed on Rumination and Self-Blame, with BD participants scoring higher than unaffected relatives, who scored higher than healthy controls (Green et al., 2011). The BD participants also scored higher on Catastrophising than both the unaffected relatives and healthy control participants. The BD I group reported lower scores on the Putting into Perspective subscale than the unaffected relatives.
The researchers then analysed how well the CERQ subscales predicted HPS and DASS subscale scores in the three groups. Depression subscale scores were predicted by: low Refocus on Planning and high Rumination in BD I; high Catastrophising and Self-Blame, and low Putting into Perspective in unaffected relatives; and high Self-Blame and low Positive Reappraisal in healthy controls. Anxiety subscale scores were predicted by: Rumination for BD I participants; again high Catastrophising and Self-Blame, and low Putting into Perspective for unaffected relatives; and again Self-Blame and low Positive Reappraisal in healthy controls. Stress was predicted by: high Rumination and low Positive Reappraisal in the BD I group; by again high Self-Blame and Catastrophising and low Refocus on Planning in the unaffected relatives; and by Self-Blame alone in healthy controls. Hypomanic temperament was predicted by: Rumination in the BD I group; by Catastrophising in the unaffected relatives group; but by none of the cognitive strategies in the healthy control group. Self-blame consistently predicted DASS scores in the healthy controls and unaffected relatives, Catastrophising consistently predicted DASS scores in unaffected relatives, and Rumination consistently predicted DASS and HPS scores in the BD I group. Green et al. concluded that using maladaptive cognitive strategies may predispose someone to mood dysregulation, and suggested that therapeutic interventions target cognitive strategies involved in the regulation of emotion.

Several findings from the aforementioned study are directly relevant to the present project. Maladaptive strategies were consistently overused by both the BD I and unaffected relative group. The present project employed a non-clinical sample; thus, it would be expected that individuals who are higher up on the spectrum of vulnerability to BD would employ more maladaptive coping strategies than those low on the spectrum. Findings by Green et al. also support the current project in testing two
models, one including depression scores and the other including hypomania scores. If Green et al. had merged the depression and hypomania scores into an overall BD symptom score, then they would not have observed the subtle differences between the prediction of depression and prediction of hypomania.

4.3.4 Coping styles in BD.

The type of coping response style has been found to impact on subsequent mood in BD. Pavlickova et al. (2013) examined symptoms of BD, coping responses from the Response Style Questionnaire (Nolen-Hoeksema, 1991), mood (positive and negative affect), and self-esteem. Pavlickova and colleagues prospectively measured and explored interactions between mood, self-esteem and coping strategies in BD. Participants were 48 BD individuals; 28 were in remission, 12 were experiencing a depressive episode, and 8 were experiencing a hypomanic episode. Participants' responses at two time points (up to six days apart) were measured. Mania and depression symptoms were associated with high state negative mood, low state positive mood, and low state self-esteem. With respect to coping, depression was associated with higher levels of adaptive coping, risk-taking and rumination. Negative mood was positively associated with rumination, and positive mood was negatively associated with rumination. When all variables (self-esteem and mood) were entered into multiple regression analyses, the only significant predictor of rumination at a later time point was mood. Negative mood resulted in increased rumination at a later time point, and positive mood resulted in decreased rumination. Risk-taking was predicted by high state positive affect and separately also by low state negative affect; however, again when all variables were examined at a later time point, only positive mood remained a significant predictor of risk-taking. With respect to predicting mood at the second time point, Pavlickova et al. found that an increase in state positive mood was predicted by both
adaptive coping and risk-taking while rumination predicted lowered self-esteem and positive mood at the second time point. The only model that was significant when follow-up analyses were conducted was one in which symptoms of mania and rumination predicted positive mood. Further analyses revealed that, in individuals who had lower baseline symptoms of mania, rumination predicted a decrease in positive mood, but this was not the case for individuals who had higher baseline symptoms of mania (Pavlickova et al., 2013). This study highlights that the coping styles employed by those with BD impact on state mood and self-esteem. This study also showed the coping styles employed by individuals with BD had a different impact depending on their current symptomatology (low versus high mania symptoms).

4.3.5 Coping style, BD, and response to treatment.

Parikh et al. (2013) compared the coping styles and demographics of people with BD who had undergone either 20 weeks of individual CBT treatment (50 minutes per session) or six didactic group psycho-education sessions (90 minutes per session). Parikh et al. administered the Coping Inventory for Prodromes of Mania (Wong & Lam, 1999) before treatment and then 18 months after treatment. The only significant differences concerned coping styles. Parikh et al. (2013) found that both treatment groups had a significant decline in depressive and manic symptomatology following therapy. Specifically, the coping styles of stimulation reduction and problem-directed coping were significantly increased at the follow up assessment, and a significant decrease in the coping style of denial or blame was observed at the follow up. The only significant interaction effect occurred for the blame/denial coping style: only the CBT therapy reduced participants' use of denial or blame. The authors noted that although blame and denial decreased, it did not improve participants' clinical outcome (Parikh et al., 2013). Parikh et al. concluded that both CBT and group psychotherapy helped
participants employ healthier coping styles when they noticed prodromes of mania (specifically problem-directed coping and reducing stimulation), and that they shared the effect of modifying coping styles in participants with BD I and II.

4.3.6 BD, coping, and personality traits.

Coulston et al. (2013) looked at the interaction between coping and personality traits in euthymic BD (77; BD I $n = 47$ and BD II $n = 30$) and euthymic UD ($n = 96$). They measured personality (based on the FFM), depression, anxiety, stress, fear of negative evaluation, mood disorders, dysfunctional attitude, self-esteem, social adjustment and coping (using the COPE; Carver et al., 1989). Although there were no significant differences between the two diagnostic groups on the COPE subscales, the BD group had higher scores on the subscales of Planning, Planning, Positive Re-interpretation, Active Coping and Use of Instrumental Social Support than UD participants. Analyses revealed that the UD and BD groups differed on all five personality factors. Although the BD group had higher levels of E than the UD, the levels of E in the BD group were within the normal range of personality. Coulston and colleagues suggested that it was this 'preservation' of E scores that also linked to higher adaptive coping strategies reported by BD participants, as they reported being more likely to talk about problems and feelings with their support network, were less shy, had more contact with individuals outside the family, and were more likely to depend on family members if they needed help. The authors also found no significant differences between the BD I and BD II participants on personality and coping scores. Coulston et al. concluded that, as BD individuals tend to function better than UD individuals in euthymia, maintenance therapy should focus on adaptive coping skills and cognitive strategies.
A strength of Coulston et al.’s study was the incorporation of personality when examining coping styles, showing that personality factors, such as E, may partially explain why an individual may cope a certain way. This study suggests that individuals with BD employ more active and adaptive coping strategies than individuals with UD (Coulston et al., 2013); however, this stands in contrast to previous research indicating that individuals with BD use more maladaptive coping strategies than healthy controls (Green et al., 2011), and that individuals with BD II use more maladaptive strategies than individuals with BD I (Fletcher et al., 2013).

4.4 Coping and Prodrome Management in BD

Prodromes are the symptoms first recognised at the onset of a manic or depressive episode (Lam & Wong, 1997). They can be idiosyncratic, in that they are most likely the product of psychological and biological experiences of the individual (Lam & Wong, 2005). Since individuals with BD are able to recognise and describe their own prodromal symptoms (or can be encouraged to develop these skills), this awareness can be incorporated into psychosocial management of BD (Lam et al., 2001).

The prodromes most commonly identified before the onset of a manic episode include loss of interest in sleep, sleep loss, increase in sociability, racing thoughts, increased goal-directed behaviours, excitability and restlessness, overspending, and increased self-esteem (Lam et al., 2001). In line with the findings by Lam et al. (2001), Parikh et al. (2007) found that individuals who employ coping strategies that involve modifying excessive prodromal behaviours, such as reducing the amount of things to be done to a realistic goal, relapsed significantly less when compared to people who do not employ these strategies. Recent research has also found that individuals with BD report avoiding rewarding activities in an attempt to prevent ascent into a manic episode (Edge et al., 2013).
Prodromes identified before the onset of a depressive episode include a range of difficulties including: getting out of bed, staying motivated, being interested in others or activity, being happy, avoiding worrying, staying asleep, and ignoring the urge to cry (Lam & Wong, 2005; Lam et al., 2001). Individuals who employed behavioural coping strategies, such as getting organised, relapsed significantly less (Lam et al., 2001). Individuals who employ passive coping strategies, such as increasing sleeping tablet use, may relapse more frequently (Lam et al., 2001).

4.6 Summary of Chapter 4

This chapter began with a brief discussion of coping and its conceptualisation, including the core distinction between adaptive and maladaptive coping strategies. The Brief COPE, the coping measure employed in the present study, was also briefly described. The chapter then went on to describe findings of studies examining coping in BD individuals compared with healthy controls, individuals with UD or unaffected relatives. Overall, research indicates that those with a BD diagnosis use more maladaptive coping styles such as rumination and risk taking than healthy controls and unaffected relatives. However, it was also found that unaffected relatives also employ maladaptive coping strategies more than healthy controls, reinforcing the theory that BD should be conceptualised along a spectrum. The number of episodes experienced by an individual with BD appears to negatively impact the type of coping strategy used (becoming more passive as the number of episodes increase). Studies investigating the interaction between personality and coping, and mood and self-esteem on coping were also described. The chapter finished by briefly discussing prodromes observed in individuals with BD.
5. The Present Project

5.1 Overview of Chapter 5

This chapter briefly describes the project's overarching models, namely the BD, Coping, and Sleep models. The aims will be discussed, followed by a description of the variables used to test the models. The chapter will also introduce and discuss the mood and wellbeing outcome variables that were also employed in the present project, as mood/wellbeing outcome is conceptualised as the ultimate measure of outcome. The chapter will finish with the hypotheses of the present project.

5.2 Current Project

The ultimate clinical aim of psychological research into BD is to identify areas that the individual with BD can work on to improve their mental health and wellbeing. The overarching aim of the current project was to improve understanding of BD by investigating, using self-report measures in a non-clinical sample, relationships between constructs that have attracted significant attention in the BD literature but not previously been tested in a comprehensive framework. This broad aim was concretised in the BD, Coping, and Sleep models. The previous chapters have described variables that all have been found to influence vulnerability to BD, and thus, the models were developed and shaped by the research reviewed. However, it should be noted here that: the order in which the variables appear in the models was based on the theoretical findings of previous research and the novel idea that sleep be separated into trait-like and state; that alternative models could have been investigated; and that the author imposed the direction of the arrows on to the BD, Coping, and Sleep models.

Driven by the clinical question of how might a person vulnerable to BD moderate the potentially serious consequences of poor sleep for mood and wellbeing, the BD, Coping, and Sleep models have a primary focus on coping styles and/or state
sleep as potential mediators in the models relating trait-like sleep variables (in a modified measure of trait-like sleep), trait vulnerability to BD (measured by the sGBI, see below), in relation to normal range outcome measures, specifically satisfaction with life (SWL) and state mood (specifically, positive affect [PA] and negative affect [NA]). For completeness, preliminary analyses investigated the related trait variables of the FFM of personality (measured by the API; Murray et al., 2009) and BIS and BAS (measured by the BIS/BAS scales; Carver & White, 1994), however personality and motivation factors were not the focus of the present project.

Figure 4 displays the BD, Coping, and Sleep models. Although one model is presented, the sole point of difference between the two models is the “DEP/HYP” variable. One of the models included vulnerability to BD through sGBI-Depression scores, and the other model included sGBI-Hypomania scores. All other variables included in analyses were the same for both models. As in most figurative representations, causality is assumed to run broadly from left to right in the figure. On the figure's left are variables conceptualised as temperamental in nature (closer to genotype), specifically trait-like sleep variables and BIS/BAS scores, followed by trait vulnerability to BD (first model used sGBI-Depression scores and the second model used sGBI-Hypomania scores) and personality; in the middle, the putative mediators of coping styles and state sleep; and finally on the right hand side the outcome variables, of mood (PA and NA) and wellbeing (SWL scores). The solid arrowed lines represent relationships demonstrated in previous research and reviewed above; the dotted lines depict the focus of investigation in the current project.
Figure 4. Conceptual models and hypotheses. The sole point of difference between the two models concerned the “DEP/HYP” variable. The first model included “DEP” scores (representing vulnerability to BD through the use of sGBI-Depression scores), and the second model included “HYP” scores (representing vulnerability to BD through the use of sGBI-Hypomania scores). All other variables were the same for both models. Grey solid lines depict known correlations. The dotted black and solid red arrows depict the unknown relationships and the focus of the present research. The numbers depict the hypotheses tested. The variables of BIS/BAS and Personality are shaded as they were only involved in preliminary analyses.

5.3 Aims

The present project pursued four aims in a single cross-sectional study. The first was to explore previously determined associations between personality, BIS/BAS, and vulnerability to BD. The second aim was to conduct a novel investigation of trait-like sleep variables as correlates of vulnerability to BD. The third aim was to investigate the role of state sleep within the two models. And finally, the fourth aim of the study was to investigate the role of coping within the two models.

5.4 Measuring the Variables in the Present Project

The following section will briefly describe which measures and subscale scores were used to capture the variables examined, presented in three sections; predictors,
mediators, and measuring outcomes. The psychometric properties of all the scales mentioned below will be described in the Method section.

### 5.4.1 Predictors.

Five different scales were employed to measure the different predictors in the current models; these being the sGBI (Poulios et al., 2010), the modified ASWS, the CSM (Smith et al., 1989), the API (Murray et al., 2009), and the BIS/BAS scales (Carver & White, 1994). The sGBI (Poulios et al., 2010) was used to measure vulnerability to BD using two subscale scores; sGBI-Depression scores, which were used to capture proneness to BD depression; and sGBI-Hypomania scores, which were used to capture proneness to BD hypomania. As mentioned earlier, the two models tested differed by the inclusion of either sGBI-Depression or sGBI-Hypomania scores. Trait-like sleep was captured using the modified ASWS and the CSM. The modified ASWS produced five subscale scores, these being GTB, FA, SA, RS, and WU. As previously mentioned, although the modified version of the ASWS was employed, the only psychometric tests performed on this scale was assessing internal consistency. The CSM produced an overall Morningness score. The five subscales of the API, specifically Neuroticism, Extraversion, Openness to Experience, Agreeableness and Conscientiousness scores, were used to the FFM of personality. And finally, the BIS/BAS scales, specifically BIS, BAS Drive, BAS Fun Seeking and BAS Reward Responsiveness scores, were used to measure motivation traits.

### 5.4.2 Mediators.

Coping styles and state sleep were conceptualised as mediators in the two models tested. The Brief COPE (Carver, 1997) was used to measure coping styles. The Brief COPE was reduced to three latent coping styles; Active Coping, Support Seeking, and Avoidant Coping (described in the Method section), and these three scores were
employed as predictors of outcome. State sleep was also conceptualised as a mediator in the BD, Coping, and Sleep models, and an overall state sleep score was captured by the PSQI (Buysse et al., 1989).

5.4.3 Measuring outcomes.

Because the present project aimed to understand how to improve clinical outcomes, it was important to ground the proposed models in clinically-meaningful outcome variables. The sample used in the present project was non-clinical, thus it was important to choose outcome variables that would be reliable and valid in the general population. Taking these two issues into account, the ultimate dependent variables in both models were mood and wellbeing. As the design was to test and explore the BD, Coping, and Sleep models, two normal range outcome variables were employed, these being state mood, as measured by the Positive Affect Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988), and SWL as measured by the Satisfaction with Life Scale (Diener, Emmons, Larsen, & Griffin, 1985).

Individuals vulnerable to BD have been found to endorse high levels of both positive affect and negative affect (Gruber, Kogan, Mennin, & Murray, 2013; Hofmann & Meyer, 2006; Lovejoy & Steuerwald, 1995). Hofmann and Meyer (2006) found that higher manic symptoms were significantly associated with higher positive and negative affect scores; whereas higher depressive symptom scores were significantly associated with higher negative affect scores. Similarly, individuals with cyclothymia endorsed higher state and trait positive affect than individuals with depression, and similarly elevated levels of negative affect like depressed individuals, when compared to controls (Lovejoy & Steuerwald, 1995).

The PANAS is a widely used measure of state mood and is quick to administer, generating minimal response burden. Many studies have employed the PANAS to
measure mood in clinical samples (Brouwers et al., 2013; Pérez Benítez et al., 2009; Silva, Ortiz, Quiñones, Vera-Villarroel, & Slachevsky, 2011) and non-clinical samples (Hofmann & Meyer, 2006; Suhr & Tsanadis, 2007; Talbot et al., 2010; Tanay, Lotan, & Bernstein, 2012). The present project used the PANAS to capture the mood outcome variable scores of PA and NA.

Another measure of wellbeing outcome is the individual’s perceived satisfaction with life. Many studies have examined perceived Quality of Life (QoL) of individuals diagnosed with BD (Leidy, Palmer, Murray, Robb, & Revicki, 1998; Michalak, Murray, Young, & Lam, 2007, 2008; Murray & Michalak, 2007). QoL in BD is significantly impaired compared to the general population (Arnold, Witzeman, Swank, McElroy, & Keck, 2000; Latalova, Prasko, Diveky, Kamaradova, & Velartova, 2011; Robb, Cooke, Devins, Young, & Joffe, 1997; Sierra, Livianos, & Rojo, 2005), even during euthymia (Cooke, Robb, Young, & Joffe, 1996; Robb et al., 1997), and correlates with the severity of depression experienced (Yatham et al., 2004). Individuals with BD report significant impairments in working, functioning socially, performing and accomplishing daily tasks (Yatham et al., 2004). Furthermore, individuals with BD who also suffer from sleep complaints report impaired quality of life (Giglio et al., 2009). However, as mentioned earlier, the present project focussed on normal range outcome.

Another brief and low burden questionnaire is the Satisfaction with Life Scale (SWLS) by Diener et al. (1985). The SWLS is a five-item self-report questionnaire that has also been used in both clinical (Arrindell, Meeuwesen, & Huyse, 1991; Wu & Wu, 2008) and non-clinical (Arrindell, Heesink, & Feij, 1999; Hultell & Gustavsson, 2008) populations. The SWLS was used to capture wellbeing (SWL scores) in the present project.
5.5 Hypotheses

In order to assess which personality and motivational traits had the strongest associations to vulnerability to BD, all personality and motivational traits were included in all analyses investigated for the first aim. Furthermore, as there are mixed findings as to which BAS subscale (Drive, Fun Seeking or Reward Responsiveness) is most influential in the prediction of vulnerability to BD, Hypotheses 5 and 6, were framed with the exploratory qualification ‘at least one of’ (see below). For the first aim it was hypothesised that:

1) There would be a significant positive correlation between sGBI-Depression scores and Neuroticism scores.
2) There would be a significant negative correlation between sGBI-Hypomania scores and Agreeableness scores.
3) There would be a significant positive correlation between sGBI-Hypomania scores and Extraversion scores.
4) Neuroticism scores would positively predict vulnerability to BD (in both models) in the context of all personality and motivation traits.
5) Vulnerability to hypomania would be positively predicted by at least one of the BAS subscales scores in the context of all personality and motivation traits.
6) Vulnerability to depression would be positively predicted by at least one of the BIS and BAS subscale scores in the context of all personality and motivation traits.

As described in Chapter 3, difficulties with sleep are strongly associated with BD. Sleep problems may also precede the onset of BD. As sleep appears to be as an important mechanism in BD, the project sought to investigate if sleep factors predicted vulnerability to BD in the context of already well-established personality and
motivational variables. Specifically, in Chapter 2 it was established that personality, particularly the domain of Neuroticism, is a predictor of BD, and that motivational systems, specifically the BIS/BAS systems, are associated with vulnerability to BD. Although it is believed that trait-like sleep will have an influence on vulnerability to BD, there has been an absence of research investigating the influence of trait-like sleep as a predictor of vulnerability to BD in the context of personality and motivation traits, and so Hypothesis 10 was also framed with the exploratory qualification of ‘at least one of’. For the second aim it was hypothesised that:

7) There would be a significant negative correlation between Morningness scores and sGBI-Hypomania scores.

8) There would be a significant negative correlation between Morningness and sGBI-Depression scores.

9) There would be a significant negative correlation between at least one of the trait-like sleep variables and vulnerability to BD.

10) That at least one of the trait-like sleep variables would negatively predict vulnerability to BD scores in the context of personality traits and motivation traits.

Once establishing that trait-like sleep factors were still significant predictors of vulnerability to BD in the context of measures of personality and motivational systems, the next phase of investigations was to investigate the role of state sleep as a mediator in the BD, Coping, and Sleep models. Thus, for the third aim it was hypothesised that:

11) State sleep would act as a mediator in a model relating trait-like sleep variables and vulnerability to BD (operationalised as sGBI-Depression scores in the first model and sGBI-Hypomania scores in the second model tested) and mood/wellbeing outcome variables.
Finally, most clinicians aim to try and improve outcome by examining coping styles. Chapter 4 described research that has examined coping styles employed by individuals with a BD diagnosis. The present project aimed to further understand how coping styles operate in the context of sleep, vulnerability to BD, and mood/wellbeing outcome variables. Like Hypotheses 5, 6, and 10, Hypothesis 12 was also framed with the exploratory qualification of ‘at least one of’ as it is believed that coping is involved in predicting mood and wellbeing outcomes but the literature has not provided strong direction about which will be the most influential. And so, for the fourth aim it was hypothesised that:

12) At least one of the coping styles would predict each of the mood/wellbeing outcome variables in the context of vulnerability to BD.

13) Coping styles would act as a mediator in a model relating trait-like sleep variables and vulnerability to BD (sGBI-depression scores in the first model and sGBI-hypomania models in the second model) to mood/wellbeing outcome variables.

5.4 Summary of Chapter 5

Using a non-clinical sample and a two-dimensional approach to vulnerability to BD, the single study of the present project sought to illuminate the role of sleep (conceptualised in both state and trait-like terms) and coping in mood and wellbeing outcomes. Bivariate and multivariate predictions have been introduced, and Chapter 6 details the methods used to test these predictions.
6. Method

6.1 Overview of Chapter 6

This chapter will describe the methods used in the single cross-sectional study to investigate the project's aims and hypotheses. The chapter begins by describing the overall study design, participants, procedure, and measures. The chapter concludes with a detailed description of the statistical analyses employed.

6.2 Overall Study Design

A correlational design was used. Participants were drawn from the general population. The sole exclusion criterion was age, in that participants had to be over 18 years. The constructs outlined in the BD, Coping, and Sleep models were measured using standardised self-report measures with sound psychometric properties (with the exception of the purpose-developed trait-like sleep measure). Responses were analysed using correlation, multivariate regressions, regression-based mediation analyses (Sobel test) and SEM analyses.

6.3 Participants

A total of 925 participants began the survey, however 642 participants completed the survey and so the 283 incomplete responses were deleted from the data set as they were deemed to have withdrawn their consent (see Appendix A for Informed Consent Form). Of these 642 responses, four cases were removed from the data set because they either had no variation in response (e.g., selecting '1' to every item) or were completed in an unrealistic time. Multivariate outliers (30 cases) were also deleted (see Results section). Of the 608 participants included for analyses, 461 were female (75.8 %) and 147 were male (24.2 %). Five hundred and ninety-eight participants specified their age. Mean age was 27.9 year ($SD = 10.1$), ranging from 18 to 62, and the median age was 25 years.
Self-reported presence of a mental disorder diagnosis was assessed to help characterise the sample. Approximately one-third of respondents ($n = 185$) self-reported having been diagnosed with one or more mental diseases. Diagnoses included depression (111), anxiety related disorders (58), BD (39), obsessive compulsive disorder (13), post-traumatic stress disorder (13), attention deficit hyperactivity disorder (7), eating disorder (5), panic disorder (4), personality disorder (3) such as borderline personality disorder, adjustment disorder (2), psychotic disorder (2), Asperger’s Syndrome (2), autism (1), schizoaffective disorder (1) and body dysmorphic disorder (1). The present study did not have enough of a diagnosed population to investigate the validity of the two models by separating participants into a diagnosed versus undiagnosed population. Future research could investigate how other diagnoses impact the models.

6.4 Materials

An online survey was compiled from previously validated measures. Measures were selected based on their validity and reliability, whether or not they had been used in similar research, and used in other published work.

6.4.1 Pilot study.

An initial draft of the survey was 310 items long. Following ethics approval, a pilot study was conducted to investigate user experience. A hard-copy version of the survey was printed and given to five individuals who varied in age and occupation. They were also asked how they found completing the questionnaire. The general comments made by all participants were that the questionnaire was “very long”, and “at times repetitive”. When asked which parts of the questionnaire were most repetitive, all participants referred to the General Behaviour Inventory.
Based on the pilot study, it was decided that a shortened version of the GBI employed (see below) and some other quality of life measures were also not included in the final survey. This reduced the total items to 214. The questionnaires and items included in the online survey follow.

6.4.2 Trait measures.

6.4.2.1 The short General Behavior Inventory (sGBI).

The General Behavior Inventory (GBI; Depue et al., 1981) is a commonly used, validated measure of vulnerability to BD. The GBI is a 73-item questionnaire that investigates vulnerability to unipolar and bipolar depression by assessing cyclothymic patterns over time (Goodwin & Jamison, 2007). The original GBI generates three scores: hypomania (19 items), depression (46 items), and biphasic (8 items), and when looking at four of the biphasic items, mood lability can also be analysed.

The GBI is highly sensitive (.76) and specific (.99) in detecting bipolar affective conditions (Goodwin & Jamison, 2007). The GBI has an internal consistency value of $\alpha = .90 - .96$, and good test-retest reliability correlations of .71–.75 (Depue & Klein, 1988). The participants in the pilot study found this measure to be long and repetitive; thus the sGBI was employed here (Poulios et al., 2010).

The sGBI (Poulios et al., 2010) was developed for use in this project by conducting exploratory factor analysis on previously collected GBI databases (see Appendix B). The top 20 ranking items for depression and hypomania/lability were selected. The psychometric properties of the sGBI were then compared with those of the full-GBI and with external correlates. Statistical analyses found that correlations produced by the sGBI were almost identical to those produced by the full-GBI. Statistical analysis comparing correlations found no statistically significant difference between the correlations produced by the sGBI and those of the full-GBI.
As with the full GBI, the sGBI is scored using a four-point scale. Responses range from 0 (*never or hardly ever*) to 3 (*very often or almost constantly*). Of the 20 items, nine items measure vulnerability to hypomania; one item measures lability; and ten items measure vulnerability to depression. Example items include: *Have there been times of several days or more when you were so sad that it was quite painful or you felt that you couldn’t stand it?* (depression) and *Have you had periods of extreme happiness and high energy lasting several days or more when what you saw, heard, smelled, tasted, or touched seemed vivid or intense?* (hypomania). High scores on the sGBI indicate higher vulnerability to BD. For the purpose of the present study, the single item that measured lability was not included and so the nine items for hypomania and the ten items for depression were employed, and the subscales of the sGBI are called sGBI-Depression and sGBI-Hypomania. The sGBI can be viewed in Appendix C.

### 6.4.2.2 Composite Scale of Morningness.

The Composite Scale is a 13-item multiple-choice questionnaire used to measure circadian preference (Smith et al., 1989). The Composite Scale has been found to be a reliable and valid measure of circadian preference in adolescent and adult samples (Randler, 2008). It has adequate predictive validity and internal consistency $\alpha$ value of .9 (Guthrie, Ash, & Bendapudi, 1995). Three of the items have five choices and the remaining items have four choices (see Appendix D). The Composite Scale categorises respondents as *evening type, intermediate type,* or *morning type*. Individuals with a total score of 22 and less are considered evening types; those with scores ranging between 23 and 43 are considered intermediate types; and those with a total score of 44 and above are considered morning types. However, the present study interpreted scores on a continuum with higher scores indicating a higher proneness to *Morningness*. 
The Composite Scale was chosen above other chronotype measures because the Horne and Ostberg (1976) Morningness-eveningness questionnaire had been found to be too lengthy for research purposes (Smith et al., 1989); the Torsvall and Akerstedt (1980) scale was found to have low internal consistency and reliability (Smith et al., 1989); and the psychometric properties of the Folkard, Monk, and Lobban (1979) scale were found to be low (Smith et al., 1989). The Composite Scale was developed using the strongest items from the Horne and Ostberg and Torsvall and Akerstedt's scales (Smith et al., 1989).

6.4.2.3 Modified Adult Sleep Wake Scale.

The modified Adult Sleep Wake Scale (modified ASWS) was employed to measure trait-like sleep behaviours (see Appendix E). The modified ASWS was adapted from the Adult Sleep Wake Scale (Fortunato et al., 2008). The items of the original scale ask respondents about their sleep over the past month. The authors' permission was sought to investigate trait-sleep, that is, instead of asking respondents about specific aspects of sleep over the past month they were asked about sleep for most of your [their] life.

The modified ASWS is comprised of 25 items asking about five behavioural domains of sleep, these being GTB (going to bed), FA (falling asleep), SA (maintaining sleep or staying asleep), RS (reinitiating sleep), and WU (returning to wakefulness or waking up). Example items include: "In general I have to make myself go to bed" (GTB); "When I'm in bed and it's time to fall asleep I am not sleepy" (FA); "After I fall asleep but during the night I awaken more than once" (SA); "After waking up during the night I drift back off to sleep easily" (RS); and "In the morning, I wake up rested and alert" (WU). Although the modified ASWS had unknown validity, the original ASWS has been found to have good reliability, and test-retest reliability has been found to
range between .67 and .82 (Fortunato et al., 2008). Like the original form, the modified ASWS is scored on a six-point Likert scale where responses range from 1 (always) to 6 (never), some questions are reverse coded, and higher scores on the modified ASWS subscales indicate good sleep.

6.4.2.4 The Australian Personality Inventory (API).

The Australian Personality Inventory (API) is a 50-item questionnaire that measures the domains of the FFM (Murray et al., 2009). The five domains of personality that are measured are Neuroticism (N), Extraversion (E), Openness to experience (O), Agreeableness (A), and Conscientiousness (C). Example items for each domain include: "Often feel blue" (N); "Feel comfortable around people" (E); "Have a vivid imagination" (O); "Believe that others have good intentions" (A); and "Pay attention to details" (C). Respondents are asked to rate how accurate each statement is using a 5-point Likert-scale ranging from 1 (very inaccurate) to 5 (very accurate). Reverse scoring is applied to 25 of the items and scale scores are calculated. Higher scores reflect higher levels of the trait. The API has been found to be an adequate measure of the five personality domains, being internally consistent and valid (Murray et al., 2009). The API can be viewed in Appendix F.

6.4.2.5 The BIS/BAS Scales.

The BIS/BAS Scales (Carver & White, 1994) are self-report measures of the BIS and BAS motivations systems (Voigt et al., 2009). As previously stated the 20-item BIS/BAS scales measure BIS, BAS Drive, BAS Fun Seeking and BAS Reward Responsiveness. Internal consistency reliabilities for each subscale are as follows: \( \alpha = .74 \) for BIS, \( .73 \) for BAS Reward Responsiveness, \( .76 \) for BAS Drive, and \( .66 \) for BAS Fun Seeking. Items are scored using a 5-point Likert-type scale. Responses range from 1 (strongly disagree) to 4 (strongly agree). Example items include: "Criticism or
scolding hurts me quite a bit" (BIS); "When I get something I want, I feel excited and energised" (BAS Reward Responsiveness); "I go out of my way to get things I want" (BAS Drive); and "I often act on the spur of the moment" (BAS Fun Seeking). High scores on each of the subscales indicate higher levels of the trait. The full BIS/BAS scale can be viewed in Appendix G.

6.4.2.6 Brief COPE.

The Brief COPE is a 28-item scale that measures both adaptive and maladaptive coping styles (Carver, 1997). The Brief COPE was employed over the full COPE to keep the total number of survey items down. There are two items each for the following coping styles: active coping ($\alpha = .68$), planning ($\alpha = .73$), positive reframing ($\alpha = .64$), acceptance ($\alpha = .57$), humour ($\alpha = .73$), religion ($\alpha = .82$), using emotional support ($\alpha = .71$), using instrumental support ($\alpha = .64$), self-distraction ($\alpha = .71$), denial ($\alpha = .54$), venting ($\alpha = .50$), substance use ($\alpha = .90$), behavioural disengagement ($\alpha = .65$) and self-blame ($\alpha = .69$; Carver, 1997). Respondents are asked to rate how they would usually react to stress using a four-point rating scale ranging from 1 (I usually don’t do this at all) to 4 (I usually do this a lot). As recommended by Carver (Carver, 1997), the statement introducing the brief COPE was taken from the Full COPE questionnaire. The decision to use the introduction from the Full COPE was made because it asks about how one ‘usually’ copes, while the Brief COPE’s introduction asks about coping styles before surgery. The Brief COPE can be viewed in Appendix H.

Previous research identified three (Sadeh et al., 2004) or four latent factors (Kapsou et al., 2010) underlying responses to COPE items. The present project conducted an exploratory factor analysis on data post-collection to see if the 14 subscales could be reduced (see Appendix I). Statistical analyses and theoretical considerations suggested that three subscales would be optimal for the current study.
The three resulting subscales were named *Active Coping* (items 2, 7, 14, and 25), *Support Seeking* (items 5, 10, and 15) and *Avoidant Coping* (items 3, 6, 8, and 16). Example items include: "I concentrate my efforts on doing something about the situation I'm in" (Active Coping); "I get emotional support from others" (Support Seeking); and "I say to myself 'this isn't real'" (Avoidant coping). A measurement model was also run using AMOS to ensure that all items contributed to their respective subscales (see Appendix I). Higher scores on subscales of the Brief COPE indicate greater usage of these coping strategies.

6.4.3 State Measures.

6.4.3.1 The Pittsburgh Sleep Quality Index.

The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) is a 24-item questionnaire that measures sleep over the past month (see Appendix J). Of the 24 items, five are rated by roommates or bed partners and are only useful for clinical information. The remaining 19 items are self-rated and inquire about sleep duration, disturbance, quality, latency, habitual sleep efficacy, use of medications, and daytime dysfunction over the last month. Items are scored using a three-point scale, with higher overall scores indicating poorer sleep. The PSQI has been found to be internally consistent (\(\alpha = .83\)) and appropriately reliable over time (Buysse et al., 1989). The PSQI captures sleep profile and daytime dysfunction (Boudebesse et al., 2014), and has been recommended for use when measures such as actigraphy are not available (Geoffroy et al., 2015).

6.4.3.2 The Positive Affect Negative Affect Schedule (PANAS).

The Positive Affect Negative Affect Schedule (PANAS; Watson et al., 1988) is a commonly used instrument composed of 10 single-word items used to describe negative affect (NA) and 10 items used to describe positive affect (PA). Examples of
items include "irritable" (NA), "interested" (PA), "strong" (PA) and "nervous" (NA) (see Appendix K). Respondents are asked to rate how much they feel each emotion using a five-point scale ranging from 1 (very slightly or not at all) to 5 (extremely). In the present study, the time frame set was right now, that is, at the present moment. The PANAS is a reliable and valid measure with adequate psychometric properties (Crawford & Henry, 2004), with internal consistency values of .89 for PA and .85 for NA (Crawford & Henry, 2004).

6.4.3.3 The Satisfaction with Life Scale (SWLS).

The SWLS is a 5-item self-report questionnaire that measures global life satisfaction (Diener et al., 1985). The SWLS is scored using a seven-point scale, with responses ranging from 1 (strongly disagree) to 7 (strongly agree; see Appendix L). An example item is "I am satisfied with my life". High scores indicate greater SWL. In the original validated sample, the test-retest correlation coefficient was found to be .82 and the scale produced an internal consistency coefficient (Cronbach’s α) of .87 (Diener et al., 1985). Across samples, internal consistency values range from .79 to .89 (Pavot & Diener, 2008).

6.4.3.4 Descriptive and demographic items.

The online survey also contained a number of single item measures to collect further demographic and descriptive information from participants. One item asked participants how many hours they slept on average. There were also three demographic items asking participants for their date of birth, gender, and where they currently reside. Another item asked if participants had been diagnosed with a mental illness. If participants ticked ‘yes’ to that item they were presented with three open questions asking to provide the diagnosis, who made the diagnosis and how long ago it was made. There was also an item that asked participants if their work involved rotating shifts.
Finally, two items at the end of the survey asked for additional comments and if participants found completing the questionnaire distressing.

6.5 Procedure

Once the survey was finalised, ethics clearance for an amended procedure was sought and granted by the Swinburne University Human Research Ethics Committee (SUHREC, Project 2009/002, see Appendix M). The survey was advertised on national and international websites (see Appendix N), to undergraduate psychology students at Swinburne University of Technology, the "snow-balling" email method and also via the author’s social media Facebook page. From the national and international websites, potential participants were able to click on a link to the survey. The first page was an Informed Consent form (see Appendix A). Anonymity of participants was maintained as no identifying data were collected.

Data collection began in August 2009 and ceased in June 2010. Data were downloaded from the Opinio website into an SPSS file. Incomplete responses (i.e., responses that were begun but not finished, \( N = 283 \)) were deleted from the dataset. Data were screened for responses that appeared to be invalid, specifically if the questionnaire was completed in an unrealistic time (less than five minutes) or if there was no variation at all in responses (for example, when selecting ‘1’ or ‘0’ for every item). Four responses met these criteria and were subsequently deleted from the data set, leaving 638 completed responses. As previously mentioned, a further 30 cases were removed as they were multivariate outliers. The remaining data were analysed using the following strategy.

6.6 Analytic Strategy

6.6.1 Preparation of data.

The statistical packages employed to analyse the data were SPSS (version 20,
IBM) and AMOS (version 20, IBM). The data set was screened for any potential data errors or missing values, through the examination of score frequencies and ranges. Missing values were replaced using the Full Information Maximum Likelihood method, as it produces the least bias (von Hippel, 2013). Data were further screened to assess suitability for inferential statistics. For more detail, the reader is directed to the Results chapter. The following sections will describe the analyses that were carried out and how they were interpreted (see Table 5 for an overview).

6.6.2 Correlational analyses.

Pearson’s $r$ was employed to calculate the associations between known trait correlates of vulnerability to depression and hypomania. Following convention in the social sciences an $r$ value between .50 and 1.00 is considered a large/strong relationship; an $r$ value between .30 and .49 is considered a medium/moderate strength relationship; and $r$ values between .10 and .29 are considered a small/weak relationship (Cohen, 1977; Rosenthal, 2012). As can be seen in Table 5, correlation analyses were used to assess the first, second and fourth aims. The first aim, which was to investigate if the present data set performed in a similar way as previous research, was investigated with correlations that explored vulnerability to BD with all personality and motivation traits. Correlation analyses were also run to investigate the second aim, which was to explore how trait-like sleep variables interacted with all other variables of personality, motivation, and vulnerability to BD. Correlation analyses were also run as preliminary analyses for the fourth aim, specifically Hypothesis 12, which was to investigate the relationships between coping styles, vulnerability to BD, and mood/wellbeing outcome variables.
### Table 5

*Analyses employed for each hypothesis (continued on next page)*

<table>
<thead>
<tr>
<th>Aim</th>
<th>Hypothesis</th>
<th>C</th>
<th>R</th>
<th>S. T.</th>
<th>SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1: There would be a significant positive correlation between sGBI-Depression scores and Neuroticism scores.</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2: There would be a significant negative correlation between sGBI-Hypomania scores and Agreeableness scores.</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>3: There would be a significant positive correlation between sGBI-Hypomania scores and Extraversion scores.</td>
<td>✓</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>4: Neuroticism scores would positively predict vulnerability to BD (in both models) in the context of all personality and motivation traits.</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5: Vulnerability to hypomania would be positively predicted by at least one of the BAS subscales scores in the context of all personality and motivation traits.</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>6: Vulnerability to depression would be positively predicted by at least one of the BIS and BAS subscale scores in the context of all personality and motivation traits.</td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>7: There would be a significant negative correlation between Morningness scores and sGBI-Hypomania scores.</td>
<td>✓</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>8: There would be a significant negative correlation between Morningness and sGBI-Depression scores.</td>
<td>✓</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

*Note. C = Correlation; R = Regression; S.T. = Sobel Test for Mediation; SEM = structural equation modelling. ✓ = test used.*
Table 5 continued...

<table>
<thead>
<tr>
<th>Aim</th>
<th>Hypothesis</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>There would be a significant negative correlation between at least one of the trait-like sleep variables and vulnerability to BD.</td>
</tr>
<tr>
<td>10</td>
<td>That at least one of the trait-like sleep variables would negatively predict vulnerability to BD scores in the context of personality traits and motivation traits.</td>
</tr>
<tr>
<td>3</td>
<td>State sleep would act as a mediator in a model relating trait-like sleep variables and vulnerability to BD (operationalised as sGBI-Depression scores in the first model and sGBI-Hypomania scores in the second model tested) and mood/wellbeing outcome variables.</td>
</tr>
<tr>
<td>4</td>
<td>At least one of the coping styles would predict each of the mood/wellbeing outcome variables in the context of vulnerability to BD.</td>
</tr>
<tr>
<td>13</td>
<td>Coping styles would act as a mediator in a model relating trait-like sleep variables and vulnerability to BD (sGBI-depression scores in the first model and sGBI-hypomania models in the second model) to mood/wellbeing outcome variables.</td>
</tr>
</tbody>
</table>

Note. C = Correlation; R = Regression; S. T. = Sobel Test for Mediation; SEM = structural equation modelling. ✓ = test used.

6.6.3 Regression Analyses.

Standard multiple regression analyses were employed to investigate how well multiple independent variables (IV’s) predicted a dependent variable (DV; Tabachnick & Fidell, 2007). Multiple regression analyses also quantify the unique contribution of each IV to the DV (Pallant, 2007). Multiple Regression analyses require the data set to
be free of outliers, to be normal in shape, and homoscedastic. The required sample size was calculated using two equations, the first to investigate how many responses were needed to run multiple correlations, and the second to ensure that individual predictors could be tested, specifically $N \geq 50 + 8m$ (where $m$ = number of IV's), and $N \geq 104 + m$, respectively. There were 21 IV's (made up of 2 from the sGBI; 5 from the modified ASWS; 1 from the CS; 3 from the Brief COPE; 4 from the BIS/BAS scale; 5 from the API; and 1 from the PSQI); thus, the minimum number of cases required were 218 and 125 to ensure the predictors could be tested. Furthermore, Green (1991, as cited in Tabachnick & Fidell, 2007) recommends that for non-normally distributed data without transformations, more cases are needed. The equation suggested to calculate this is $N \geq (8/f^2) + (m – 1)$, where $f^2$ equals .02 (small effect), .15 (medium effect), and .35 (large effect; Tabachnick & Fidell, 2007). This formula produces sample size estimates, and for present purposes calculations ranged from 43 (for a large effect size) to 420 (for a small effect size). Thus, the 608 cases obtained were adequate for multiple regression analyses.

As can be seen in Table 5, regression analyses were carried out to test five of the 13 hypotheses. Regression analyses were performed to address the first aim, specifically to investigate if: Neuroticism positively predicted sGBI-Depression and sGBI-Hypomania scores (Hypothesis 4); if at least one of the BAS subscales positively predicted sGBI-Hypomania scores (Hypothesis 5); and at least one of the BIS and BAS subscales positively predicted sGBI-Depression scores (Hypothesis 6). When investigating the predictive power of personality and motivation traits, all personality and motivation traits were included in each of the regression analyses.

In order to investigate the second aim, specifically to test Hypotheses 10, two models were run in which either sGBI-Depression or sGBI-Hypomania scores were the
dependent variable. The first model included only trait-like sleep variables (all five subscales of the modified ASWS and Morningness), and the second included both trait-like sleep variables and personality and motivation traits.

Finally, regression analyses were also employed to assess the test Hypothesis 12 (of the fourth aim), which investigated if at least one of the coping styles (Active Coping, Support Seeking and Avoidant Coping) predicted mood/wellbeing outcome variables in the context of vulnerability to BD (including either sGBI-Depression or sGBI-Hypomania scores).

6.6.3.1. Regression-based mediation analyses

Regression-based mediation analyses were employed to assess the third and fourth aim. Significance of these mediation analyses was assessed using the Sobel test Z-statistic. The Sobel test determines if the effect of the IV on the DV is reduced significantly by the inclusion of the mediator (Baron & Kenny, 1986). This test also requires the correlation between the IV and the mediator to be significant. The Sobel test equation that was employed was:

\[ Z = \frac{a \times b}{\sqrt{b^2 \times s_a^2 + a^2 \times s_b^2}} \]

where \( a \) is the unstandardized regression coefficient when the IV predicts the mediator, \( b \) is the unstandardized regression coefficient when the mediator predicts the DV when the IV is also a predictor, \( s_a \) is the standard error of \( a \), and \( s_b \) is the standard error of \( b \) (Preacher & Leonardelli, 2001).

Full mediation is shown when a significant standardised beta value between an IV and DV no longer remains significant when both the mediator and IV act as predictors of the DV (Tabachnick & Fidell, 2007). Partial mediation is shown when the
size of the standardised beta value of the IV decreases in size but is still significant
when the mediator is introduced as a predictor (Tabachnick & Fidell, 2007).

Although the Sobel test works well in large samples, if the data are not normally
distributed a better alternative is to perform bootstrapping analyses, which do not
impose assumptions about distribution (Preacher & Leonardelli, 2001). Thus, SEM with
bootstrapping analyses were run in order to confirm the regression-based mediation
analyses.

As can be seen in Table 5, regression-based mediation analyses (Sobel tests)
were employed to test if state sleep (Hypothesis 11) or coping (Hypothesis 13) acted as
a mediator in models relating trait-like sleep variables, vulnerability to BD (either sGBI-
Depression or sGBI-Hypomania scores), and mood/wellbeing variables (state mood and
SWL). The regression-based mediation analyses (using the Sobel Z-statistic to
determine significance of mediation) were performed as preliminary analyses to
investigate the role of both coping and state sleep as mediators in the right hand side of
the two conceptual models, which were ultimately tested using SEM. These preliminary
analyses only investigated either state sleep or coping styles as mediators between
vulnerability to BD (either sGBI-Depression or sGBI-Hypomania scores) and
mood/wellbeing outcome variables (PA, NA, and SWL scores). The SEM analyses
were later run to assess the full conceptual models, as SEM permits the addition of
predictors (in this case the addition of trait-like sleep variables).

6.6.4 SEM: Structural Equation Modelling.

SEM is not a single technique, but a term used to describe a set of procedures
(Kline, 2011). SEM is also referred to as causal modelling, analysis of covariance
structures, covariance structure modelling, or covariance structure analysis (Kline,
2011; Tabachnick & Fidell, 2007). SEM includes path analysis, factor analysis, analysis
of covariance, analysis of variance, classical test theory, non-recursive economic modelling and principal component analysis (Holmes-Smith, 2010). SEM does not produce statistics that show causality but rather generates confirmatory statistics for a specified model (Kline, 2011).

There are three major contexts in which SEM is used. The first is a strictly confirmatory context, where one is seeking to accept or reject a model based on consistency with the data (Kline, 2011). The second is in the examination of alternative models, where more than one model is specified for a particular construct or theory, and the model in which that data fits will be accepted, and all other models will be rejected (Kline, 2011). The final and most widely used is for model generation; that is, the modification of an existing model so that it becomes parsimonious, makes theoretical sense and closely parallels the data (Kline, 2011). The present study employed SEM for model generation.

There are two broad categories of variables used in SEM, these being latent and observed (Kline, 2011). The observed variables are the data collected and can be continuous, ordinal or categorical. Latent variables in SEM are always continuous variables, as the latent variables are hypothetical factors or constructs, and are assumed to reflect a continuum (Kline, 2011). Given that the scales used here are reliable and due to the complexity of the model, a full measurement SEM was not conducted because this was outside of the scope of the present study. Instead models, with observed variables only, were analysed.

Some of the benefits of using SEM over path analysis and exploratory factor analyses alone are that SEM can: estimate relationships between dependent variables; take into consideration the possibility of correlations between measurement error; and separate the unique measurement error (Kline, 2011). Among statisticians there has
been some debate about the use of cross-sectional data in SEM. The MacArthur
approach specifies that cross-sectional data cannot be used in SEM as temporal
precedence (that the variable precedes the outcome) cannot be assumed, and so
recommends that data be collected at different time points (Kraemer, Kiernan, Essex, &
Kupfer, 2008). However, the Baron and Kenny approach does not specify the temporal
precedence eligibility criterion and thus cross-sectional data can be used (Kraemer et al.,
2008). Thus, a possible limitation of the present study was the use of cross-sectional
data in SEM analyses.

There is no equation to calculate an appropriate sample size for SEM (Kline,
2011). Instead several factors should be taken into consideration. Firstly, the more
complex the model, the larger the sample size should be; secondly, different types of
estimation methods have different assumptions and, thus, differ in appropriate sample
size required; and lastly, the distribution of the data set also impacts on the
recommended sample size, that is if the data has a non-normal distribution, then a larger
data set is required (Kline, 2011).

The present study employed the Maximum Likelihood method of estimation.
Kline (2011) recommends the N:q rule (where \( N \) is the number of cases and \( q \) is the
number of parameters in the model) for this particular method. The N:q rule is a ratio,
and it has been recommended that an ideal ratio is 20:1 (Kline, 2011). The model
examined in the present study had 59 parameters, and only 608 cases; although, this
number is small by the ratio standard (based on the parameters of the present study an
ideal \( N \) would have been 1,180), it is still above the minimum (which has a ratio of
10:1). Kline (2011) cites that a common sample size is 200. If this number were to be
considered, then 608 cases would be more than adequate. Lastly, there is also the issue
of having too many cases, where too many cases may inflate the significance of the model, but again no absolute value has been specified (Kline, 2011).

The central aim of SEM is to find support that the specified model (implied covariance matrix) and the data set (sample model) do not differ (Kline, 2011). The first statistic often checked is the model test statistic (model chi-square, $\chi^2$). If this value has a non-significant $p$-value ($p > .05$) then the specified model is accepted (Kline, 2011). If however, the model does not fit ($p < .05$) the model is not necessarily wrong. The $\chi^2$ can be affected by sample size, especially if the sample size is very large, and there are small discrepancies between the models. This can result in a statistically significant $\chi^2$ (Kline, 2011). Kline (2011) defines very large sample sizes as 5,000 cases, and typical sample sizes, that do not impact the significance of $\chi^2$, as 200 to 300. Other factors that can impact the significance value of $\chi^2$ are correlation size, unique variance, and multivariate non-normality (Kline, 2011). If there are concerns with sample size, non-normality, correlation size, or unique variance, then it is still worthwhile examining other fit indices (Kline, 2011).

Here, seven values were used to investigate the goodness of fit of the two models. These included: significance of the chi-square ($\chi^2$) was greater than .05; Bollen-Stine’s $p$, which is a significance value of the model in non-normal data sets, greater than .05; Root Mean Square Error of Approximation (RMSEA) less than .05; Standardised Root Mean Residual (SRMR) less than .06; Goodness of Fit (GFI) greater than .95; Adjusted Goodness of Fit (AGFI) greater than .95; Tucker Lewis Index (TLI) greater than .95 (but less than 1.0 as this indicates overfit); and Comparative Fit Index (CFI) greater than .95 (Holmes-Smith, 2010).

As previously mentioned, SEM analyses were also conducted to confirm the regression-based mediation analyses. SEM analyses were employed to test if state sleep
(Hypothesis 11) and/or coping (Hypothesis 13) acted as a mediator in models relating trait-like sleep variables and vulnerability to BD (sGBI-Depression or sGBI-Hypomania scores) and mood/wellbeing outcome variables (PA, NA, and SWL). Figure 5 presents the data analysis plan.
Figure 5. Data analysis plan. Statistical procedures performed.
7. Results

7.1 Overview of Chapter 7

The chapter begins with a description of data cleaning and reports descriptive statistics. The first of the data analyses addressed investigations regarding personality traits, motivation traits, and vulnerability to BD. This section will describe correlation and regression analyses. The next section will describe analyses investigating trait-like sleep variables and vulnerability to BD. The final section will describe analyses that investigated mediation by state sleep and coping variables. The chapter ends with a summary table that presents the aims, hypotheses, whether they were supported, and where evidence of their support can be found.

7.2 Data Cleaning

Data were screened for missing values, skewness, kurtosis, and multicollinearity. A missing values analysis found that the data that were missing were at random. Furthermore, as the sample size was large ($N = 638$) and there was not more than 5% missing on any variable, it was decided to impute the missing data (as recommended by Tabachnick & Fidell, 2007).

Table 6

<table>
<thead>
<tr>
<th>No. of items</th>
<th>Frequency of responses missing (% response rate)</th>
</tr>
</thead>
<tbody>
<tr>
<td>108</td>
<td>0 (100 %)</td>
</tr>
<tr>
<td>43</td>
<td>1 (99.8)</td>
</tr>
<tr>
<td>22</td>
<td>2 (99.7)</td>
</tr>
<tr>
<td>13</td>
<td>3 (99.5)</td>
</tr>
<tr>
<td>5</td>
<td>4 (99.4)</td>
</tr>
<tr>
<td>5</td>
<td>5 (99.2)</td>
</tr>
<tr>
<td>6</td>
<td>6 (99.1)</td>
</tr>
<tr>
<td>2</td>
<td>8 (98.8)</td>
</tr>
<tr>
<td>1</td>
<td>10 (98.4)</td>
</tr>
<tr>
<td>1</td>
<td>13 (97.9)</td>
</tr>
</tbody>
</table>

Note. Total items = 206
As can be seen in Table 6 the total percentage of missing values was small. The Full Information Maximum Likelihood method was then employed to impute missing values. Skewness and kurtosis values were then calculated to assess univariate normality (see Table 7). As can be seen in Table 7 the majority of variables were negatively skewed and kurtosis values were below zero, suggesting heavier tails than those found in a normal distribution. Histograms were also examined.

Table 7

<table>
<thead>
<tr>
<th>Skewness</th>
<th>Kurtosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vulnerability to Depression (sGBI-Depression)</td>
<td>0.94</td>
</tr>
<tr>
<td>Vulnerability to Hypo/mania (sGBI-Hypomania)</td>
<td>0.79</td>
</tr>
<tr>
<td>Morningness</td>
<td>-0.11</td>
</tr>
<tr>
<td>Going to Bed (GTB)</td>
<td>-0.12</td>
</tr>
<tr>
<td>Falling Asleep (FA)</td>
<td>-0.29</td>
</tr>
<tr>
<td>Staying Asleep (SA)</td>
<td>-0.36</td>
</tr>
<tr>
<td>Reinitiating Sleep (RS)</td>
<td>-0.30</td>
</tr>
<tr>
<td>Waking Up (WU)</td>
<td>-0.20</td>
</tr>
<tr>
<td>Neuroticism (N)</td>
<td>0.09</td>
</tr>
<tr>
<td>Extraversion (E)</td>
<td>-0.28</td>
</tr>
<tr>
<td>Openness to Experience (O)</td>
<td>-0.35</td>
</tr>
<tr>
<td>Agreeableness (A)</td>
<td>-0.58</td>
</tr>
<tr>
<td>Conscientiousness (C)</td>
<td>-0.20</td>
</tr>
<tr>
<td>BAS Drive (BASd)</td>
<td>0.90</td>
</tr>
<tr>
<td>BAS Fun Seeking (BASfs)</td>
<td>-0.02</td>
</tr>
<tr>
<td>BAS Reward Responsiveness (BASrr)</td>
<td>-0.39</td>
</tr>
<tr>
<td>BIS</td>
<td>-0.42</td>
</tr>
<tr>
<td>Active Coping (AcC)</td>
<td>-0.29</td>
</tr>
<tr>
<td>Support Seeking (SS)</td>
<td>-0.09</td>
</tr>
<tr>
<td>Avoidant Coping (AvC)</td>
<td>1.14</td>
</tr>
<tr>
<td>State Sleep (PSQI)</td>
<td>0.77</td>
</tr>
<tr>
<td>Positive Affect (PA)</td>
<td>0.30</td>
</tr>
<tr>
<td>Negative Affect (NA)</td>
<td>1.69</td>
</tr>
<tr>
<td>Satisfaction with Life (SWL)</td>
<td>-0.37</td>
</tr>
</tbody>
</table>

In order to test for the need to transform data, the `ladder` and `gladder` commands from the statistical package STATA version 11 (StataCorp, 2009) were employed. The
ladder command calculates chi-square values for each possible transformation of a chosen variable. The gladder command presents these analyses in graphic form (normal probability plots). The aim is to produce the smallest chi-square value possible. An example of the outputs can be viewed in Appendix O, where the variable of sGBI-Depression scores was analysed. It was determined that only six variables would be improved by transformation. Table 8 presents the variables that were transformed, how they were transformed, and new skewness and kurtosis values. Following transformation, these variables still exhibited some skew, as can be seen in Table 8.

Mahalanobis' Distance was used to screen for multivariate outliers. Mahalanobis’ Distance is the distance each case sits from the centroid and this multivariate distance is evaluated using a $\chi^2$ distribution. For example, one of the regression analyses had the proposed mediators (Active Coping, Support Seeking, Avoidant Coping, and State Sleep) as IV’s and SWL as a DV, thus the critical value for a regression with four degrees of freedom was 18.47 when $p < .001$ (Tabachnick & Fidell, 2007). All cases that had a Mahalanobis' Distance above this value were removed from the data set. This reduced the total number of cases from 638 to 608.

Table 8

<table>
<thead>
<tr>
<th>Transformed variables</th>
<th>Transformation applied</th>
<th>Skewness</th>
<th>Kurtosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>sGBI-Depression</td>
<td>Square root</td>
<td>0.93</td>
<td>0.44</td>
</tr>
<tr>
<td>sGBI-Hypomania</td>
<td>Square root</td>
<td>0.82</td>
<td>0.09</td>
</tr>
<tr>
<td>A</td>
<td>Squared</td>
<td>-0.61</td>
<td>0.36</td>
</tr>
<tr>
<td>BASrr</td>
<td>Squared</td>
<td>-0.09</td>
<td>-0.42</td>
</tr>
<tr>
<td>NA</td>
<td>Log</td>
<td>1.50</td>
<td>1.74</td>
</tr>
<tr>
<td>PSQI</td>
<td>Square root</td>
<td>0.66</td>
<td>0.06</td>
</tr>
</tbody>
</table>

Note. A = Agreeableness; BASrr = BAS Reward Responsiveness; NA = Negative Affect; PSQI = Pittsburgh Sleep Quality Index scores
7.3 Psychometric Properties of Scales

Table 9 presents the means, standard deviation, range of observed scores for each scale, the theoretical ranges of scores per scale, published means, and Cronbach’s alpha for each scale from the present sample. Table 9 indicates that the scales had adequate reliability as Cronbach’s alpha was above .7 in all cases (Bland & Altman, 1997). The mean scores were comparable to those obtained in previous research (which mainly employed undergraduate or non-clinical samples, however, for a full description of samples see Appendix P). The missing values imputation caused one case to fall outside the scale's range and was Winsorized (Tukey, 1962). This particular case had a total score of 30.55 for the SA variable, 0.55 over the theoretical maximum, and so it was replaced with the value of 30.
Table 9

**Psychometric Properties of Each Scale and Subscale**

<table>
<thead>
<tr>
<th>Scale</th>
<th>M</th>
<th>SD</th>
<th>Range (observed)</th>
<th>Range (theoretical)</th>
<th>Published means</th>
<th>Alpha (α)</th>
</tr>
</thead>
<tbody>
<tr>
<td>sGBI Depression&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7.18</td>
<td>5.26</td>
<td>0-26</td>
<td>0-30</td>
<td>11.18</td>
<td>.85</td>
</tr>
<tr>
<td>Hypomania&lt;sup&gt;b&lt;/sup&gt;</td>
<td>7.69</td>
<td>5.60</td>
<td>0-25</td>
<td>0-27</td>
<td>6.25</td>
<td>.89</td>
</tr>
<tr>
<td>Composite Scale</td>
<td>33.30</td>
<td>7.95</td>
<td>14-55</td>
<td>13-55</td>
<td>23.6</td>
<td>.90</td>
</tr>
<tr>
<td>Mod-ASWS Going to bed</td>
<td>18.01</td>
<td>6.53</td>
<td>5-30</td>
<td>5-30</td>
<td>18.0</td>
<td>.92</td>
</tr>
<tr>
<td>Falling asleep</td>
<td>19.73</td>
<td>5.77</td>
<td>5-30</td>
<td>5-30</td>
<td>20.90</td>
<td>.90</td>
</tr>
<tr>
<td>Staying asleep</td>
<td>20.31</td>
<td>5.30</td>
<td>5-30</td>
<td>5-30</td>
<td>20.1</td>
<td>.87</td>
</tr>
<tr>
<td>Reinitiating sleep</td>
<td>20.28</td>
<td>5.62</td>
<td>5-30</td>
<td>5-30</td>
<td>21.1</td>
<td>.89</td>
</tr>
<tr>
<td>Returning to Wakefulness</td>
<td>17.70</td>
<td>5.54</td>
<td>5-30</td>
<td>5-30</td>
<td>16.85</td>
<td>.92</td>
</tr>
<tr>
<td>API Neuroticism</td>
<td>28.67</td>
<td>8.41</td>
<td>10-50</td>
<td>10-50</td>
<td>25.7</td>
<td>.89</td>
</tr>
<tr>
<td>Extraversion</td>
<td>32.82</td>
<td>7.36</td>
<td>11-50</td>
<td>10-50</td>
<td>34.3</td>
<td>.87</td>
</tr>
<tr>
<td>Openness</td>
<td>37.80</td>
<td>5.85</td>
<td>13-50</td>
<td>10-50</td>
<td>36.4</td>
<td>.76</td>
</tr>
<tr>
<td>Agreeableness&lt;sup&gt;b&lt;/sup&gt;</td>
<td>37.01</td>
<td>6.06</td>
<td>13-50</td>
<td>10-50</td>
<td>37.7</td>
<td>.81</td>
</tr>
<tr>
<td>Conscientiousness</td>
<td>34.47</td>
<td>6.75</td>
<td>14-50</td>
<td>10-50</td>
<td>36.1</td>
<td>.85</td>
</tr>
<tr>
<td>BIS/BAS BAS Drive</td>
<td>9.98</td>
<td>2.29</td>
<td>4-16</td>
<td>4-16</td>
<td>10.7</td>
<td>.80</td>
</tr>
<tr>
<td>BAS Fun Seeking</td>
<td>11.24</td>
<td>2.32</td>
<td>4-16</td>
<td>4-16</td>
<td>11.9</td>
<td>.74</td>
</tr>
<tr>
<td>BAS Reward Responsiveness&lt;sup&gt;b&lt;/sup&gt;</td>
<td>16.19</td>
<td>2.15</td>
<td>9-20</td>
<td>5-20</td>
<td>17.6</td>
<td>.72</td>
</tr>
<tr>
<td>BIS</td>
<td>21.17</td>
<td>3.56</td>
<td>8-28</td>
<td>7-28</td>
<td>22.0</td>
<td>.80</td>
</tr>
<tr>
<td>Pittsburgh Sleep Quality Index</td>
<td>6.37</td>
<td>3.25</td>
<td>0-17</td>
<td>0-21</td>
<td>7.4</td>
<td>.81</td>
</tr>
<tr>
<td>Brief COPE Active coping</td>
<td>11.65</td>
<td>2.65</td>
<td>4-16</td>
<td>4-16</td>
<td>-</td>
<td>.83</td>
</tr>
<tr>
<td>Support Seeking</td>
<td>10.41</td>
<td>3.33</td>
<td>4-16</td>
<td>4-16</td>
<td>-</td>
<td>.91</td>
</tr>
<tr>
<td>Avoidant coping</td>
<td>8.21</td>
<td>2.53</td>
<td>5-18</td>
<td>5-20</td>
<td>-</td>
<td>.69</td>
</tr>
<tr>
<td>PANAS Positive affect</td>
<td>25.10</td>
<td>9.56</td>
<td>10-50</td>
<td>10-50</td>
<td>29.7&lt;sup&gt;+&lt;/sup&gt;</td>
<td>.94</td>
</tr>
<tr>
<td>Negative affect&lt;sup&gt;c&lt;/sup&gt;</td>
<td>16.18</td>
<td>7.19</td>
<td>10-44</td>
<td>10-50</td>
<td>14.8&lt;sup&gt;++&lt;/sup&gt;</td>
<td>.90</td>
</tr>
<tr>
<td>Satisfaction With Life Scale</td>
<td>21.97</td>
<td>7.55</td>
<td>5-35</td>
<td>5-35</td>
<td>23.5</td>
<td>.91</td>
</tr>
</tbody>
</table>

*Note. Mod-ASWS = Modified ASWS; Published means for sGBI (Poullos et al., 2010); Composite Scale (Smith et al., 1989); Adult Sleep Wake Scale (Fortunato et al., 2008); BIS/BAS scales (Jorm et al., 1999) (mean values were taken from age groups 18-29 mean and female sample as majority of current sample is female); Pittsburgh Sleep Quality Index (Buysse et al., 1989); Neuroticism, Extraversion, Openness to Experience, Agreeableness and Conscientiousness (Murray et al., 2009); Positive Affect and Negative Affect (+(Watson et al., 1988); ++(Crawford & Henry, 2004)); Satisfaction with Life Scale (Diener et al., 1985).*

<sup>a</sup>Summary statistics for the square root transformation of the variable.

<sup>b</sup>Summary statistics for the squared transformation of the variable.

<sup>c</sup>Summary statistics for the log transformation of the variable.
7.4 Data Analyses

7.4.1 Personality, motivation, and vulnerability to BD.

The first series of correlations considered the relationship between trait personality variables, trait motivation variables, and vulnerability to BD (sGBI-Depression and sGBI-Hypomania) scores. The results of these correlations can be viewed in Table 10.

Table 10

<table>
<thead>
<tr>
<th></th>
<th>BASd</th>
<th>BASrr</th>
<th>BASfs</th>
<th>BIS</th>
<th>N</th>
<th>E</th>
<th>O</th>
<th>A</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>sGBI Depression</td>
<td>.12**</td>
<td>.12**</td>
<td>.17**</td>
<td>.27**</td>
<td>.53**</td>
<td>-.12**</td>
<td>.10*</td>
<td>-.27**</td>
<td>-.31**</td>
</tr>
<tr>
<td>sGBI Hypomania</td>
<td>- .01</td>
<td>.03</td>
<td>.01</td>
<td>.40**</td>
<td>.72**</td>
<td>-.30**</td>
<td>.03</td>
<td>-.31**</td>
<td>-.40**</td>
</tr>
</tbody>
</table>

Note. N = 608, * p < .05, ** p < .01. sGBI = Shortened version of the General Behaviour Inventory; BASd = BAS Drive; BASrr = BAS Reward Responsiveness; BASfs = BAS Fun Seeking; N = Neuroticism; E = Extraversion; O = Openness to Experience; A = Agreeableness; C = Conscientiousness.

As can be seen in Table 10, there was a significant correlation between sGBI-Depression and Neuroticism scores, which was positive in direction and moderate in size, supporting Hypothesis 1. There was a significant negative correlation between sGBI-Hypomania and Agreeableness scores, supporting Hypothesis 2. There was a significant negative correlation between sGBI-Hypomania and Extraversion scores, so Hypothesis 3 was not supported. The other relationships worth noting were those between BIS and sGBI-Hypomania scores and between Conscientiousness and sGBI-Hypomania scores. Both of these correlations were moderate in strength and significant (p < .01). Inter-correlations for all variables in Table 10 can be viewed in Appendix Q.

Standard multiple regression analyses were conducted to further investigate the relationships between personality traits (five domains of the FFM) and motivation traits...
(four subscales of the BIS/BAS scales) as predictors of vulnerability to BD (sGBI-Depression and sGBI-Hypomania scores). For all regression analyses, preliminary analyses indicated that assumptions of multi-collinearity, homoscedasticity, outliers, and linearity were met.

Table 11 displays the results of multiple regression analyses that examined the prediction of sGBI-Hypomania scores by personality and motivation trait variables and Table 12 displays the results of multiple regression analyses that examined the prediction of sGBI-Depression scores by personality and motivation trait variables.

Table 11

Personality and Motivation Trait predictors of sGBI-Hypomania Scores

<table>
<thead>
<tr>
<th></th>
<th>Standardised Weights</th>
<th>95 % CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β</td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>.66**</td>
<td>[.39, .49]</td>
</tr>
<tr>
<td>E</td>
<td>-.06</td>
<td>[-.10, .00]</td>
</tr>
<tr>
<td>O</td>
<td>.07*</td>
<td>[.01, .13]</td>
</tr>
<tr>
<td>A</td>
<td>-.03</td>
<td>[-.09, .03]</td>
</tr>
<tr>
<td>C</td>
<td>-.11**</td>
<td>[-.14, -.03]</td>
</tr>
<tr>
<td>BASd</td>
<td>.05</td>
<td>[-.05, .28]</td>
</tr>
<tr>
<td>BASrr</td>
<td>.02</td>
<td>[-.15, .24]</td>
</tr>
<tr>
<td>BASfs</td>
<td>.10**</td>
<td>[.07, .40]</td>
</tr>
<tr>
<td>BIS</td>
<td>-.00</td>
<td>[-.12, .11]</td>
</tr>
<tr>
<td>$R^2$</td>
<td></td>
<td>.55**</td>
</tr>
<tr>
<td>$F$</td>
<td></td>
<td>81.06**</td>
</tr>
</tbody>
</table>

Note. N = 608. CI = confidence interval. N= Neuroticism; E = Extraversion; O = Openness to Experience; A = Agreeableness; C = Conscientiousness; BASd = BAS Drive; BASrr = BAS Reward Responsiveness; BASfs = BAS Fun Seeking  
* p < .05. ** p < .01.
Table 12

*Personality and Motivation Trait predictors of sGBI-Depression Scores*

<table>
<thead>
<tr>
<th>Trait</th>
<th>Standardised Weights</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>.53**</td>
<td>[.28, .39]</td>
</tr>
<tr>
<td>E</td>
<td>.01</td>
<td>[-.05, .06]</td>
</tr>
<tr>
<td>O</td>
<td>.07*</td>
<td>[.00, .13]</td>
</tr>
<tr>
<td>A</td>
<td>-.07</td>
<td>[-.12, .01]</td>
</tr>
<tr>
<td>C</td>
<td>-.10*</td>
<td>[-.14, -.02]</td>
</tr>
<tr>
<td>BASd</td>
<td>.08</td>
<td>[-.01, .36]</td>
</tr>
<tr>
<td>BASrr</td>
<td>.06</td>
<td>[-.06, .37]</td>
</tr>
<tr>
<td>BASfs</td>
<td>.17**</td>
<td>[.19, .57]</td>
</tr>
<tr>
<td>BIS</td>
<td>-.05</td>
<td>[-.20, .06]</td>
</tr>
</tbody>
</table>

\[ R^2 = .37** \]
\[ F = 38.92** \]

*Note. N = 608. CI = confidence interval. N= Neuroticism; E = Extraversion; O = Openness to Experience; A = Agreeableness; C = Conscientiousness; BASd = BAS Drive; BASrr = BAS Reward Responsiveness; BASfs = BAS Fun Seeking. *p < .05. **p < .01.*

As can be seen in Tables 11 and 12, Neuroticism positively predicted both sGBI-Depression and sGBI-Hypomania scores, and was the strongest predictor of vulnerability to BD (sGBI-Depression and sGBI-Hypomania scores) in the context of all personality and motivation traits, providing support for Hypothesis 4. The other personality traits that also significantly predicted sGBI-Hypomania and sGBI-Depression scores were Openness and Conscientiousness scores.

Hypothesis 5, that one or more of the BAS subscales would positively predict sGBI-Hypomania scores in the context of all personality and motivation traits, was also supported (see Table 11). BAS Fun Seeking was the only significant predictor of sGBI-Hypomania scores. As can be seen in Table 12, again BAS Fun Seeking was the only predictor of sGBI-Depression scores, supporting Hypothesis 6.

7.4.2 Trait-like sleep variables and vulnerability to BD.

To investigate the second aim, which was to determine whether trait-like sleep variables are correlates of BD, bivariate correlations were calculated (seen in Table 13).
Higher scores on the subscales of the modified ASWS are indicative of better sleep. Thus, poor sleep was associated with vulnerability to BD. The relationship between Morningness and sGBI-Hypomania scores was negative in direction and weak in size, but significant, supporting Hypothesis 7. As predicted there was a negative relationship between Morningness and sGBI-Depression scores supporting Hypothesis 8; however, this correlation was small in size. All correlations between trait-like sleep variables of the modified ASWS (GTB, FA, SA, RS, and WU scores) and vulnerability to BD (sGBI-Depression scores and sGBI-Hypomania scores) were negative in direction and significant, supporting Hypothesis 9. Inter-correlations between all the variables in Table 13 can be viewed in Appendix R.

The exploration of the second aim was continued by investigating if at least one trait-like sleep variable (GTB, FA, SA, RS, WU, or Morningness scores) would negatively predict vulnerability to BD (sGBI-Depression and sGBI-Hypomania scores), when forced to share variance with traits of personality (FFM) and motivation (BIS/BAS subscales; Hypothesis 10). These analyses were performed in two stages, the first stage explored the impact of trait-like sleep variables (GTB, FA, SA, RS, WU, and Morningness) alone (Model 1) on both sGBI-Depression (see Table 14) and sGBI-
Hypomania (see Table 15), and the second stage explored the impact of trait-like sleep variables in the context of both personality and motivation trait variables (Model 2).

Table 14

Predictors of sGBI-Depression Scores

<table>
<thead>
<tr>
<th>Variable</th>
<th>β</th>
<th>95 % CI</th>
<th>β</th>
<th>95 % CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>GTB</td>
<td>-.29**</td>
<td>[-.22, -.09]</td>
<td>-.19**</td>
<td>[-.22, -.09]</td>
</tr>
<tr>
<td>FA</td>
<td>-.10</td>
<td></td>
<td>-.09*</td>
<td>[-.16, -.00]</td>
</tr>
<tr>
<td>SA</td>
<td>-.11**</td>
<td>[-.13, .03]</td>
<td>-.05</td>
<td>[-.13, .03]</td>
</tr>
<tr>
<td>RS</td>
<td>-.17**</td>
<td>[-.18, -.03]</td>
<td>-.11**</td>
<td>[-.18, -.03]</td>
</tr>
<tr>
<td>WU</td>
<td>-.07</td>
<td></td>
<td>.09</td>
<td>[.00, .18]</td>
</tr>
<tr>
<td>M</td>
<td>.12*</td>
<td></td>
<td>.06</td>
<td>[-.03, .10]</td>
</tr>
<tr>
<td>N</td>
<td>.48**</td>
<td>[.24, .36]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>.04</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O</td>
<td>.05</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>-.05</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>-.08*</td>
<td>[-.13, -.00]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BASd</td>
<td>.07</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BASrr</td>
<td>.05</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BASfs</td>
<td>.14**</td>
<td>[.13, .49]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BIS</td>
<td>.04</td>
<td>[-.18, .07]</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

R² = .22
F = 28.22**

Note. N = 608. CI = confidence interval. GTB = Going to Bed; FA = Falling Asleep; SA = Staying Asleep; RS = Reinitiating Sleep; WU = Waking Up; M = Morningness; N = Neuroticism; E = Extraversion; O = Openness to Experience; A = Agreeableness; C = Conscientiousness; BASd = BAS Drive; BASrr = BAS Reward Responsiveness; BASfs = BAS Fun Seeking

* p < .05. ** p < .01.

As can be seen in Table 14, the trait-like sleep variables of GTB, SA and RS (Model 1) negatively predicted sGBI-Depression scores and when personality and motivation traits were included in the analyses (Model 2), GTB and RS remained significant negative predictors of sGBI-Depression scores. Although, Neuroticism was the largest predictor of sGBI-Depression scores, explaining 9.7 % of the additional variance, GTB scores was the next strongest predictor of sGBI-Depression scores. Furthermore, although SA was no longer a significant predictor, RS scores remained a
significant predictor, and FA became a significant predictor when personality and motivation trait variables were included in analyses, providing preliminary support for Hypothesis 10.

Table 15

*Predictors of sGBI-Hypomania Scores*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Model 1</th>
<th>Model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Standardised Weights</td>
<td>Standardised Weights</td>
</tr>
<tr>
<td></td>
<td>Model 1</td>
<td>Model 2</td>
</tr>
<tr>
<td>GTB</td>
<td>-.20**</td>
<td>-.10**</td>
</tr>
<tr>
<td>FA</td>
<td>-.11*</td>
<td>-.12**</td>
</tr>
<tr>
<td>SA</td>
<td>-.12**</td>
<td>-.05</td>
</tr>
<tr>
<td>RS</td>
<td>-.17**</td>
<td>-.07</td>
</tr>
<tr>
<td>WU</td>
<td>-.21**</td>
<td>.04</td>
</tr>
<tr>
<td>M</td>
<td>.12**</td>
<td>.02</td>
</tr>
<tr>
<td>N</td>
<td>.61**</td>
<td>.35, .46</td>
</tr>
<tr>
<td>E</td>
<td>-.04</td>
<td></td>
</tr>
<tr>
<td>O</td>
<td>.06</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>-.01</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>-.08*</td>
<td>[-.12, -.01]</td>
</tr>
<tr>
<td>BASd</td>
<td>.04</td>
<td>[-.06, .26]</td>
</tr>
<tr>
<td>BASrr</td>
<td>.01</td>
<td>[-.15, .22]</td>
</tr>
<tr>
<td>BASfs</td>
<td>.08*</td>
<td>[.02, .34]</td>
</tr>
<tr>
<td>BIS</td>
<td>.01</td>
<td>[-.10, .12]</td>
</tr>
</tbody>
</table>

Note. N = 608. CI = confidence interval. GTB = Going to Bed; FA = Falling Asleep; SA = Staying Asleep; RS = Reinitiating Sleep; WU = Waking Up; M = Morningness; N = Neuroticism; E = Extraversion; O = Openness to Experience; A = Agreeableness; C = Conscientiousness; BASd = BAS Drive; BASrr = BAS Reward Responsiveness; BASfs = BAS Fun Seeking; *p < .05. **p < .01.

As can be seen in Table 15 all of the trait-like sleep variables predicted sGBI-Hypomania scores, with the majority being negative in direction (Model 1). The WU and GTB variables were the strongest predictor of sGBI-Hypomania scores. When the trait-like sleep variables were forced to share variance with traits of personality and motivation (Model 2) in the prediction of sGBI-Hypomania scores, Neuroticism (Model 2) was the again largest contributor, explaining 16 % of the total variance. However, the
trait-like sleep variables of GTB and FA remained significant negative predictors of sGBI-Hypomania scores, providing support for Hypothesis 10.

### 7.4.3 Coping variables as predictors of mood and wellbeing outcomes.

To address the hypothesis that at least one coping style would predict mood/wellbeing outcome variables in the context of vulnerability to BD (Hypothesis 12), correlational analyses were first run to explore bivariate relationships between coping and vulnerability to BD and mood/wellbeing variables. Table 16 presents these correlations.

#### Table 16

<table>
<thead>
<tr>
<th></th>
<th>SWL</th>
<th>PA</th>
<th>NA</th>
</tr>
</thead>
<tbody>
<tr>
<td>sGBI-Depression</td>
<td>-.38**</td>
<td>-.01</td>
<td>.39**</td>
</tr>
<tr>
<td>sGBI-Hypomania</td>
<td>-.53**</td>
<td>-.20**</td>
<td>.44**</td>
</tr>
<tr>
<td>Active Coping</td>
<td>.35**</td>
<td>.37**</td>
<td>-.16**</td>
</tr>
<tr>
<td>Support Seeking</td>
<td>.25**</td>
<td>.12**</td>
<td>-.04</td>
</tr>
<tr>
<td>Avoidant Coping</td>
<td>-.35**</td>
<td>-.15**</td>
<td>.41**</td>
</tr>
</tbody>
</table>

*Note. \(N = 608\). SWL = Satisfaction with Life scores; PA = Positive Affect scores; NA = Negative Affect Scores.

* \(p < .05\). ** \(p < .01\). *** \(p < .001\)

Patterns of associations shown in Table 16 were in line with previous literature. Vulnerability to BD was associated with low PA and SWL and high NA. Active Coping scores (adaptive) were associated with high SWL and PA, as expected, and Avoidant Coping scores (indicative of maladaptive coping) were associated with poorer mood/wellbeing outcomes. As can further be seen in Table 16, SWL scores were negatively correlated with sGBI-Depression, sGBI-Hypomania, and Avoidant Coping scores, and significant positive correlations with Active Coping and Support Seeking scores. Correlations were generally of moderate to large strength.
Significant correlations were observed between PA scores and all the coping styles and sGBI-Hypomania scores. The largest correlation observed between PA scores was with Active Coping scores. This relationship was moderate in strength and positive in direction.

Significant correlations were observed between NA scores and both sGBI-Depression and sGBI-Hypomania scores as well as with Active and Avoidant Coping scores. The strongest correlation regarding NA scores was with sGBI-Hypomania scores which was positive in direction and moderate in strength. The second strongest correlation observed for NA scores was with Avoidant Coping scores; this too was positive in direction and moderate in strength.

To investigate the predictive power of coping styles on mood/wellbeing outcome variables (in the context of either sGBI-Depression or sGBI-Hypomania scores) hierarchical regression analyses were run. Tables 17 to 22 present these analyses. Scores for either sGBI-Depression or sGBI-Hypomania were entered at stage 1, and then Active Coping, Support Seeking and Avoidant Coping scores were entered at stage 2. Tables 17 and 18 show hierarchical regression analyses predicting SWL scores, Tables 19 and 20 present hierarchical regression analyses predicting PA scores, and Tables 21 and 22 present hierarchical regression analyses predicting NA scores.
Table 17

Hierarchical Regression Analyses Predicting SWL: Standardised Weights

<table>
<thead>
<tr>
<th>Variables</th>
<th>Stage 1</th>
<th>Stage 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Squared Part-Correlations</td>
<td>β</td>
</tr>
<tr>
<td>sGBI-Depression</td>
<td>.14 -.38***</td>
<td>.06 -.27***</td>
</tr>
<tr>
<td>AcC</td>
<td>.04 .22***</td>
<td>.01 .12**</td>
</tr>
<tr>
<td>SS</td>
<td>.01 &lt;.01</td>
<td>.12**</td>
</tr>
<tr>
<td>AvC</td>
<td>&lt;.01</td>
<td>-.12**</td>
</tr>
<tr>
<td>$R^2$</td>
<td>.14***</td>
<td>.25***</td>
</tr>
<tr>
<td>$F$</td>
<td>101.16***</td>
<td>51.38***</td>
</tr>
<tr>
<td>$AR^2$</td>
<td>.11***</td>
<td></td>
</tr>
<tr>
<td>$ΔF$</td>
<td>49.78***</td>
<td></td>
</tr>
</tbody>
</table>

*Note. $N = 608$. AcC = Active Coping; SS = Support Seeking; AvC = Avoidant Coping.
* $p < .05$. ** $p < .01$. *** $p < .001$

Table 18

Hierarchical Regression Analyses Predicting SWL: Standardised Weights

<table>
<thead>
<tr>
<th>Variables</th>
<th>Stage 1</th>
<th>Stage 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Squared Part-Correlations</td>
<td>β</td>
</tr>
<tr>
<td>sGBI-Hypomania</td>
<td>.28 -.53***</td>
<td>.14 -.43***</td>
</tr>
<tr>
<td>AcC</td>
<td>.02 .18***</td>
<td>.01 .11**</td>
</tr>
<tr>
<td>SS</td>
<td>.01 &lt;.01</td>
<td>-.06</td>
</tr>
<tr>
<td>AvC</td>
<td>&lt;.01</td>
<td></td>
</tr>
<tr>
<td>$R^2$</td>
<td>.28***</td>
<td>.34***</td>
</tr>
<tr>
<td>$F$</td>
<td>233.20**</td>
<td>76.76***</td>
</tr>
<tr>
<td>$AR^2$</td>
<td>.06***</td>
<td></td>
</tr>
<tr>
<td>$ΔF$</td>
<td>156.44***</td>
<td></td>
</tr>
</tbody>
</table>

*Note. $N = 608$. AcC = Active Coping; SS = Support Seeking; AvC = Avoidant Coping.
* $p < .05$. ** $p < .01$. *** $p < .001$

Table 17 shows, that at Stage 1, sGBI-Depression scores explained 14.3% of the variance in SWL scores. The addition of coping styles (Active Coping, Support Seeking and Avoidant Coping scores) explained an additional 11.1% of the variation in SWL scores, and this improvement was significant. The sGBI-Depression scores remained the most important predictor of SWL scores at both stages.
Table 18 shows that, at Stage 1, sGBI-Hypomania scores explained 27.8% of the variation in SWL scores. The addition of the three coping styles (Active Coping, Support Seeking and Avoidant Coping scores) explained an additional 5.9% of the variation in SWL scores; this too was a significant improvement. The sGBI-Hypomania scores remained the most important predictor of SWL scores.

Table 19

Hierarchical Regression Analyses Predicting PA: Standardised Weights

<table>
<thead>
<tr>
<th>Variables</th>
<th>Stage 1</th>
<th>Stage 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Squared Part-Correlations</td>
<td>β</td>
</tr>
<tr>
<td>sGBI-Depression</td>
<td>&lt;.01</td>
<td>-.01</td>
</tr>
<tr>
<td>AcC</td>
<td>.04</td>
<td>.37***</td>
</tr>
<tr>
<td>SS</td>
<td>.01</td>
<td>.01</td>
</tr>
<tr>
<td>AvC</td>
<td>&lt;.01</td>
<td>-.01</td>
</tr>
<tr>
<td>$R^2$</td>
<td>&lt;.01</td>
<td>.14***</td>
</tr>
<tr>
<td>$F$</td>
<td>.06</td>
<td>25.02***</td>
</tr>
<tr>
<td>$\Delta R^2$</td>
<td></td>
<td>.14***</td>
</tr>
<tr>
<td>$\Delta F$</td>
<td></td>
<td>24.96***</td>
</tr>
</tbody>
</table>

*Note. N = 608. AcC = Active Coping; SS = Support Seeking; AvC = Avoidant Coping.

Table 19 shows, that at Stage 1, sGBI-Depression scores did not significantly predict PA scores, nor did they explain any (0.0%) of the variance in PA scores. The addition of coping styles (Active Coping, Support Seeking and Avoidant Coping scores) explained an additional 14.2% of the variation in PA scores, and this was a significant improvement. The most important predictor was Active Coping scores, explaining 10.6% of the variation in PA scores.
Table 20

*Hierarchical Regression Analyses Predicting PA: Standardised Weights*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Stage 1</th>
<th>Stage 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Squared Part-</td>
<td>Squared Part-</td>
</tr>
<tr>
<td></td>
<td>Correlations</td>
<td>Correlations</td>
</tr>
<tr>
<td></td>
<td>β</td>
<td>β</td>
</tr>
<tr>
<td>sGBI-Hypomania</td>
<td>.04</td>
<td>.01</td>
</tr>
<tr>
<td></td>
<td>-.20***</td>
<td>-.13**</td>
</tr>
<tr>
<td>AcC</td>
<td>.10</td>
<td>.37***</td>
</tr>
<tr>
<td>SS</td>
<td>&lt;.01</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>AvC</td>
<td>&lt;.01</td>
<td>-.07</td>
</tr>
</tbody>
</table>

\[ R^2 = .04*** \]
\[ F = 24.63*** \]
\[ ΔR^2 = .11*** \]
\[ ΔF = 2.56*** \]

*Note. N = 608. AcC = Active Coping; SS = Support Seeking; AvC = Avoidant Coping.
* * p < .05. ** p < .01. *** p < .001

Table 20 shows that, at Stage 1, sGBI-Hypomania scores predicted 3.9% of the variation in PA scores. Although this percentage was low, it was significant. The addition of coping styles (Active Coping, Support Seeking and Avoidant Coping scores) explained an additional 11.4% of the variation in PA scores and this improvement was significant. Again, Active Coping scores were the largest predictor of PA scores, explaining 10.3% of the variation in PA scores at Stage 2.

Table 21

*Hierarchical Regression Analyses Predicting NA: Standardised Weights*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Stage 1</th>
<th>Stage 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Squared Part-</td>
<td>Squared Part-</td>
</tr>
<tr>
<td></td>
<td>Correlations</td>
<td>Correlations</td>
</tr>
<tr>
<td></td>
<td>β</td>
<td>β</td>
</tr>
<tr>
<td>sGBI-Depression</td>
<td>.15</td>
<td>.06</td>
</tr>
<tr>
<td></td>
<td>.39***</td>
<td>.28***</td>
</tr>
<tr>
<td>AcC</td>
<td>&lt;.01</td>
<td>-.01</td>
</tr>
<tr>
<td>SS</td>
<td>&lt;.01</td>
<td>.06</td>
</tr>
<tr>
<td>AvC</td>
<td>.06</td>
<td>.29***</td>
</tr>
</tbody>
</table>

\[ R^2 = .15*** \]
\[ F = 110.44 \]
\[ ΔR^2 = .08*** \]
\[ ΔF = 66.18*** \]

*Note. N = 608. AcC = Active Coping; SS = Support Seeking; AvC = Avoidant Coping.
* * p < .05. ** p < .01. *** p < .001
Table 21 shows, that at Stage 1, sGBI-Depression scores explained 15.4 % of the variance in NA scores. The addition of coping styles (Active Coping, Support Seeking and Avoidant Coping scores) explained an additional 7.3 % of the variation in NA scores, and this improvement was significant. At Stage 2, sGBI-Depression scores remained the most important predictor of NA scores, explaining 6.1 % of the variation. However, Avoidant Coping scores also explained 6.0 % of the variation in NA scores.

Table 22

Hierarchical Regression Analyses Predicting NA: Standardised Weights

<table>
<thead>
<tr>
<th>Variables</th>
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<th></th>
<th>Stage 2</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Squared Part-Correlations</td>
<td>β</td>
<td>Squared Part-Correlations</td>
<td>β</td>
</tr>
<tr>
<td>sGBI-Hypomania</td>
<td>.20</td>
<td>.44***</td>
<td>.09</td>
<td>-.34**</td>
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<tr>
<td>AcC</td>
<td>&lt;.01</td>
<td>.03</td>
<td>&lt;.01</td>
<td>.06</td>
</tr>
<tr>
<td>SS</td>
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<td>.06</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AvC</td>
<td>.05</td>
<td>.27***</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| R²              | .20***  |       | .25***  |       |
| F               | 149.12*** |     | 50.67*** |     |
| ΔR²             |         |       | .05***  |       |
| ΔF              |         |       | 98.45*** |       |

Note. N = 608. AcC = Active Coping; SS = Support Seeking; AvC = Avoidant Coping. * p < .05. ** p < .01. ***p < .001

Table 22 shows, that at Stage 1, sGBI-Hypomania scores explained 19.7 % of the variation in NA scores. The addition of coping styles (Active Coping, Support Seeking and Avoidant Coping scores) explained an additional 5.5 % of the variation in NA scores. The sGBI-Hypomania scores remained the most important predictor of NA scores (explaining an additional 8.6 % of the total variance at Stage 2) and again Avoidant Coping scores were the next most important predictors of variance in NA scores (explaining an additional 5.0 % of the total variance at Stage 2).

In summary, results from the hierarchical regression analyses (Tables 17–22) indicated that although sGBI-Depression and sGBI-Hypomania scores mostly remained
important predictors of mood and wellbeing outcome variables (with the exception of sGBI-Depression and PA scores), coping styles were significant predictors of mood/wellbeing variable scores. The patterns of associations were in line with bivariate analyses in Table 16, and consistent with predictions. These findings support Hypothesis 11, that coping predicts mood and wellbeing outcome variables in the context of vulnerability to BD.

7.4.4 Regression-based mediation analyses

Regression-based mediation analyses were also conducted to determine if state sleep (PSQI scores), and coping (Active, Support Seeking, and Avoidant coping scores) acted as full or partial mediators (M) between vulnerability to BD (measured by sGBI-Depression and sGBI-Hypomania scores) and mood/wellbeing variables (PA, NA, and SWL scores). Regression-based mediation analyses can only include one IV, one mediator and one DV. These analyses were carried out to test the first phase of Hypotheses 11 and 13. The regression-based mediation analyses were also carried out to explore if coping and state sleep variables acted as mediators between either sGBI-Depression or sGBI-Hypomania scores, and outcome variables, without the influence of trait-like sleep variables. Figure 3, which included trait-like sleep variables (all modified ASWS subscales and Morningness), all mediators (PSQI, Active Coping, Support Seeking and Avoidant Coping scores), and all mood and wellbeing outcome variables (PA, NA and SWL scores) was tested using SEM, described later. Regression-based analyses that examined if coping variables or state sleep acted as mediators of each other (that is, if coping variables mediated interactions between vulnerability to BD and state sleep, or if state sleep mediated interactions between vulnerability to BD and coping variables) were also carried out to inform the placing of both coping and state sleep variables in later SEM analyses, that ultimately tested Hypotheses 11 and 13.
Table 23 presents summary results of the regression-based mediation analyses. Table 23 shows that none of the state sleep (PSQI scores) or coping (Active, Support Seeking, and Avoidant coping scores) variables acted as full mediators between sGBI-Depression or sGBI-Hypomania scores and any of PA, NA or SWL scores. The analyses presented in Table 23 show preliminary support for Hypothesis 11 (that PSQI score would act as a mediator in models relating trait-like sleep, sGBI-Depression or sGBI-Hypomania scores to coping variables and mood/wellbeing variables) as trait-like sleep variables were not included in the regression-based mediation analyses. These analyses confirmed the right hand side of the conceptual models that included interactions between vulnerability to BD, state sleep and mood/wellbeing outcome variables. As can be seen in Table 23, PSQI scores acted as a partial mediator between sGBI-Depression scores and NA scores; between sGBI-Depression and SWL scores; between sGBI-Hypomania and PA scores; between sGBI-Hypomania and NA scores; and between sGBI-Hypomania and SWL scores.

The analyses presented in Table 23 provide preliminary support for Hypothesis 13, as trait-like sleep variables were not included in the regression-based mediation analyses and only one IV, mediator, and DV could be tested at a time. These analyses illuminate some, but not all, aspects of the conceptual models (see Figure 4) that included interactions between vulnerability to BD (either sGBI-Depression or sGBI-Hypomania scores), coping variables (Active Coping, Support Seeking and Avoidant Coping scores), and mood/wellbeing outcome (PA, NA, SWL scores) variables. As can be seen in Table 23, the following partial mediating relationships were identified when sGBI-Depression was the IV: Active Coping (M) and NA scores (DV); Active Coping (M) and SWL scores (DV); Support Seeking (M) and SWL (DV) scores; Avoidant
Table 23

*Results of Regression Analyses Investigating Full and Partial Mediation*

<table>
<thead>
<tr>
<th>IV</th>
<th>Potential Mediator (M)</th>
<th>DV</th>
<th>IV and M Correlations</th>
<th>Beta for IV when predicting DV</th>
<th>Beta for IV when M and IV predicting DV</th>
<th>Sobel’s z statistic</th>
<th>Type of Mediation</th>
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<tr>
<td>sGBI-Depression</td>
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<td>.39**</td>
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<td>PSQI</td>
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<td>-.09*</td>
<td>-3.06**</td>
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<td>-.09*</td>
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<td>AvC</td>
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<td>-.16**</td>
<td>-.09*</td>
<td>-3.80**</td>
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<td>-.01</td>
<td>-3.68**</td>
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<tr>
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<td>-.01</td>
<td>0.05</td>
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<td>-.01</td>
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<tr>
<td>AcC</td>
<td>SWL</td>
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<td>-.01</td>
<td>-.01</td>
<td>-3.68**</td>
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<tr>
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<td>.01</td>
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<td>-.01</td>
<td>-2.44*</td>
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<tr>
<td>SS</td>
<td>SWL</td>
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<td>-.01</td>
<td>-.01</td>
<td>-2.44*</td>
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<td>-3.70**</td>
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<tr>
<td>AvC</td>
<td>SWL</td>
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<td>-.01</td>
<td>.06</td>
<td>-3.70**</td>
<td>None</td>
<td></td>
</tr>
</tbody>
</table>

| sGBI-Hypomania      | AcC                    | PSQI   | -.28**                 | .47**                          | .45**                                  | 2.06*               | Part              |
| SS                  | PSQI                   | -.18** | .47**                  | .45**                          | 2.86**                                 | Part                |
| AvC                 | PSQI                   | .47**  | .47**                  | .45**                          | 2.86**                                 | Part                |
| PSQI                | AcC                    | .47**  | -.28**                 | -.23**                         | -2.14*                                 | Part                |
| PSQI                | SS                     | .47**  | -.28**                 | -.23**                         | -2.14*                                 | Part                |
| PSQI                | AvC                    | .47**  | -.28**                 | -.23**                         | -2.14*                                 | Part                |
| PSQI                | PA                     | .47**  | -.28**                 | -.23**                         | -2.14*                                 | Part                |
| PSQI                | NA                     | .47**  | -.28**                 | -.23**                         | -2.14*                                 | Part                |
| PSQI                | SWL                    | .47**  | -.28**                 | -.23**                         | -2.14*                                 | Part                |
| AcC                 | PA                     | -.28** | -.28**                 | -.28**                         | -2.14*                                 | Part                |
| AcC                 | NA                     | -.28** | -.28**                 | -.28**                         | -2.14*                                 | Part                |
| AcC                 | SWL                    | -.28** | -.28**                 | -.28**                         | -2.14*                                 | Part                |
| SS                  | PA                     | -.18** | -.18**                 | -.18**                         | -1.99*                                 | Part                |
| SS                  | NA                     | -.18** | -.18**                 | -.18**                         | -1.99*                                 | Part                |
| SS                  | SWL                    | -.18** | -.18**                 | -.18**                         | -1.99*                                 | Part                |
| AvC                 | PA                     | -.18** | -.18**                 | -.18**                         | -1.99*                                 | Part                |
| AvC                 | NA                     | -.18** | -.18**                 | -.18**                         | -1.99*                                 | Part                |
| AvC                 | SWL                    | -.18** | -.18**                 | -.18**                         | -1.99*                                 | Part                |

*Note.* Full = Full Mediation; Part = Partial Mediation; None = No Mediation; AcC = Active Coping; SS = Support Seeking; AvC = Avoidant Coping; PSQI = Pittsburgh Sleep Quality Index scores; PA = Positive Affect; NA = Negative Affect; SWL = Satisfaction with Life  
* * * p < .05. ** * * p < .01. *** * * * p < .001
Coping (M) and NA scores; and Avoidant Coping (M) and SWL (DV). For regression-based mediation analyses that included sGBI-Hypomania scores as the IV the following partial mediating relationships were identified: Active Coping (M) and PA (DV) scores; Active Coping (M) and SWL (DV) scores; Support Seeking (M) and PA (DV) scores; Support Seeking (M) and SWL (DV) scores; Avoidant Coping (M) and NA (DV) scores; and with Avoidant Coping (M) and SWL (DV). The largest z-score was produced by Avoidant Coping scores, when acting as a mediator between sGBI-Depression and NA scores; and between sGBI-Hypomania and NA scores.

Regression-based analyses indicated that both state sleep scores and coping variables acted as partial mediators when the other was a DV. The only exceptions were when sGBI-Hypomania was the IV, PSQI scores (M) did not mediate NA (DV) scores, and NA (M) scores did not mediate PSQI (DV) scores.

In summary, both state sleep (PSQI scores) and coping styles (Active Coping, Support Seeking, and Avoidant Coping scores) acted as partial mediators relating vulnerability to BD (sGBI-Depression or sGBI-Hypomania scores) to mood/wellbeing variables. Furthermore, the regression-based mediation analyses indicated that PSQI and coping scores (Active Coping, Support Seeking, and Avoidant Coping) acted as partial mediators for each other when sGBI-Depression or sGBI Hypomania scores are the IV’s. As previously mentioned, SEM analyses were conducted to test the mediating role of state sleep scores and coping variables at the same time, as well as the inclusion of trait-sleep variables and all mood and wellbeing outcome variables.

7.4.5 Modelling analyses.

Finally, SEM with bootstrapping analyses were conducted to investigate the more complex mechanistic hypotheses arising from Figure 4. This was done using the
conceptual models, where two models were tested, one containing the sGBI-Depression scores and the other model containing the sGBI-Hypomania scores.

To assess multivariate normality of the joint distributions of the data, Mardia’s coefficient, a measure of multivariate kurtosis, was calculated for both models. Values of 3 or greater are indicative of multivariate non-normality (Cunningham, 2008). For the model containing sGBI-Depression scores Mardia’s coefficient was 11.51. Mahalanobis’ Distances were examined, and no specific cases were found to be over the recommended range; thus, it was deduced that this value was due to joint distributions being kurtotic in nature, rather than outliers. Similarly, Mardia’s coefficient for the model containing sGBI-Hypomania scores was 10.85. Due to the presence of non-normality, Bollen-Stine’s $p$, Bootstrapping (2000 iterations) and 95 % Confidence Intervals were calculated for all weights.

The initial theorised models generated poor fit indices ($\chi^2$; see Tables 24 and 27) and so were revised as proposed by the Modification Indices, with deletion of non-significant pathways. Table 24 shows the fit indices and modifications made at each step for the model that contained sGBI-Depression scores. Table 27 does the same for the model containing sGBI-Hypomania scores. The final models are shown in Figures 6 and 7. Standardised Direct and Indirect Effect Table Outputs from AMOS for the sGBI-Depression scores can be seen in Appendix S, and those for the model containing sGBI-Hypomania scores can be viewed in Appendix T.

As noted above (Section 6.6.4), if there are concerns with non-normality, it is still worthwhile examining other fit indices (Kline, 2011). As can be seen in Tables 24 and 27, although both the chi-square ($\chi^2$) and Bollen Stine’s $p$ suggest that the final models do not fit well, other commonly accepted model fit indices indicate good fit. Furthermore, each pathway was shown to be significant when 95 % bias-corrected
confidence intervals were calculated (see Tables 25 and 28 for significance of pathway and value of each pathway). The models also appeared to explain a reasonable percentage of the variation for most of the variables (see Tables 26 and 29).
Table 24

Model fit indices for the Structural Equation Model containing sGBI-Depression scores exploring the interactions between trait-like Sleep, Coping, State Sleep, Mood and SWL variables (continued on next page)

<table>
<thead>
<tr>
<th>Model</th>
<th>Model Description</th>
<th>Fit Indices</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>$\chi^2$</td>
</tr>
<tr>
<td>0</td>
<td>Model based on theoretical assumptions (full mediation)</td>
<td>2612.074</td>
</tr>
<tr>
<td>1</td>
<td>Co-varied modified ASWS</td>
<td>1833.960</td>
</tr>
<tr>
<td>2</td>
<td>Co-varied errors of bCOPE</td>
<td>1674.226</td>
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<td>3</td>
<td>Co-varied errors of mood/wellbeing variables (PA,NA and SWL)</td>
<td>1531.211</td>
</tr>
<tr>
<td>4</td>
<td>According to MI added pathway from WU to Morningness</td>
<td>1042.316</td>
</tr>
<tr>
<td>5</td>
<td>Deleted pathways that were not significant (WU to Dep; AcC to PSQI)</td>
<td>1045.415</td>
</tr>
<tr>
<td>6</td>
<td>Deleted non-significant pathway from Morningness to Depression</td>
<td>1048.038</td>
</tr>
<tr>
<td>7</td>
<td>Deleted non-significant pathway FA to Dep</td>
<td>1051.405</td>
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<tr>
<td>8</td>
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<td>9</td>
<td>As per MI added pathway from SA to PSQI</td>
<td>614.553</td>
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<tr>
<td>10</td>
<td>As per MI added pathway from GTB to Morningness</td>
<td>520.119</td>
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<tr>
<td>11</td>
<td>Added pathway from WU to PA</td>
<td>459.910</td>
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<tr>
<td>12</td>
<td>Deleted pathway between PSQI and PA (CR over 0.05)</td>
<td>462.091</td>
</tr>
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Table 24 continued...

<table>
<thead>
<tr>
<th>Model</th>
<th>Model Description</th>
<th>( \chi^2 )</th>
<th>d.f.</th>
<th>Sig.</th>
<th>Bollen-Stine's RMSEA</th>
<th>SRMR</th>
<th>GFI</th>
<th>AGFI</th>
<th>TLI</th>
<th>CFI</th>
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<td>.098</td>
<td>.093</td>
<td>.920</td>
<td>.858</td>
<td>.826</td>
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<td>14</td>
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<td>.927</td>
<td>.868</td>
<td>.847</td>
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<td>15</td>
<td>Added pathway from AcC to PA</td>
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<td>&lt; .001</td>
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<td>&lt; .001</td>
<td>.081</td>
<td>.069</td>
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<td>.881</td>
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<td>&lt; .001</td>
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<td>119.965</td>
<td>48</td>
<td>&lt; .001</td>
<td>&lt; .001</td>
<td>.050</td>
<td>.039</td>
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<td>.942</td>
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<td>25</td>
<td>Added pathway from Dep to PSQI</td>
<td>111.028</td>
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<td>&lt; .001</td>
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<td>.038</td>
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<td>26</td>
<td>Added pathway from Dep to PA</td>
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<td>&lt; .001</td>
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<td>Added pathway from GTB to SS</td>
<td>96.935</td>
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<td>.966</td>
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<tr>
<td>28</td>
<td>Added pathway from WU to Avoidant Final model</td>
<td>91.464</td>
<td>44</td>
<td>&lt; .001</td>
<td>&lt; .001</td>
<td>.042</td>
<td>.026</td>
<td>.979</td>
<td>.950</td>
<td>.968</td>
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*Note.* RMSEA = Root Mean Square Error of Approximation; SRMR = Standardised Root Mean Residual; GFI = Goodness of Fit; AGFI = Adjusted Goodness of Fit; TLI = Tucker Lewis Index; CFI = Comparative Fit Index; AcC = Active Coping; AvC = Avoidant Coping; Dep = sGBI-Depression; FA = Falling Asleep; GTB = Going to Bed; MI = Modification Indices; NA = Negative Affect; RS = Reinitiating Sleep; PA = Positive Affect; SA = Staying Asleep; SS = Support Seeking; SWL = Satisfaction with Life; WU = Returning to Wakefulness.
Table 25

Standardised Regression Weights of Model containing sGBI-Depression Scores and Significance of Pathways

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Estimate</th>
<th>Lower</th>
<th>Upper</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>sGBI-Depression ← GTB</td>
<td>-.299</td>
<td>-.363</td>
<td>-.226</td>
<td>.001</td>
</tr>
<tr>
<td>sGBI-Depression ← SA</td>
<td>-.135</td>
<td>-.225</td>
<td>-.045</td>
<td>.004</td>
</tr>
<tr>
<td>sGBI-Depression ← RS</td>
<td>-.209</td>
<td>-.293</td>
<td>-.121</td>
<td>.001</td>
</tr>
<tr>
<td>Support Seeking ← sGBI-Depression</td>
<td>-.124</td>
<td>-.207</td>
<td>-.042</td>
<td>.003</td>
</tr>
<tr>
<td>Avoidant Coping ← sGBI-Depression</td>
<td>.377</td>
<td>.300</td>
<td>.452</td>
<td>.001</td>
</tr>
<tr>
<td>Avoidant Coping ← RS</td>
<td>-.088</td>
<td>-.153</td>
<td>-.017</td>
<td>.016</td>
</tr>
<tr>
<td>Support Seeking ← GTB</td>
<td>.112</td>
<td>.029</td>
<td>.200</td>
<td>.004</td>
</tr>
<tr>
<td>Avoidant Coping ← WU</td>
<td>-.088</td>
<td>-.159</td>
<td>-.008</td>
<td>.027</td>
</tr>
<tr>
<td>Active Coping ← sGBI-Depression</td>
<td>-.110</td>
<td>-.198</td>
<td>-.028</td>
<td>.006</td>
</tr>
<tr>
<td>PSQI ← Avoidant Coping</td>
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<td>.010</td>
<td>.120</td>
<td>.018</td>
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<tr>
<td>PSQI ← Support Seeking</td>
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<td>-.121</td>
<td>-.022</td>
<td>.003</td>
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<td>PSQI ← SA</td>
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<td>-.346</td>
<td>-.215</td>
<td>.001</td>
</tr>
<tr>
<td>PSQI ← FA</td>
<td>-.351</td>
<td>-.416</td>
<td>-.287</td>
<td>.001</td>
</tr>
<tr>
<td>Active Coping ← WU</td>
<td>.229</td>
<td>.152</td>
<td>.302</td>
<td>.001</td>
</tr>
<tr>
<td>PSQI ← RS</td>
<td>-.163</td>
<td>-.222</td>
<td>-.099</td>
<td>.001</td>
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<tr>
<td>PSQI ← WU</td>
<td>-.126</td>
<td>-.186</td>
<td>-.065</td>
<td>.001</td>
</tr>
<tr>
<td>PSQI ← sGBI-Depression</td>
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<td>.024</td>
<td>.162</td>
<td>.006</td>
</tr>
<tr>
<td>SWL ← PSQI</td>
<td>-.167</td>
<td>-.246</td>
<td>-.091</td>
<td>.001</td>
</tr>
<tr>
<td>Morningness ← s1</td>
<td>.608</td>
<td>.570</td>
<td>.645</td>
<td>.001</td>
</tr>
<tr>
<td>Morningness ← WU</td>
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<td>.588</td>
<td>.680</td>
<td>.001</td>
</tr>
<tr>
<td>Morningness ← GTB</td>
<td>.292</td>
<td>.237</td>
<td>.345</td>
<td>.001</td>
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<tr>
<td>NA ← Avoidant Coping</td>
<td>.278</td>
<td>.189</td>
<td>.362</td>
<td>.001</td>
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<tr>
<td>NA ← PSQI</td>
<td>.113</td>
<td>.041</td>
<td>.189</td>
<td>.001</td>
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<td>PA ← WU</td>
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<td>.354</td>
<td>.002</td>
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<tr>
<td>SWL ← Avoidant Coping</td>
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<td>-.190</td>
<td>-.026</td>
<td>.015</td>
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<tr>
<td>PA ← Active Coping</td>
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<td>.387</td>
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<tr>
<td>SWL ← Active Coping</td>
<td>.208</td>
<td>.127</td>
<td>.279</td>
<td>.001</td>
</tr>
<tr>
<td>Morningness ← RS</td>
<td>-.116</td>
<td>-.165</td>
<td>-.071</td>
<td>.001</td>
</tr>
<tr>
<td>SWL ← sGBI-Depression</td>
<td>-.212</td>
<td>-.305</td>
<td>-.129</td>
<td>.001</td>
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<tr>
<td>NA ← sGBI-Depression</td>
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<td>.151</td>
<td>.303</td>
<td>.001</td>
</tr>
<tr>
<td>SWL ← Support Seeking</td>
<td>.112</td>
<td>.041</td>
<td>.181</td>
<td>.001</td>
</tr>
<tr>
<td>PA ← sGBI-Depression</td>
<td>.099</td>
<td>.026</td>
<td>.177</td>
<td>.008</td>
</tr>
</tbody>
</table>

Note. CI = confidence interval; GTB = Going to Bed; FA = Falling Asleep; SA = Staying Asleep; RS = Reinitiating Sleep; WU = Returning to Wakefulness; NA = Negative Affect; PA = Positive Affect; SWL = Satisfaction with Life; PSQI = Pittsburgh Sleep Quality Index scores; s1 = error for SWL.
Figure 6. The final BD, Sleep and Coping model containing sGBI-Depression scores. GTB = Going to bed; FA = Falling Asleep; SA = Staying Asleep; RS = Reinitiating Sleep; WU = Waking up; M = Morningness; BDDep = sGBI-Depression scores; StSl = State Sleep as measured by PSQI scores; AcC = Active Coping; SS = Support Seeking; AvC = Avoidant Coping; PA = Positive Affect; NA = Negative Affect; and SWL = Satisfaction with life.

Table 26

Squared Multiple Correlations Showing How Much of the Variance of Each Variable was Explained by the Model Containing sGBI-Depression Scores

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Estimate</th>
<th>Lower</th>
<th>Upper</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>sGBI-Depression</td>
<td>.210</td>
<td>.152</td>
<td>.266</td>
<td>.002</td>
</tr>
<tr>
<td>Avoidant Coping</td>
<td>.195</td>
<td>.139</td>
<td>.259</td>
<td>.001</td>
</tr>
<tr>
<td>Support seeking</td>
<td>.038</td>
<td>.012</td>
<td>.071</td>
<td>.002</td>
</tr>
<tr>
<td>PSQI</td>
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<td>.543</td>
<td>.641</td>
<td>.005</td>
</tr>
<tr>
<td>Active coping</td>
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<td>.039</td>
<td>.120</td>
<td>.002</td>
</tr>
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<td>Morningness</td>
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<td>.584</td>
<td>.675</td>
<td>.001</td>
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<td>SWL</td>
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<td>.337</td>
<td>.003</td>
</tr>
<tr>
<td>PA</td>
<td>.217</td>
<td>.156</td>
<td>.272</td>
<td>.002</td>
</tr>
<tr>
<td>NA</td>
<td>.234</td>
<td>.166</td>
<td>.302</td>
<td>.002</td>
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</table>

Note: CI = confidence interval. PSQI = state sleep scores; SWL = Satisfaction with Life scores; PA = Positive Affect; NA = Negative Affect
Table 27

Model fit indices for the Structural Equation Model containing sGBI-Hypomania scores exploring the interactions between trait-like Sleep, State Sleep, Mood and SWL variables (continued on next page)

<table>
<thead>
<tr>
<th>Model</th>
<th>Model Description</th>
<th>( \chi^2 )</th>
<th>d.f.</th>
<th>Sig.</th>
<th>Bollen-Stine’s ( p )</th>
<th>RMSEA</th>
<th>SRMR</th>
<th>GFI</th>
<th>AGFI</th>
<th>TLI</th>
<th>CFI</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Model based on theoretical assumptions (full mediation and modified ASWS covaried)</td>
<td>1852.922</td>
<td>66</td>
<td>&lt; .001</td>
<td>&lt; .001</td>
<td>.211</td>
<td>.208</td>
<td>.731</td>
<td>.572</td>
<td>.221</td>
<td>.435</td>
</tr>
<tr>
<td>1</td>
<td>Deleted non-significant pathway from AcC to PSQI</td>
<td>1854.624</td>
<td>67</td>
<td>&lt; .001</td>
<td>&lt; .001</td>
<td>.210</td>
<td>.209</td>
<td>.731</td>
<td>.579</td>
<td>.223</td>
<td>.435</td>
</tr>
<tr>
<td>2</td>
<td>Co-vary errors of coping variables</td>
<td>1728.822</td>
<td>64</td>
<td>&lt; .001</td>
<td>&lt; .001</td>
<td>.207</td>
<td>.203</td>
<td>.749</td>
<td>.588</td>
<td>.252</td>
<td>.474</td>
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<tr>
<td>3</td>
<td>Co-vary errors of SWL, PA and NA</td>
<td>1585.807</td>
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<td>&lt; .001</td>
<td>&lt; .001</td>
<td>.203</td>
<td>.199</td>
<td>.775</td>
<td>.613</td>
<td>.281</td>
<td>.518</td>
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<tr>
<td>4</td>
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<td>1096.912</td>
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<td>&lt; .001</td>
<td>.169</td>
<td>.170</td>
<td>.831</td>
<td>.705</td>
<td>.503</td>
<td>.672</td>
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<tr>
<td>5</td>
<td>As per MI pathway from FA to PSQI added</td>
<td>790.049</td>
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<td>&lt; .001</td>
<td>.143</td>
<td>.117</td>
<td>.860</td>
<td>.750</td>
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<td>&lt; .001</td>
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<td>.110</td>
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<td>.701</td>
<td>.810</td>
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<td>&lt; .001</td>
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<td>.107</td>
<td>.893</td>
<td>.802</td>
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<td>506.67</td>
<td>56</td>
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<td>&lt; .001</td>
<td>.115</td>
<td>.098</td>
<td>.900</td>
<td>.813</td>
<td>.769</td>
<td>.858</td>
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<td>9</td>
<td>As per MI pathway from sGBI-Hyp to SWL added</td>
<td>419.363</td>
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<td>&lt; .001</td>
<td>&lt; .001</td>
<td>.104</td>
<td>.082</td>
<td>.911</td>
<td>.831</td>
<td>.809</td>
<td>.885</td>
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<td>As per MI, added pathway from WU to PA</td>
<td>353.664</td>
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<td>&lt; .001</td>
<td>.096</td>
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<td>.072</td>
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Table 27 continued...

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<th>Model</th>
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<th>χ²</th>
<th>d.f.</th>
<th>Sig.</th>
<th>Bollen-Stine’s p</th>
<th>RMSEA</th>
<th>SRMR</th>
<th>GFI</th>
<th>AGFI</th>
<th>TLI</th>
<th>CFI</th>
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<td>.870</td>
<td>.862</td>
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<td>&lt;.001</td>
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<td>.055</td>
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<td>.902</td>
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<td>&lt;.001</td>
<td>.073</td>
<td>.050</td>
<td>.951</td>
<td>.902</td>
<td>.908</td>
<td>.946</td>
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<td>.939</td>
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<td>&lt;.001</td>
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<td>.960</td>
<td>.980</td>
</tr>
<tr>
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<td>Deleted non-significant pathway from AcC to PSQI</td>
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<td>&lt;.001</td>
<td>.048</td>
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<td>.974</td>
<td>.942</td>
<td>.960</td>
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<td>As per MI added pathway from GTB to SS</td>
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<td>&lt;.001</td>
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<td>.028</td>
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<td>As per MI added pathway from GTB to PA</td>
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<td>.001</td>
<td>.045</td>
<td>.028</td>
<td>.976</td>
<td>.946</td>
<td>.965</td>
<td>.982</td>
</tr>
<tr>
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<td>As per MI added pathway from RS to AcC-Final model</td>
<td>96.085</td>
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<td>.043</td>
<td>.027</td>
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<td>.967</td>
<td>.984</td>
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Note. RMSEA = Root Mean Square Error of Approximation; SRMR = Standardised Root Mean Residual; GFI = Goodness of Fit; AGFI = Adjusted Goodness of Fit; TLI = Tucker Lewis Index; CFI = Comparative Fit Index; AcC = Active Coping; AvC = Avoidant Coping; FA = Falling Asleep; GTB = Going to Bed; MI = Modification Indices; NA = Negative Affect; Reinitiating Sleep; PA = Positive Affect; sGBI-Hyp = sGBI Hypomania scores; SA = Staying Asleep; SS = Support Seeking; SWL = Satisfaction with Life; WU = Returning to Wakefulness.
Table 28

**Standardised Regression Weights of Model containing sGBI-Hypomania scores and Significance of Pathways**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Estimate</th>
<th>Lower</th>
<th>Upper</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morningness ← WU</td>
<td>.635</td>
<td>.588</td>
<td>.680</td>
<td>.001</td>
</tr>
<tr>
<td>Morningness ← RS</td>
<td>-.116</td>
<td>-.165</td>
<td>-.071</td>
<td>.001</td>
</tr>
<tr>
<td>Morningness ← GTB</td>
<td>.292</td>
<td>.237</td>
<td>.345</td>
<td>.001</td>
</tr>
<tr>
<td>sGBI-Hypomania ← GTB</td>
<td>-.204</td>
<td>-.296</td>
<td>-.105</td>
<td>.001</td>
</tr>
<tr>
<td>sGBI-Hypomania ← FA</td>
<td>-.114</td>
<td>-.209</td>
<td>-.010</td>
<td>.027</td>
</tr>
<tr>
<td>sGBI-Hypomania ← SA</td>
<td>-.124</td>
<td>-.215</td>
<td>-.034</td>
<td>.009</td>
</tr>
<tr>
<td>sGBI-Hypomania ← RS</td>
<td>-.170</td>
<td>-.263</td>
<td>-.080</td>
<td>.001</td>
</tr>
<tr>
<td>sGBI-Hypomania ← WU</td>
<td>-.211</td>
<td>-.322</td>
<td>-.079</td>
<td>.002</td>
</tr>
<tr>
<td>sGBI-Hypomania ← Morningness</td>
<td>.124</td>
<td>.010</td>
<td>.248</td>
<td>.031</td>
</tr>
<tr>
<td>PSQI ← sGBI-Hypomania</td>
<td>.153</td>
<td>.087</td>
<td>.220</td>
<td>.001</td>
</tr>
<tr>
<td>PSQI ← FA</td>
<td>-.352</td>
<td>-.415</td>
<td>-.290</td>
<td>.001</td>
</tr>
<tr>
<td>PSQI ← SA</td>
<td>-.272</td>
<td>-.337</td>
<td>-.206</td>
<td>.001</td>
</tr>
<tr>
<td>PSQI ← RS</td>
<td>-.167</td>
<td>-.227</td>
<td>-.105</td>
<td>.001</td>
</tr>
<tr>
<td>PSQI ← WU</td>
<td>-.115</td>
<td>-.178</td>
<td>-.052</td>
<td>.001</td>
</tr>
<tr>
<td>Support Seeking ← PSQI</td>
<td>-.155</td>
<td>-.242</td>
<td>-.075</td>
<td>.001</td>
</tr>
<tr>
<td>Active Coping ← sGBI-Hypomania</td>
<td>-.220</td>
<td>-.302</td>
<td>-.138</td>
<td>.001</td>
</tr>
<tr>
<td>Support Seeking ← sGBI-Hypomania</td>
<td>-.108</td>
<td>-.189</td>
<td>-.020</td>
<td>.014</td>
</tr>
<tr>
<td>Avoidant Coping ← sGBI-Hypomania</td>
<td>.441</td>
<td>.362</td>
<td>.514</td>
<td>.001</td>
</tr>
<tr>
<td>Active Coping ← WU</td>
<td>.178</td>
<td>.102</td>
<td>.253</td>
<td>.001</td>
</tr>
<tr>
<td>Avoidant Coping ← RS</td>
<td>-.082</td>
<td>-.148</td>
<td>-.009</td>
<td>.017</td>
</tr>
<tr>
<td>SWL ← PSQI</td>
<td>-.092</td>
<td>-.170</td>
<td>-.014</td>
<td>.021</td>
</tr>
<tr>
<td>PA ← Active Coping</td>
<td>.299</td>
<td>.227</td>
<td>.370</td>
<td>.001</td>
</tr>
<tr>
<td>SWL ← Active Coping</td>
<td>.195</td>
<td>.126</td>
<td>.263</td>
<td>.001</td>
</tr>
<tr>
<td>SWL ← Support Seeking</td>
<td>.108</td>
<td>.042</td>
<td>.175</td>
<td>.001</td>
</tr>
<tr>
<td>NA ← Avoidant Coping</td>
<td>.231</td>
<td>.145</td>
<td>.324</td>
<td>.001</td>
</tr>
<tr>
<td>PA ← WU</td>
<td>.327</td>
<td>.254</td>
<td>.400</td>
<td>.001</td>
</tr>
<tr>
<td>SWL ← sGBI-Hypomania</td>
<td>-.403</td>
<td>-.480</td>
<td>-.327</td>
<td>.001</td>
</tr>
<tr>
<td>NA ← sGBI-Hypomania</td>
<td>.344</td>
<td>.265</td>
<td>.415</td>
<td>.002</td>
</tr>
<tr>
<td>PA ← GTB</td>
<td>-.099</td>
<td>-.175</td>
<td>-.024</td>
<td>.012</td>
</tr>
</tbody>
</table>

**Note:** CI = confidence interval; GTB = Going to Bed; FA = Falling Asleep; SA = Staying Asleep; RS = Reinitiating Sleep; WU = Returning to Wakefulness; NA = Negative Affect; PA = Positive Affect; SWL = Satisfaction with Life; PSQI = Pittsburgh Sleep Quality Index scores.
Figure 7. The final BD, Sleep and Coping model containing sGBI-Hypomania scores. GTB = Going to bed; FA = Falling Asleep; SA = Staying Asleep; RS = Reinitiating Sleep; WU = Waking up; M = Morningness; BDHyp = sGBI-Hypomania scores; StSl = State Sleep as measured by Pittsburgh Sleep Quality Index scores; AcC = Active Coping; SS = Support Seeking; AvC = Avoidant Coping; PA = Positive Affect; NA = Negative Affect; and SWL = Satisfaction with life.

Table 29

Squared Multiple Correlations Showing the Variance of Each Variable Explained by the Model Containing sGBI-Hypomania Scores

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Estimate</th>
<th>Lower</th>
<th>Upper</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morningness</td>
<td>.630</td>
<td>.584</td>
<td>.675</td>
<td>.001</td>
</tr>
<tr>
<td>sGBI-Hypomania</td>
<td>.252</td>
<td>.188</td>
<td>.305</td>
<td>.005</td>
</tr>
<tr>
<td>PSQI</td>
<td>.599</td>
<td>.543</td>
<td>.641</td>
<td>.004</td>
</tr>
<tr>
<td>Avoidant Coping</td>
<td>.227</td>
<td>.167</td>
<td>.297</td>
<td>.001</td>
</tr>
<tr>
<td>Support Seeking</td>
<td>.052</td>
<td>.024</td>
<td>.090</td>
<td>.001</td>
</tr>
<tr>
<td>Active Coping</td>
<td>.105</td>
<td>.061</td>
<td>.157</td>
<td>.001</td>
</tr>
<tr>
<td>SWL</td>
<td>.339</td>
<td>.264</td>
<td>.408</td>
<td>.002</td>
</tr>
<tr>
<td>NA</td>
<td>.246</td>
<td>.177</td>
<td>.314</td>
<td>.002</td>
</tr>
<tr>
<td>PA</td>
<td>.216</td>
<td>.158</td>
<td>.271</td>
<td>.002</td>
</tr>
</tbody>
</table>

Note. CI = confidence interval. PSQI = Pittsburgh Sleep Quality Index scores; SWLS = Satisfaction with Life scores; NA = Negative Affect; PA = Positive Affect
Table 29 indicates the variances explained by the model. For example, the three predictors of Morningness (GTB, RS and WU scores) explained 63% of its variance, while the model explained 59.9% of the variation in PSQI scores.

Comparison of the two models underscores the utility of using a two-dimensional approach to BD vulnerability (see Tables 25 and 28). For the model that contained sGBI-Depression scores the following pathways were unique to that model: WU to Avoidant Coping; Avoidant Coping to PSQI scores; Avoidant Coping to SWL scores; and PSQI to NA scores. The unique pathways present in the model that included sGBI-Hypomania scores were: GTB to PA scores; FA to sGBI-Hypomania scores; WU to sGBI-Hypomania scores; Morningness to sGBI-Hypomania scores; and sGBI-Hypomania to NA scores.

To compare the difference in size between direct and indirect effects of the pathways in the model, the indirect effect of PSQI scores and coping variables (Active Coping, Support Seeking, and Avoidant Coping) was calculated for each direct pathway that was present between trait-like sleep or vulnerability to BD and mood/wellbeing variables (PA, NA, and SWL scores). Two tables present these comparisons; the first table presents effect sizes produced from the model that contained sGBI-Depression scores (Table 30), and the second for the model that contained sGBI-Hypomania scores (see Table 31). These tables (Table 30 and 31) indicate that the indirect effect of pathways containing mediators were not as large as the direct effects.

In summary, as illustrated in Figures 6 and 7, the actual models produced differed from the conceptual models in that coping variables (Active Coping, Support Seeking, and Avoidant Coping scores) did not influence state sleep (measured by PSQI score) within the models. Avoidant Coping scores informed PSQI scores when sGBI-Depression scores were included in the model, and Active Coping scores did not inform
PSQI scores in either model. It was hypothesised that both PSQI scores (Hypothesis 11) and coping styles (Active Coping, Support Seeking, and Avoidant Coping scores) (Hypothesis 13) would act as mediators in models relating trait-like sleep variables (GTB, FA, SA, RS, WU, and Morningness scores), vulnerability to BD (sGBI-Depression or sGBI-Hypomania scores), and mood/wellbeing variables (PA, NA, and SWL scores). Hypotheses 11 and 13 were partially supported, as coping styles (Active Coping, Support Seeking, and Avoidant Coping scores) and state sleep (PSQI scores) acted as partial mediators (as there were both direct and indirect pathways between mediators and IV’s and DV’s). For a full summary of the aims, hypotheses and their support, refer to Table 32.

Table 30

Comparison of Direct and Indirect Pathways in the Model containing sGBI-Depression Scores

<table>
<thead>
<tr>
<th>A → C</th>
<th>Direct Effect size</th>
<th>p</th>
<th>Mediator (M) A→M</th>
<th>M →C Effect size</th>
<th>p (total)</th>
</tr>
</thead>
<tbody>
<tr>
<td>WU→ PA</td>
<td>.289</td>
<td>.002</td>
<td>PSQI -.126</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AcC .229</td>
<td>.314</td>
<td>.072</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>SS -</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AvC -.088</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td>sGBI-D→ SWL</td>
<td>-.212</td>
<td>.001</td>
<td>PSQI .091</td>
<td>-.167</td>
<td>-.015</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AcC -.110</td>
<td>.208</td>
<td>.023</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>SS -.124</td>
<td>.112</td>
<td>-.014</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AvC .377</td>
<td>-.109</td>
<td>-.041</td>
</tr>
<tr>
<td>sGBI-D→ PA</td>
<td>.099</td>
<td>.008</td>
<td>PSQI .091</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AcC -.110</td>
<td>.314</td>
<td>-.035</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>SS -.124</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AvC .377</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td>sGBI-D→ NA</td>
<td>.228</td>
<td>.001</td>
<td>PSQI .091</td>
<td>.113</td>
<td>.010</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AcC -.110</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>SS -.124</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AvC .377</td>
<td>.278</td>
<td>.105</td>
</tr>
</tbody>
</table>

Note. A = Independent Variable; C = Dependent Variable; PSQI = State Sleep; AcC = Active Coping; SS = Support Seeking; AvC = Avoidant Coping; sGBI-D = sGBI-Depression; NA = Negative Affect; PA = Positive Affect; SWL = Satisfaction with Life; WU = Returning to Wakefulness.
### Table 31

**Comparison of Direct and Indirect Pathways in the Model containing sGBI-Hypomania Scores**

<table>
<thead>
<tr>
<th>A → C</th>
<th>Direct Effect size</th>
<th>p</th>
<th>Mediator (M) A→M</th>
<th>M → C</th>
<th>Indirect Effect size</th>
<th>p (total)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GTB → PA</td>
<td>-0.099</td>
<td>0.012</td>
<td>PSQI -</td>
<td>-</td>
<td>0</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AcC -</td>
<td>0.299</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>SS</td>
<td>0.111</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AvC</td>
<td>-</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td>WU → PA</td>
<td>0.327</td>
<td>0.001</td>
<td>PSQI</td>
<td>-</td>
<td>0.114</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AcC</td>
<td>0.178</td>
<td>0.299</td>
<td>0.053</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>SS</td>
<td>-</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AvC</td>
<td>-</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td>sGBI-H → SWL</td>
<td>-0.403</td>
<td>0.001</td>
<td>PSQI</td>
<td>0.143</td>
<td>-</td>
<td>0.092</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AcC</td>
<td>-</td>
<td>0.220</td>
<td>0.195</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>SS</td>
<td>-</td>
<td>0.145</td>
<td>0.109</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AvC</td>
<td>0.441</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td>sGBI-H → NA</td>
<td>0.344</td>
<td>0.002</td>
<td>PSQI</td>
<td>0.143</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AcC</td>
<td>-</td>
<td>0.220</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>SS</td>
<td>-</td>
<td>0.145</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AvC</td>
<td>0.441</td>
<td>-</td>
<td>0.231</td>
</tr>
</tbody>
</table>

*Note. A = Independent Variable; C = Dependent Variable; PSQI = State Sleep; AcC = Active Coping; SS = Support Seeking; AvC = Avoidant Coping; GTB = Going to Bed; sGBI-H = sGBI-Hypomania; NA = Negative Affect; PA = Positive Affect; SWL = Satisfaction with Life; WU = Returning to Wakefulness.*
Table 32

**Table of Aims and Hypotheses**

<table>
<thead>
<tr>
<th>Aim</th>
<th>Hypotheses</th>
<th>Support</th>
<th>Table</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>To explore previously determined associations between personality traits, motivation traits and vulnerability to BD.</td>
<td>1) That there will be a positive correlation between sGBI-depression scores and Neuroticism scores.&lt;br&gt;2) That there will be a negative correlation between sGBI-hypomania scores and Agreeableness scores.&lt;br&gt;3) That there will be a positive correlation between sGBI-hypomania scores and Extraversion scores.&lt;br&gt;4) That Neuroticism would positively predict vulnerability to BD (both sGBI-Depression and sGBI-Hypomania scores) in context of personality and motivation traits.&lt;br&gt;5) That one or more of the BAS subscales (BASd, BASfs, and BASrr scores) would positively predict sGBI-Hypomania scores.&lt;br&gt;6) That one or more of the BIS and BAS subscales (BASd, BASfs, and BASrr scores) would positively predict sGBI-Depression scores.</td>
<td>Full 10&lt;br&gt;Full 10&lt;br&gt;Not 10&lt;br&gt;Full 11 &amp; 12&lt;br&gt;Full 11&lt;br&gt;Full 12</td>
</tr>
<tr>
<td>2</td>
<td>To conduct a novel investigation of trait-like sleep variables as correlates of vulnerability to BD.</td>
<td>7) That there would be a negative correlation between Morningness and sGBI-Hypomania scores.&lt;br&gt;8) That there would be a negative correlation between Morningness and sGBI-Depression scores.&lt;br&gt;9) That there would be a negative correlation between at least one trait-like sleep variable (GTB, FA, SA, RS, and WU-higher scores indicating healthy sleep) and sGBI-Depression and sGBI-Hypomania scores.&lt;br&gt;10) That at least one of the trait-like sleep variables (GTB, FA, SA, RS, WU, or Morningness scores) would negatively predict vulnerability to BD (sGBI-Depression and sGBI-Hypomania scores) in the context of personality traits (operationalised as N, E, O, A, and C) and motivation traits (operationalised as BIS, BASd, BASfs, and BASrr scores).</td>
<td>Full 13&lt;br&gt;Full 13&lt;br&gt;Full 13&lt;br&gt;Full 14 &amp; 15</td>
</tr>
<tr>
<td>3</td>
<td>To investigate the role of state sleep within the two models (one containing sGBI-Depression scores and the other containing sGBI-Hypomania scores).</td>
<td>11) That state sleep (PSQI scores) would act as a mediator in models relating trait-like sleep variables (GTB, FA, SA, RS, WU, and Morningness), vulnerability to BD (sGBI-Depression containing model or sGBI-Hypomania containing model) and mood/wellbeing outcome variables (PA, NA, and SWL scores).</td>
<td>Partial 23, 30, &amp; 31 Mediation</td>
</tr>
<tr>
<td>4</td>
<td>To investigate the role of coping styles and understand how they operate in the context of sleep, vulnerability to BD and mood/wellbeing outcome variables.</td>
<td>12) That at least one coping style (AcC, SS, and AvC) would predict mood and wellbeing outcome in the context of vulnerability to BD (sGBI-Depression and sGBI-Hypomania scores).&lt;br&gt;13) That coping would act as a mediator in models relating trait-like sleep variables (GTB, FA, SA, RS, WU, and Morningness) and vulnerability to BD (sGBI-Depression containing model or sGBI-Hypomania containing model) to mood/wellbeing outcome variables (PA, NA, and SWL scores).</td>
<td>Full 17–22&lt;br&gt;Partial 23, 30, &amp; 31 Mediation</td>
</tr>
</tbody>
</table>

**Note.** Full= hypothesis fully supported; Not = hypothesis not supported; Partial Mediation = hypothesis partially supported; BASd = BAS Drive; BASfs = BAS Fun Seeking; BASrr = BAS Reward Responsiveness; AcC = Active Coping; SS = Support Seeking; AvC = Avoidant Coping.
8.0 Discussion

8.1 Overview of Chapter 8

The following chapter will discuss findings from the present project in relation to other published research. Findings regarding personality and motivation will be discussed first, followed by the role of sleep, and then the role of coping. An integration of findings and recommendations for future studies will follow. Limitations of the present study will be described and the chapter will finish with a general conclusion.

8.2 Aims

There were four research aims. The first was to explore previously determined associations between personality (FFM as measured by the API; Murray et al., 2009) and motivation (as measured by the BIS/BAS scales; Carver & White, 1994) trait correlates, and vulnerability to BD (sGBI-Depression and sGBI-Hypomania scores). The second aim was to conduct a novel investigation of trait-like sleep variables as correlates of vulnerability to BD. The third aim was to investigate the role of state sleep in the models relating trait-like sleep variables, coping styles, and mood/wellbeing variables to vulnerability to BD (in a model containing sGBI-Depression scores and in a model containing sGBI-Hypomania scores). Finally, the fourth aim of the study was to investigate the role of coping within these two models. Findings related to the aims and corresponding hypotheses will be discussed under three main sections (Personality and BIS/BAS as trait correlates of BD; Sleep and BD; and Coping).

8.2.1 Personality and motivation as trait correlates of BD.

The first aim was assessed by means of correlation and multiple regression analyses. Hypothesis 1, that there would be a significant positive correlation between sGBI-Depression and Neuroticism scores, was supported. This finding is consistent
with previous studies (Kim et al., 2011; Murray et al., 2007; Quilty et al., 2009) and speaks to the concept of Neuroticism and what it ascertains. Specifically, that individuals’ vulnerable to psychological distress will score higher on Neuroticism than individuals who are less vulnerable (McCrae & Costa, 1992).

Hypothesis 4, that Neuroticism would predict vulnerability to both sGBI-Depression and sGBI-Hypomania, in the context of all personality and motivation traits, was supported. This finding aligns with that of Quilty et al. (2009), but differs to that of Murray et al. (2007) and Lozano and Johnson (2001) who found that Neuroticism predicted only depression, and not hypomania scores. The link between Neuroticism and depression was maintained.

Regarding the prediction of sGBI-Hypomania scores, previous research has identified a significant large ($r = .50$) correlation between Neuroticism and trait-mania scores (Murray et al., 2007); and a significant weak correlation ($r = .24$) between Neuroticism and mania symptoms (Quilty et al., 2009). More recently however, and as previously described, Quilty et al. (2013) observed that lower facets of Neuroticism (specifically volatility and angry hostility) predicted mania severity scores. A possible explanation for Neuroticism predicting sGBI-Hypomania scores in the present study is that individuals with BD are observed to experience symptoms and episodes of depression more frequently than they experience mania (Beesdo et al., 2009). Another possible explanation for the association between Neuroticism and mania is that individuals who suffer from BD, who also report a higher incidence of depressive episodes, have possibly not yet experienced a manic episode, or under-report the occurrence of manic episodes (Perlis et al., 2006; Quilty et al., 2013).

Neuroticism predicted both sGBI-Depression and sGBI-Hypomania scores in the context of personality and motivation traits. This is a theoretically interesting
finding, as personality as a predictor of vulnerability to BD in the context of motivation traits has not been investigated before, and shows Neuroticism to be the strongest personality or motivation trait correlate of vulnerability to BD. As briefly discussed, the findings regarding Neuroticism continue to support the theory behind what the Neuroticism scale aims to measure, vulnerability to psychopathology. This finding is also consistent with work that has suggested that the recurrence of depressive episodes is influenced by Neuroticism traits in individuals diagnosed with BD I (Kim et al., 2011). Findings from the present project continue to support the construct of Neuroticism as a primary correlate of vulnerability to BD. The clinical implications from the findings regarding Neuroticism is that the behaviours that Neuroticism scales measure, specifically the facets of anxiety, hostility, impulsivity, depression, self-consciousness, and vulnerability be used as screening tools, not only in those who are suspected to meet criteria for a BD diagnosis, but also to individuals of the general public and those who may be experiencing sub-clinical mood disturbances. This could translate to treatments that could address stress management, mindfulness or CBT to address anxiety (Norton, Abbott, Norberg, & Hunt, 2015; Stratford, Cooper, Di Simplicio, Blackwell, & Holmes, 2015), and the importance of stopping and assessing the risks and benefits of a potentially appealing situation.

Hypothesis 2, that there would be a significant negative correlation between sGBI-Hypomania and Agreeableness scores, was also supported. This finding also aligns with work by Murray et al. (2007) and Quilty et al. (2009). Agreeableness is characterised by traits such as modesty, trust and compliance, and a lack of these traits is consistent with a manic episode (Quilty et al., 2009).

Hypothesis 3, that there would be a significant positive correlation between sGBI-Hypomania scores and Extraversion, was not supported. Instead, a significant,
moderate negative relationship was found. This finding is in contrast to the findings of Murray et al. (2007) and Quilty et al. (2009) who found a positive correlation between Extraversion and GBI hypomania scores. The sample and the measure used by the present study can be ruled out as possible explanations for this change in direction, as Murray et al. (2007) used a similar sample to the present project. Although, both Quilty et al. (2009) and Murray et al. (2007) employed the NEO-PI-R, the API has been found to be internally consistent and valid (Murray et al., 2009). It is possible that the relationship between sGBI-Hypomania and the positive emotional quality of Extraversion is more complex and possibly more unreliable as a measure of vulnerability to BD than previously thought.

Hypothesis 5, that at least one of the BAS subscale scores would positively predict sGBI-Hypomania in the context of all personality and motivation traits, was supported. BAS Fun Seeking predicted sGBI-Hypomania scores. This is in contrast to Jones et al. (2007) who found BAS Reward Responsiveness and BAS Fun Seeking to predict hypomania scores, but consistent with work by Meyer et al. (1999) who also found BAS Fun Seeking to be the only BAS subscale to predict mania scores derived from the GBI (Meyer et al., 1999).

Additionally Hypothesis 6, that at least one of the BIS and BAS subscale scores would positively predict sGBI-Depression scores in the context of all personality and motivation traits, was also supported. BAS Fun Seeking was again the only significant predictor of sGBI-Depression scores. This stands in contrast to research that found BIS predicted depressive symptoms (Meyer et al., 1999) and risk of depression in non-clinical samples (Alloy et al., 2008). These findings are also inconsistent to that of Markarian and colleagues (2013), who not only found that BIS significantly correlated with depression scores, but that both BIS and BAS had an indirect effect on depression,
anxiety, and stress scores via reported difficulty with emotion regulation. Findings regarding the predictive power of the BIS scale alone align with suggestions by Meyer et al. (2001), that BIS alone is not an operational vulnerability factor of BD and is rather dependent on state-depression symptomatology. Furthermore, although the BIS subscale significantly correlated with sGBI-Depression scores, when forced to share variance with the personality trait variables, it was no longer predictive of vulnerability to BD depression scores, meaning that, in the context of personality trait variables, BIS scores did not significantly account for the variance explained, but BAS Fun Seeking did.

The finding that BAS Fun Seeking was the sole predictor of sGBI-Depression scores should not come as a complete surprise. In addition to the predictive power of BAS Fun Seeking in relation to depression scores, correlation analyses from the present study revealed a positive, albeit weak, relationship between sGBI-Depression scores and the BAS Fun Seeking scale. This finding is in line with that of Meyer et al. (1999) who also observed a positive and weak relationship between GBI-Depression scores and the BAS Fun Seeking scale in a low risk sample. Furthermore, the BAS Fun Seeking scale has been previously employed to measure impulsivity, a key feature of mania in BD (Giovanelli et al., 2013). BAS Fun Seeking has predicted progression along the bipolar spectrum of diagnosis over time (Alloy et al., 2012). Even in individuals in BD remission, elevated levels of impulsivity have been noted (Strakowski et al., 2010; Swann, Anderson, Dougherty, & Moeller, 2001). Alloy et al. (2012) also suggested that vulnerability to BD may be due to underlying high impulsivity and BAS sensitivities. Furthermore, recent research identified trait impulsivity (as measured by the self-report scale the Barratt Impulsiveness Scale; Patton, Stanford, & Barratt, 1995) and impulsive decision-making (as measured by the behavioural task the Cambridge Gambling Task;
Cambridge Cognition, 2006) as a risk factor for developing BD, suggesting that impulsivity is viewed as an endophenotype of BD and another vulnerability marker of BD (Wessa et al., 2015). The clinical implications of these findings add support to the use of therapies that target the behaviours that the BAS Fun Seeking subscales measures. Specifically, addressing the desire to seek novel experiences with less regard for the consequences, such as spending money, alcohol or substance use, or getting into situations which may result in harm. The therapies could include behaviours that target impulsive behaviours, such as those used in Dialectical Behaviour Therapy as suggested by Moeller, Barratt, Dougherty, Schmitz, and Swann (2001), and contingent strategies or pharmacotherapy (Loree, Lundahl, & Ledgerwood, 2015). These therapies could be applied not just to individuals already diagnosed with BD, but to those who are experiencing sub-clinical levels of BD affective disturbances and those who are at risk of BD (e.g., siblings, offspring, and unaffected relatives).

Personality traits and the BAS subscale of Fun Seeking predicted sGBI-Depression and sGBI-Hypomania scores in a similar way to that of previous research. These findings supported the five of the six hypotheses related to the first aim. Findings of the present study suggest that vulnerability to BD is associated with high scores on scales that measure Neuroticism, and low scores on scales measuring Extraversion, Agreeableness, and Conscientiousness. Although sGBI-Depression scores significantly correlated (positively) with all subscales of the BIS/BAS scales, and sGBI-Hypomania correlated with BIS (again positively), the only BIS/BAS subscale, in the context of all personality and motivation traits, that was positively associated to sGBI-Depression and sGBI-Hypomania scores was that of BAS Fun Seeking. BAS Fun Seeking has been associated with dysfunctional impulse control (Leone & Russo, 2009), which overlaps with Criterion B7 for a Manic Episode (American Psychiatric Association, 2013).
Furthermore, a facet captured by Neuroticism is also impulsivity. It is possible that there is content overlap in items that measure Neuroticism and BAS Fun Seeking. These findings suggest that scoring high on vulnerability to BD is associated with scoring high on measures of impulsivity (Giovanelli et al., 2013). Findings from the present project indicate that another trait correlate of vulnerability to BD is impulsive behaviour (as measured by both Neuroticism and BAS Fun Seeking). This aligns with recent research suggesting that not only is impulsivity a trait of BD, but that it is also considered an endophenotype of BD (Lombardo et al., 2012; Wessa et al., 2015). However, to be considered a true endophenotype, impulsivity scores would have to be shown to be heritable and also biologically correlated (Lombardo et al., 2012).

The variables of personality and BIS/BAS subscales were not included in the regression-based mediation and SEM analyses. They were only included to explore previously determined associations between personality and motivation trait correlates and vulnerability to BD (sGBI-Depression and sGBI-Hypomania scores) in order to understand vulnerability to BD from a personality and motivation viewpoint. Findings from the present project provide some validity evidence for the use of the sGBI to measure vulnerability to BD. Although it is too late for the present project, another measure that could be employed to measure vulnerability to BD that also aligns with a dimensional view of BD is the 7 Up 7 Down Inventory (Youngstrom et al., 2013).

The sGBI subscales scores were associated with personality and motivation traits that have been previously associated with vulnerability to BD, specifically, with Neuroticism and BAS Fun Seeking. Advantages of employing the sGBI-subscale scores over personality and motivation traits include the sGBI's ability to measure the dimensionality of BD: Neuroticism rates individuals on their vulnerability to general psychological distress (not specific to BD; McCrae & Costa, 1992); BAS Fun Seeking
has been correlated with impulsive behaviour, but impulsive behaviour is also characteristic of other psychological diagnoses (e.g., Carli et al., 2014; Dolan & Anderson, 2002; Heinrich, 2013; Loney, Frick, Clements, Ellis, & Kerlin, 2003; Smulders, Esselink, Cools, & Bloem, 2014; Tomassini et al., 2012); and both Neuroticism and BAS Fun Seeking measure impulsivity.

8.2.2 Sleep and BD.

The second aim was to conduct a novel investigation of trait-like sleep variables as correlates and predictors of vulnerability to BD. There were four hypotheses tested in relation to this aim (Hypotheses 7, 8, 9, and 10). Hypothesis 7, that there would be a negative relationship between Morningness and sGBI-Hypomania scores, was supported. Although the relationship was significant and negative in direction, it was weak in size (-.21). Hypothesis 8, that there would be a negative relationship between Morningness and sGBI-Depression scores, was also supported. Again, although the relationship was significant and negative in direction, it was weak in size (-.15). Despite the size of the relationships, the findings regarding Hypotheses 7 and 8 provide some evidence that individuals vulnerable to BD are more likely score lower on measures of Morningness, which may suggest a trait preference for evening activity. Furthermore, the magnitude of the relationships that the present study identified were comparable to those of previous research that also observed that individuals diagnosed with BD scored lower on Morningness (Mansour et al., 2005; Wood et al., 2009). Both Mansour et al. (2005) and Wood et al. (2009) identified relationships that were weak to moderate in size (correlations values described in section 3.4.3), and Bullock et al. (2014), who, like the present project, employed a student sample, also observed a weak, but significant, correlation between morningness and depression-proneness. Findings from the present study are consistent with those of previous studies that have found a trait-evening
preference in individuals who are either vulnerable (Bullock et al., 2014) or diagnosed with BD (Ahn et al., 2008; Mansour et al., 2005; Wood et al., 2009). These findings also suggest that an internal preference for eveningness may be another endophenotype of BD, which is consistent with investigations of circadian function as an endophenotype (Lenox et al., 2002). These findings suggest that another early vulnerability marker of BD, or variable that could be included in the early screening of BD, is chronotype preference. Clinicians could view individuals who endorse a higher preference for evening activity as being more vulnerable or susceptible to a BD diagnosis. Clinicians could also tailor therapy to address chronotype preference, like that of IPSRT, where the regulation of daily rhythms, particularly decreasing activities that may disrupt circadian rhythms, is a focus.

Hypothesis 9, that there would be a negative relationship between trait-like sleep variables (higher scores indicative of healthy sleep) and both sGBI-Depression and sGBI-Hypomania scores, was also fully supported. All five subscales of the modified ASWS negatively correlated with sGBI-Depression and sGBI-Hypomania scores. The magnitude of these correlations ranged from weak (- .15) to moderate, with the largest correlation being that between FA and sGBI-Hypomania scores (- .39). Results suggest that vulnerability to BD is associated with difficulty getting to bed, falling asleep, maintaining sleep, reinitiating sleep if awoken, waking up in the morning, and a preference to be active relatively later in the day.

Sleep disturbances are documented as precursors to the onset of BD (Ritter et al., 2015) and persist during periods of wellness (Brill et al., 2011; Harvey et al., 2005). They have been documented to occur in adolescence in individuals who subsequently develop BD (Lunsford-Avery et al., 2012; Ritter et al., 2011). Sleep difficulties continue to be a problem, as approximately 70 % of individuals with BD suffer from clinically
disturbed sleep (Harvey et al., 2005) which persists during periods of euthymia (Brill et al., 2011; Harvey et al., 2005). Findings from the present study add further support to the concept that sleep-dysregulation is a trait-like core feature of BD (Jones et al., 2006).

8.2.2.1 Sleep as a predictor of vulnerability to BD.

To further investigate trait-like sleep variables as correlates of vulnerability to BD, the present project investigated if trait-like sleep variables still predicted vulnerability to BD when forced to share variance with (within the context of) personality (FFM) and motivation (BIS/BAS subscales) traits. Hypothesis 10, that at least one trait-like sleep variable would negatively predict vulnerability to BD when forced to share variance with personality and motivation traits, was supported. This hypothesis was tested in two stages, the first stage regression analyses only included trait-like sleep variables as predictors of either sGBI-Depression or sGBI-Hypomania scores (Model 1), and the second stage included trait-like sleep variables as well as personality and motivation traits to predict either sGBI-Depression or sGBI-Hypomania scores (Model 2). The first stage analyses indicated that the subscales of GTB (largest predictor), SA and RS, negatively predicted sGBI-Depression scores. All subscales of the modified ASWS (GTB, FA, SA, RS, and WU) negatively predicted sGBI-Hypomania scores, with the largest being WU scores. Interestingly, although Morningness negatively correlated with both sGBI-Depression and sGBI-Hypomania scores in the bivariate analyses, in the context of the other trait-like sleep variables (Model 1), and trait-like sleep, personality, and motivation traits (Model 2), it positively predicted both sGBI-Depression and sGBI-Hypomania scores. However, this is likely due to a suppression effect as Morningness correlated highly with WU ($r = .74$) and GTB ($r = .56$) scores.
When personality and motivation traits were included in the prediction of sGBI-Depression scores, GTB and RS scores remained significant negative predictors, FA became significant, and both SA and Morningness no longer predicted of sGBI-Depression scores. Regarding the prediction of sGBI-Hypomania scores, only GTB and FA remained significant negative predictors in the context of personality and BIS/BAS subscale scores, and FA became slightly more significant ($p < .05$ to $p < .01$). The results suggest that some trait-like sleep variables predicted sGBI-Depression and sGBI-Hypomania scores in the context of personality and motivation traits. These findings suggest that trait-like sleep variables (GTB and FA particularly) were stronger predictors of vulnerability to BD (sGBI-Depression or sGBI-Hypomania scores) than the previously investigated quality of chronotype (Morningness; Bullock et al., 2014; Mansour et al., 2005; Wood et al., 2009).

Employing the modified ASWS highlighted the importance of measuring trait-like sleep variables. As previously mentioned, the ASWS was modified to measure trait-like sleep, however, testing the psychometric properties of the modified ASWS (beyond internal consistency) was beyond the scope of the present project. Thus, there is still a need for measures that enquire about trait-like sleep variables. Future research could test the modified ASWS used in the present project for qualities such as test-retest reliability. Furthermore, when considering items for scales that measure trait-like sleep variables it would be pertinent to continue to include items that enquire about behaviours related to falling asleep (such as being unable to settle or how long it takes to fall asleep), going to bed (avoidance or putting off going to bed), and reinitiating sleep if woken up through the night (the degree of difficulty experienced going back to sleep or how long it takes to return to sleep). Furthermore, as Ritter et al. (2015) found, sleep
disturbances preceded the onset of BD, particularly related to falling asleep; thus, screening tools could be employed in the early detection of affective disturbance.

Moreover, although Neuroticism remained the strongest predictor of sGBI-Depression and sGBI-Hypomania scores, some trait-like sleep variables remained significant. These findings suggest for the first time that despite one’s ‘vulnerability to psychopathology’, difficulties going to bed (for sGBI-Depression and sGBI-Hypomania scores), falling asleep (for sGBI-Depression and sGBI-Hypomania scores), and reinitiating sleep (for sGBI-Depression scores only) play a role in vulnerability to BD. Findings from the present project have implications for the current understanding of BD, encouraging more research into the sleep behaviours of falling asleep and getting to bed as critical features of vulnerability to BD, possibly even more important that Morningness (which they are highly correlated with).

Trait-like sleep variables are important in the detection of proneness to BD in the general population and should be considered when investigating vulnerability to BD. These findings also indicate that individuals who have difficulty with trait-like sleep behaviours may be more at risk or vulnerable to affective disturbance, than individuals who experience healthy sleep (Ritter et al., 2015). These findings highlight the need to conduct further research into the importance of sleep and circadian rhythm dysregulation in the aetiology of BD (Gruber et al., 2009; Hudson et al., 1992; Jones et al., 2006; Robillard et al., 2013; Wehr, Sack, & Rosenthal, 1987). This further suggests and also supports the concept that sleep disturbance is an endophenotype of BD (Lenox et al., 2002). Lenox et al. (2002) state that in order for markers to be considered an endophenotype they need to be heritable, associated with illness, be state independent (meaning they would occur during periods of euthymia), occur within the family, and that the prevalence of the marker occur in non-affected relatives at a higher rate than the
general population. The findings of the present project suggest that sleep difficulties are state independent and are associated with degree of vulnerability to BD. More research is required to assert sleep disturbance as an endophenotype of BD. Thus, it is recommended that future studies employ a longitudinal approach that investigates the trait-like sleep difficulties of falling asleep and going to bed, whilst addressing each of the criteria outlined by Lenox et al. (2002), mentioned above. To do so, would require a large affected familial sample, as well as a large sample of healthy matched control families, whilst recording affective symptoms, sleep disturbances, and prevalence of conversion to BD.

Given the clinical application of the findings of the present project regarding trait-like sleep variables, and taken into consideration along with recent research that found sleep quality preceded the onset of BD (Ritter et al., 2015), it is recommended that trait-like sleep variables be considered early screening markers when an individual is at risk (e.g., offspring, sibling, or unaffected relative) or is displaying sub-clinical levels of affective disturbance. Finally, although current psychological interventions that treat BD recognise the importance of sleep, the findings of the present project emphasise the importance of screening for poor sleep and potentially using that as an engagement strategy when working with clients.

8.2.2.2 Mediation by State Sleep.

Following investigations of trait-like sleep variables, the present project sought to investigate the role of state sleep in the two BD, Sleep and Coping models that included trait-like sleep variables, vulnerability to BD (sGBI-Depression scores in the first model and sGBI-Hypomania scores in the second model), and mood/wellbeing variables. The separation of sleep into trait-like and state variables was novel to the present project and so the following analyses were conducted to see if that separation
was supported by the data. Analyses investigating the potential mediating role of state sleep were done in two stages. The first stage included regression-based mediation analyses which tested one IV (sGBI-Depression or sGBI-Hypomania scores), the one mediator (state sleep as measured by PSQI scores) and one DV (either coping styles of Active Coping, Support Seeking or Avoidant Coping, or mood and wellbeing variables, specifically PA, NA or SWL). The second stage included SEM where multiple IV’s (all trait-like sleep variables and sGBI-Depression or sGBI-Hypomania scores), mediators (PSQI and all three coping styles) and DV’s (PA, NA, and SWL) could be examined at the same time.

8.2.2.2.1 State Sleep as a mediator between vulnerability to BD and Coping.

Sleep deprivation is associated with making poorer coping choices, and coping may moderate sleep changes (Sadeh et al., 2004). The present project sought to investigate if this relationship between sleep and coping was reciprocal. In order to test if state sleep (as measured by PSQI score) influenced coping within the BD, Coping, and Sleep models it was important to first investigate if state sleep acted as a mediator between vulnerability to BD (sGBI-Depression or sGBI-Hypomania scores) and coping styles. This was performed using regression-based mediation analyses. Results indicated that state sleep (PSQI scores) acted as a partial mediator between: sGBI-Depression scores (IV) and each of the three coping styles (Active, Support Seeking, and Avoidant); between sGBI-Hypomania scores and Active coping; and between sGBI-Hypomania scores and Support Seeking. This finding is consistent with previous findings that have found healthy individuals, who have suffered from sleep deprivation, make poorer coping style choices (Sadeh et al., 2004). This could be because problem solving (Killgore et al., 2008; Kjellberg, 1975; Sadeh et al., 2004), emotional
intelligence, constructive thinking (Killgore et al., 2008) and decision making (Glass et al., 2011; McKenna et al., 2007) are impaired when sleep deprived.

8.2.2.2.2 State Sleep as a mediator between vulnerability to BD and mood/wellbeing variables.

The next phase of analyses investigated if state sleep acted as a mediator between vulnerability to BD (sGBI-Depression or sGBI-Hypomania) and mood/wellbeing outcome variables. Regression-based mediation analyses were conducted to investigate if state-sleep (PSQI scores) acted as a mediator between sGBI-Depression or sGBI-Hypomania scores, and each of the three mood/wellbeing variables (PA, NA, or SWL scores). Sobel Z-statistics showed that state-sleep scores acted as a partial mediator between: sGBI-Depression and NA scores; between sGBI-Depression and SWL scores; between sGBI-Hypomania and PA scores; between sGBI-Hypomania and NA scores; and between sGBI-Hypomania and SWL scores.

SEM analyses were then carried out. Full mediation by state sleep was not supported by the SEM analyses as there were pathways that went directly from some trait-like sleep variables and either sGBI-Depression or sGBI-Hypomania scores to mood and wellbeing outcome variables, bypassing state sleep. However, state sleep (PSQI scores) acted as a partial mediator between vulnerability to BD (sGBI-Depression containing model and sGBI-Hypomania containing model), coping variables, and mood/wellbeing outcome variables (PA, NA and SWL). Furthermore, SEM analyses revealed that in both models (one containing sGBI-Depression scores and the other containing sGBI-Hypomania scores) state sleep (PSQI scores) had a direct pathway to Support Seeking coping scores, meaning that it not only acted as a partial mediator between vulnerability to BD and mood/wellbeing variables (state mood and SWL), but that state sleep also influenced the coping style of Support Seeking. Findings
from the SEM analyses showed that the data fit a model in which trait-like sleep variables precede sGBI-Depression and sGBI-Hypomania scores and that state sleep acts as a partial mediator between trait-like sleep, vulnerability to BD, and measures of mood and wellbeing (state mood and SWL). These findings are consistent with previous research that has found sleep deprivation to impair mood (Fortunato & Harsh, 2006), and quality of life in BD (Giglio et al., 2009) and in healthy controls (Molzon et al., 2013). These findings suggest that, irrespective of where one sits on the continuum of vulnerability to BD, state sleep influences mood and wellbeing outcomes, asserting the importance of a good night’s sleep on perceived mood and satisfaction with life.

**8.2.2.3. Summary of sleep findings.**

The investigations regarding the role of state and trait-like sleep variables are novel to this project, as no other study, to the author's knowledge, has investigated the potential mediating effects of state sleep in models relating vulnerability to BD, coping and mood/wellbeing variables, nor has sleep been separated into trait-like and state sleep in the same study. Findings from present project suggest that not only is sleep important in the prediction of vulnerability to BD, but also that it can be conceptually separated into state and trait-like components. State-sleep has been found to impact on mood and wellbeing, and trait-like sleep has been shown to predict vulnerability to BD scores in the context of personality and motivation traits. Also, state-sleep appears to impact the coping style of support seeking, suggesting that state-sleep quality influences the seeking of support. The implications of the findings from the present project align with those of Rocha et al. (2013) who asserted that sleep acts as both a state and trait marker of BD.

Findings regarding sleep from the present project add further support to research suggesting that sleep difficulties are an endophenotype of BD (Lenox et al., 2002).
Findings from the present project align with research that consistently suggested, but not explored the association between *trait-like* sleep and BD. The trait-like findings of the present project regard the prediction of sGBI-Depression and sGBI-Hypomania scores by trait-like sleep variables when forced to share variance with personality and motivation traits. This is consistent with findings by Ritter et al. (2015) who observed that healthy adolescents that suffer from sleep disturbances later developed BD (Ritter et al., 2015). The findings of the present project also align with those of Brill et al. (2011) and Harvey et al. (2005) who observed sleep difficulties persisted during periods of euthymia. Findings also suggest that early screening tools should include measures of trait-like sleep qualities, and, as Ritter et al. (2015) suggested, that future studies investigate the protective effects of stabilising sleep in those vulnerable to BD.

Finally, findings regarding state sleep and mood/wellbeing variables suggest that a way of improving state mood and SWL in the general population is to improve state sleep. This supports multiple works, as previously mentioned, that found that sleep deprivation negatively impacted mood (Fortunato & Harsh, 2006; Franzen et al., 2008; Talbot et al., 2010; Zohar et al., 2005) and perceived wellbeing (Molzon et al., 2013).

Findings from the present project can be applied to members of the general population, individuals who are at risk of BD (poor sleepers, siblings, offspring, or unaffected relatives of BD) and individuals with affective disturbances that do not yet meet criteria for BD. Clinically, these findings suggest that one way to reduce vulnerability to BD, may be to employ trait-therapies targeting long-term sleep habits. These could include the employment of: behavioural changes like sleep hygiene (Stokes & Stokes, 2013), relaxation techniques or CBT for Insomnia (Lichstein et al., 2012; Trauer, Qian, Doyle, Rajaratnam, & Cunnington, 2015; Vallières, Morin, Guay, Célyne, & LeBlanc, 2004); physiological changes such as not consuming caffeinated beverages
before bed (Owens, Mindell, & Baylor, 2014); or hypnotic medication (Harrison & Keating, 2005; Neubauer, 2014). Future research could investigate the long term effect of sleep-targeted therapies in individuals that are deemed vulnerable to BD (either unaffected relatives or individuals who score high on measures of vulnerability to BD) and observe the rate of BD diagnoses that later manifest in this population.

8.2.3 Coping.

One of the main foci of the present project was to better understand the role of coping in a quantitative conceptualisation of BD that includes the important role of sleep (in state and trait-like forms). Biological and genetic factors, which may predispose someone to BD, are resistant to change. Nonetheless, clinicians may be able to improve mood and wellbeing outcomes by teaching more adaptive and flexible ways of coping with stressful events. Thus, the ultimate aim of the present study was to investigate if coping acted as a mediator in two models relating vulnerability to BD (one model containing sGBI-Depression scores and the other containing sGBI-Hypomania scores) to mood/wellbeing variables. If this could be demonstrated, it would make for stronger scientific grounds encouraging the use the evidence-based psychological interventions for BD. This aim was examined using hierarchical regression, regression-based mediation (Sobel Z-statistic) and SEM analyses.

8.2.3.1 The role of Coping within the BD, Coping, and Sleep models.

Before investigating if coping acted as mediator within the two models, it was important to investigate if coping first predicted mood/wellbeing outcome variables. Hypothesis 12, that at least one coping variable would predict mood/wellbeing variables (PA, NA and SWL scores) in the context of vulnerability to BD (by sharing variance with either sGBI-Depression or sGBI-Hypomania scores), was examined using hierarchical regression analyses. There were two stages to these analyses, the first stage
included either sGBI-Depression and sGBI-Hypomania scores as the IV predicting either PA, NA, or SWL scores (Model 1). The second stage of these analyses involved the inclusion of all three coping styles (Active, Support Seeking, and Avoidant coping) and either sGBI-Depression or sGBI-Hypomania scores as IV’s (Model 2). Hypothesis 12 was supported as different coping styles predicted different mood/wellbeing variables over and above variance explained by vulnerability to BD scores. Analyses indicated that SWL was significantly predicted by: Active coping (in the context of sGBI-Depression scores and sGBI-Hypomania scores); Support Seeking (in the context of both sGBI-Depression and sGBI-Hypomania scores); and Avoidant Coping scores (only in the context of sGBI-Depression scores). PA scores were significantly predicted by Active Coping scores (in the context of both sGBI-Depression and sGBI-Hypomania scores), and, in fact, Active Coping scores explained the most variance in PA scores (even in the context of sGBI-Depression and sGBI-Hypomania scores). Although both sGBI-Depression and sGBI-Hypomania scores were significant predictors of NA scores, significant variance was explained by the coping style of Avoidant Coping. The results suggest that even in the context of vulnerability to BD (as measured by sGBI-Depression and sGBI-Hypomania scores), coping styles also predict important mood/wellbeing outcomes.

These findings are consistent with previous research finding that mood and wellbeing is predicted by coping strategies and styles (Bucks et al., 2011; Christensen & Kessing, 2005; Jones et al., 2006; Lam et al., 2001; Nolen-Hoeksema, 1991; Pavlickova et al., 2013; Peay, Rosenstein, & Biesecker, 2013). To reiterate, Pavlickova et al. (2013) found that adaptive coping and risk taking predicted PA. Furthermore, in individuals with BD, passive coping strategies (maladaptive) increased with the number of
depressive or hypomanic episodes experienced by an individual with BD (Pavlickova et al., 2013).

When comparing coping styles employed by individuals with BD, unaffected relatives (of someone with a BD diagnosis) and healthy controls, Green et al. also found that not only did individuals with BD consistently over-use maladaptive coping strategies, but unaffected relatives did too (Green et al., 2011). Findings from the present project further assert the importance of educating individuals, not just on the spectrum of BD or those who are considered unaffected relatives, but any member of the general population, or those in high risk samples who may be experiencing their first episode, about consciously employing more adaptive coping responses in order to improve perceived mood and wellbeing. Furthermore, once identified as at risk of BD, it is also encouraged that psychoeducation therapy be made available to these individuals, as it has been suggested that such therapies could, in combination with medication use, possibly prevent BD (Vieta & Colom, 2004).

8.2.3.1.1 Coping as a mediator between vulnerability to BD and State Sleep.

As described in section 8.2.2.2.1, in order to test the role of coping styles (Active Coping, Support Seeking, and Avoidant Coping) within the BD, Coping, and Sleep models it was important to first assess if coping acted a mediator between vulnerability to BD (sGBI-Depression or sGBI-Hypomania scores) and state sleep (PSQI scores). Coping predicted sleep in healthy adults (Sadeh et al., 2004). The present project sought to investigate if coping styles also influenced state sleep in the general population who were measured on a continuum of vulnerability to BD. This was investigated in two different analyses, regression-based mediation analyses and modelling-based analyses (summarised in the next section). Analyses indicated that the following mediating interactions were present: Active Coping acted as partial mediator
between both sGBI-Depression and state sleep (PSQI scores), and between sGBI-Hypomania and state sleep (PSQI scores); Support Seeking acted as a partial mediator between both sGBI-Depression and state sleep (PSQI scores), and between sGBI-Hypomania and state sleep (PSQI scores); and Avoidant coping acted as a partial mediator between sGBI-Depression and state sleep (PSQI scores). The regression-based analyses suggested that coping styles would improve sleep quality; however, the regression-based analyses were limited to the analysis of one IV (either sGBI-Depression or sGBI-Hypomania scores), one mediator (any one of the three coping styles), and one DV (PSQI scores).

However, as previously mentioned in Section 8.2.2.2.2, when all variables were included in the SEM analyses (which has the advantage of including multiple IV's, mediators and DV's at the same time), none of the three coping styles impacted on state sleep. In fact, state sleep (PSQI scores) had a direct pathway to Support Seeking scores, meaning that coping did not influence state sleep within of the BD, Sleep, and Coping models (containing either sGBI-Depression or sGBI-Hypomania scores). Findings from the present project do not suggest that coping styles impact the state sleep consequences of vulnerability to BD, in the context of trait-sleep variables.

8.2.3.1.2 Mediation of Coping Styles between vulnerability to BD and mood/wellbeing variables.

The final hypothesis, that coping would act as a mediator within the BD, Coping, and Sleep models, was partially supported. Again, this question was investigated using regression-based analyses and SEM-based analyses. Regression-based mediation analyses (Sobel Z-statistic) examined one IV (sGBI-Depression or sGBI-Hypomania scores), one mediator (either Active Coping, Support Seeking, or Avoidant Coping) and one DV (either PA, NA, or SWL scores) at a time. The
regression-based analyses identified that Active Coping scores acted as a partial mediator between: sGBI-Depression and NA scores; between sGBI-Depression and SWL scores; between sGBI-Hypomania and PA scores; and between sGBI-Hypomania and SWL scores. Support Seeking was found to act as a partial mediator between: sGBI-Depression and SWL scores; between sGBI-Hypomania and PA scores; and between sGBI-Hypomania and SWL scores. Finally, Avoidant Coping was found to act as a partial mediator between: sGBI-Depression and NA scores; between sGBI-Depression and SWL scores; between sGBI-Hypomania and NA scores; and between sGBI-Hypomania and SWL scores.

These analyses suggest that coping style does have an impact on perceived wellbeing and state mood. Analysis of the final BD, Coping, and Sleep model containing the sGBI-Depression scores, produced by the SEM analyses, suggested that Avoidant Coping continued to act as a partial mediator between sGBI-Depression scores and NA scores, and between sGBI-Depression and SWL scores, alongside direct pathways between sGBI-Depression scores and all three mood/wellbeing variables (PA, NA, and SWL). SEM analyses assessing the BD, Coping, and Sleep model containing sGBI-Hypomania scores revealed that alongside direct pathways between sGBI-Hypomania and NA scores, and sGBI-Hypomania and SWL scores, all three coping styles acted as partial mediators between sGBI-Hypomania scores and mood/wellbeing variables. The specific partial mediating pathways that remained were: sGBI-Hypomania (IV) to Active Coping (partial mediator) to PA (DV) scores; sGBI-Hypomania (IV) to Active Coping (partial mediator) to SWL scores (DV); sGBI-Hypomania (IV) to Support Seeking (partial mediator) to SWL scores (DV); and sGBI-Hypomania (IV) to Avoidant Coping (partial mediator) to NA scores (DV).
The regression-based analyses suggested that coping styles would improve mood and wellbeing outcome variables; however, the regression-based analyses were limited to the analysis of one IV (either sGBI-Depression or sGBI-Hypomania scores), one mediator (any one of the three coping styles) and one DV (either PA, NA, or SWL scores). When all variables were included in the SEM analyses (which as previously stated has the advantage of including multiple variables at the same time), coping styles continued to directly impact on mood and wellbeing outcomes. In both models there were direct pathways from: Active Coping to PA; from Active Coping to SWL; from Support Seeking to SWL; and Avoidant Coping to NA. Also, in the model containing sGBI-Depression scores, there was a direct pathway from Avoidant Coping to SWL. Findings from the present project do suggest that coping styles impact mood and wellbeing consequences of vulnerability to BD, in the context of trait-sleep variables.

Both the regression-based mediation and SEM analyses supported the importance of coping styles on perceived mood/wellbeing variables. Results of the present study continue to highlight the importance of coping styles on mood/wellbeing outcome variables such as SWL and state mood. These findings continue to support theorists such as Carver and Smith (2010), Skinner and Leigh (1980) and Folkman and Moskowitz (2004), who have acknowledged the importance of coping in managing stress and add further support to previous research emphasising the importance of coping styles on mood and wellbeing (Bucks et al., 2011; Christensen & Kessing, 2005; Jones et al., 2006; Lam et al., 2001; Nolen-Hoeksema, 1991; Pavlickova et al., 2013; Peay et al., 2013). Findings suggest that anyone who may be experiencing poor affect or low SWL be taught to employ adaptive coping strategies to alleviate stress, such as seeking help from appropriate sources, taking action to improve the situation, coming up with strategies and steps to improve the situation, instead of engaging in unhelpful
maladaptive strategies such as denial, giving up, and becoming complacent. Furthermore, the present study was able to demonstrate that coping styles influence mood and wellbeing outcomes, continuing to provide scientific grounds for the use of psychological interventions.

8.3 Integration and Implications of Findings

The BD, Coping, and Sleep models investigated were complex. The conceptual model was separated into two models, with the only difference being that one contained sGBI-Depression scores and the other contained sGBI-Hypomania scores (as the variables measuring vulnerability to BD). The main emphasis of the present study was to investigate the role of state sleep and coping styles within the BD, Coping, and Sleep models that also contained trait-like sleep variables, vulnerability to BD, and mood/wellbeing. The present study identified multiple interactions between the variables tested, as summarised in Figure 8.

![Diagram](image)

*Figure 8.* The final common pathways identified by the present study. Vulnerability to BD = sGBI-Depression or sGBI-Hypomania scores. Refer to Figure 4 on p.120 for the conceptual model and hypotheses.
The primary findings of the present project are threefold. The first is the suggestion that trait-like sleep variables can be measured as a predictor of vulnerability to BD. Trait-like sleep variables predicted vulnerability to BD in the context of Neuroticism. This finding aligns with the plethora of studies that continue to show that sleep is vital in the aetiology of BD (Bauer et al., 2006; Eidelman, Talbot, Gruber, Hairston, et al., 2010; Giglio et al., 2009; Gruber et al., 2011; Harvey et al., 2006; Kaplan & Harvey, 2013; Mehl et al., 2006; Murray & Harvey, 2010; Papadimitriou, Dikeos, & Soldatos, 2003; Ritter et al., 2011; Robillard et al., 2013; Salvatore et al., 2008; Sierra et al., 2007; Sylvia et al., 2012; Talbot et al., 2009; Wehr et al., 1987). This finding is important for the field as it shows that trait-like sleep is an important construct to further explore when investigating vulnerability to, or the aetiology of BD. Figure 8 shows that trait-like sleep directly impacted vulnerability to BD, state sleep, coping, and mood/wellbeing variables, warranting further investigation into the role of trait-like sleep variables. Despite SEM analyses showing that both the chi-square ($\chi^2$) and Bollen Stine’s $p$ did not suggest good fit by the models, good fit was indicated by all of the commonly accepted model fit indices (e.g., RMSEA, SRMR, GFI, AGFI, TLI and CFI). Furthermore, as previously mentioned, non-normality can impact chi-square and Bollen Stine’s $p$. Thus, the present project validated the separation of sleep into state and trait-like components. Both models produced good fit indices when trait-like sleep and state sleep variables were entered as separate variables, and pathways went from trait-like sleep variable to state sleep; these pathways were not reciprocal. These findings challenge the concept that sleep should only be measured as if it is a state feature.

These findings encourage the development and employment of questionnaires or scales that enquire about trait-like sleep (for most of one's life) variables and not just
state sleep (the past few days to month) for general and clinical populations. As mentioned in section 8.2.2.1, reliability and validity testing should be carried out on the modified ASWS. Given the large correlations between the Waking Up and Going to Bed subscales and Morningness, it is not necessary to include chronotype in these scales. Alternatively, other scales which enquire about difficulties falling asleep and sleep quality as Ritter et al. (2015) recommended, could also be developed. Either way, the present project showed the need for measures that assess trait-like sleep.

The second finding concerns the role of state sleep. The findings of the present project suggest that state sleep acts as a partial mediator (shown by both the regression-based mediation analyses and SEM) between vulnerability to BD and mood/wellbeing outcome variables. Furthermore, state sleep directly impacted the coping style of Support Seeking, suggesting that state sleep influences the way in which individuals cope with stress. The reverse cannot be said, in that coping styles (Active Coping, Support Seeking, or Avoidant Coping) did not act as mediators between the vulnerability to BD scores (in the regression-based mediation analyses or the SEM analyses) or trait-like sleep variables (in the SEM analyses) and state sleep. Figure 8 also shows that vulnerability to BD pathways linked directly to mood/wellbeing variables, but also via state sleep. These pathways add further support to the already acknowledged claim that sleep may be a mechanism in the cause of BD (Lenox et al., 2002; Ritter et al., 2011; Rocha et al., 2013; Sylvia et al., 2012). Kaplan and Harvey (2013) investigated the effect of implementing CBT-I in 15 participants who had a diagnosis of BD I and insomnia. They found that implementing sleep restriction (regular bed and wake times) and stimulus control was enough to see improvements in sleep (Kaplan & Harvey, 2013). They recommended the following behavioural clinical interventions: monitoring sleep (from time to fall asleep, time awake during the night,
wake-up time, and time spent napping); monitoring BD mood symptoms; monitoring sleepiness; implementing regular sleep schedules throughout the week (including weekends); implementing stimulus control; encouraging support networks to make the person adhere to bed times and stimulus control; rewarding positive sleep behaviours; and encouraging the continued use of sleep restriction and stimulus control once formal treatment has ended. As Kaplan and Harvey (2013) investigated the effect of CBT-I in a sample who were already diagnosed with BD I, future research is encouraged to develop or implement strategies that focus on improving sleep in those not yet diagnosed (being anyone at risk such as offspring, siblings, unaffected relatives of BD, or poor sleepers) and investigating if this decreases vulnerability to developing BD. Another suggestion for future research is to investigate possible causes of disturbed sleep, like that of Levenson et al. (2013), who proposed a conceptual model explaining disturbed sleep via the impact of life events.

The third significant finding to come out of the present project is that mood/wellbeing outcome variables can be improved by focussing on coping styles, over and above the degree of vulnerability to BD. As can be seen in Figure 8, state sleep impacted on coping styles, which then influenced mood/wellbeing variables. Findings suggest that mood/wellbeing outcomes can be improved by focussing on coping styles, supporting several studies that have focussed on coping styles and outcome (Bucks et al., 2011; Christensen & Kessing, 2005; Jones et al., 2006; Lam et al., 2001; Nolen-Hoeksema, 1991; Pavlickova et al., 2013; Peay et al., 2013). If someone is predisposed to coping less well, it could make them more likely to experience negative mood (Catanzaro, Wasch, Kirsch, & Mearns, 2000). This was observed by Jones et al. (2006), who observed that affected children of parents with a BD diagnosis displayed more maladaptive coping styles (risk taking) than unaffected controls, and unaffected children
of BD parents. This interpretation of findings alludes to the importance of identifying maladaptive coping styles, and the education and implementation of adaptive coping styles to improve mood and wellbeing outcomes. It is hoped that implementing these behavioural changes could mitigate the manifestation of BD at a later age. Future research could conduct longitudinal research that has schools or community centres educate and upskill children and adolescents about adaptive coping styles and observe if this has an impact on the prevalence of BD diagnosis. Examples of this would be to firstly screen for children or adolescents who may be considered at risk of BD (have a parent or family member with the diagnosis), interview them about the things they do to manage their stress or problem solve, and also enquire about familial coping strategies. If maladaptive coping styles are identified (such as rumination, avoidance, or denial), educate and facilitate implementation of more adaptive coping styles (e.g., seeking help from appropriate sources, coming up with a plan of action to solve the problem, plan ahead), then, at various time points over the years interview the same participants about their coping styles and affective symptoms. Furthermore, these findings do not need to be limited to those vulnerable to BD. Future research could attempt to engage anyone who employs maladaptive coping styles and investigate the impact of teaching adaptive coping styles on overall wellbeing and mental health, whilst also monitoring prevalence of other mental health disorders.

Finally, conceptualising vulnerability to BD as a two-dimensional trait was useful, as subtle, but noticeable, findings were observed between the two dimensions. Had a total vulnerability to BD score been used instead of separating the two models into depression-proneness and hypomania-proneness, the following findings would have been missed: that RS acted as a predictor of sGBI-Depression scores but not sGBI-Hypomania scores; that all vulnerability to BD scores (sGBI-Depression and sGBI-
Hypomania scores) were significantly correlated with mood and wellbeing outcome variables, except that between sGBI-Depression and PA scores; that Avoidant Coping scores predicted SWL in the context of sGBI-Depression scores, but not in the context of sGBI-Hypomania scores; that Avoidant Coping score was the sole predictor of PA in the context of other coping styles and sGBI-Depression scores, but sGBI-Hypomania scores along with Active Coping predicted PA; that Avoidant Coping was a partial mediator between sGBI-Depression scores and PSQI scores, but not between sGBI-Hypomania and PSQI scores; that PSQI acted as a partial mediator between sGBI-Hypomania scores and PA, but not between sGBI-Depression scores and PA; that Support Seeking scores acted as a partial mediator between sGBI-Hypomania scores and PA, but not between sGBI-Depression and PA scores; and that Active Coping acted as a partial mediator between sGBI-Hypomania scores and PA, but not between sGBI-Depression and PA scores. And, as previously noted in section 7.4.5, the following unique pathways would not have been observed: WU to Avoidant Coping (model with sGBI-Depression scores); Avoidant Coping to PSQI scores (model with sGBI-Depression scores); Avoidant Coping to SWL scores (model with sGBI-Depression scores); PSQI to NA scores (model with sGBI-Depression scores); GTB to PA scores (model with sGBI-Hypomania scores); FA to sGBI-Hypomania scores (model with sGBI-Hypomania scores); WU to sGBI-Hypomania scores (model with sGBI-Hypomania scores); Morningness to sGBI-Hypomania scores (model with sGBI-Hypomania scores); and sGBI-Hypomania to NA scores (model with sGBI-Hypomania scores). These findings show that investigating the two dimensions of BD as separate (but highly correlated) aids researchers in understanding the aetiology of BD and interpreting findings in a more complex way.


8.4 Limitations of Study

A number of limitations must be recognised. Firstly, the present study employed cross-sectional, self-report data. As a cross-sectional design was employed, causality cannot be inferred (Christensen & Kessing, 2006; Markarian et al., 2013). To further investigate the potential mediating effect of state sleep on coping and mood/wellbeing variables, future research could use physiological monitors of sleep, and monitor individuals' coping styles and mood in a longitudinal study. Polysomnography is the gold standard in measuring sleep, however, it can be quite laboursome for participants and researchers (Kaplan, Talbot, Gruber, & Harvey, 2012). A less invasive mode to monitor sleep is actigraphy and it has been found to be as reliable as polysomnography (Kaplan et al., 2012), and can be worn for long periods of time. Ultimately, the gold standard in understanding the effects of sleep and coping on individuals vulnerable to BD would be best carried out in a controlled environment over a lengthy period of time. However, as sleep disruptions have been found to be prodromes to manic or depressive episodes (Lam & Wong, 2005; Sierra et al., 2007), it would be unethical to ask participants to put themselves at risk. Furthermore asking individuals who are considered "low risk" to participate in such a design might not be representative of the interested sample (Eidelman, Talbot, Gruber, & Harvey, 2010), which in present project’s case was those vulnerable to BD.

Secondly, to address the complex relationships between variables (many of which were hypotheses tested for the first time here), a modelling approach was employed. As mentioned in the Method section, an ideal sample size, based on the number of parameters included in the present study, would have been at least 1,180 cases, based on the 20:1 N:q rule described by Kline (2011). It is recommended that future research employ a larger sample to confirm the BD, Coping, and Sleep models.
Additionally, the putative directions of paths were set as an organising assumption for the project, which has proven useful in generating novel insights. However, it is important to note that alternative models could have been investigated. The author imposed the direction of the arrows on to the BD, Coping, and Sleep models. Although they were positioned based mostly on published research, and some on theoretical assumptions, future research is welcome to modify the direction of the arrows and even the content of the model and to also test possible nested models. It was not tested if sGBI-Depression or sGBI-Hypomania scores predicted trait-like sleep variables. Preliminary analyses with the causality stemming from outcome variables to trait variables (arrows in the opposite direction in SEM analyses) were tested and no significant findings were identified. The model investigated by the present project was just one way that the variables included could have been positioned.

Thirdly, the scales that were employed may have influenced the findings. To address the first aim—to explore previously determined associations between personality traits, motivation traits, and vulnerability to BD—the present study employed the API. One of the most commonly used measures of the FFM, however, is the NEO-PI-R (Bagby et al., 1996; Barnett et al., 2011; Brieger, Ehrt, Roettig, & Marneros, 2002; Canuto et al., 2010; Faustino, 2012; Kim et al., 2011; Lozano & Johnson, 2001; Murray et al., 2007; Quilty et al., 2013); thus, replicating the current study with the inclusion of this measure could further validate the present findings. If the NEO-PI-R was used, researchers could analyse findings regarding personality at the aspect and facet level, as did Quilty et al. (2013), to see if Neuroticism facets (hostility, anxiety, depression, impulsivity, self-consciousness, or vulnerability) or aspects (volatility and withdrawal; DeYoung et al., 2007) impact vulnerability to BD.
Another measure that could have been employed to measure vulnerability to BD is the Hypomanic Personality Scale (Eckblad & Chapman, 1986), which has very recently been found to predict individuals at risk of a BD spectrum diagnosis (Johnson, Carver, Joormann, & Cuccaro, 2015; Walsh et al., 2015). Although data from the present study was collected between 2009 and 2010, this scale could be employed along with sGBI in future studies. Whilst on the topic of measures employed, it is important to note here that the sGBI and personality and BIS/BAS questionnaires were similar in structure, and it is possible that shared method variance impacted on the strength of correlations (Wu & Clark, 2003). Furthermore, at the time the writer was researching questionnaires that measured adult sleep, the ASWS (Fortunato et al., 2008) was the only one of its kind. Furthermore, the modified ASWS had unknown validity at the time and it is possible that this may have influenced the data. The study would have been further improved if it was possible to collect objective measures of sleep, such that produced by actigraphy.

Fourthly, some variables remained skewed after transformation. It should also be noted, however, that with large data sets, tests of normality can be significant even if distributions are only slightly different from a normal distribution (Tabachnick & Fidell, 2007). Furthermore, a common problem with social science research is that the vast majority of variables that social scientists wish to measure are not normally distributed (Pallant, 2007), making it common practice to introduce transformations only when absolutely necessary (Cohen et al., 2003).

A number of other limitations should also be noted. The sample was predominantly female and findings may not generalise to a more gender-balanced sample. Indeed there were insufficient males in the sample to conduct separate SEM analyses to explore gender effects. Participants were not asked about (and, thus, not
screened for) physical ailments, or medications which might have impacted findings. Error may have been increased as the questionnaire was completed online, rather than under controlled conditions (Markarian et al., 2013)—potentially participants may have been interrupted during completion, thus impacting their mood. A large proportion of the sample was undergraduate students, who, although may have been socio-demographically and culturally diverse, may not be representative of the general population (Alloy et al., 2012). With regard to the impact on the findings regarding coping and generalisation of findings, the effects of culture on coping have been questioned (Miyazaki, Bodenhorn, Zalaquett, & Ng, 2008); thus, future studies may wish to conduct a similar investigation but across different cultures to see if the interactions between variables are culture-specific. Furthermore, catch items (items designed to catch individuals who not paying attention and speeding through the questionnaire) were not used. Future studies are recommended to do so, like that of Giovanelli et al. (2013), so that non-genuine responses can be removed.

Finally, although dimensional approaches (as used here) are prominent characteristics of BD, there is undoubtedly important information captured in a categorical diagnosis of BD. It is useful to consider how future research could bridge the gap between the current project’s findings and research hypotheses or clinical recommendations for individuals carrying that diagnosis. Findings regarding the importance of sleep and the use of adaptive coping styles to improve mood and wellbeing outcomes from the present study can be applied to many individuals including: the general population; those who are at risk (specifically offspring, siblings, or unaffected relatives of BD); poor sleepers; individuals presenting with sub-clinical levels of BD; or those who employ mostly maladaptive coping styles. Although there are benefits to having cut-off points and minimum criteria required to be met in order to
receive a diagnosis (for clinical and research purposes), many individuals who are yet to receive a diagnosis miss out on treatment options that would otherwise benefit them.

8.5 Conclusion

The present study tested models that involved the interactions between vulnerability to BD (including either sGBI-Depression or sGBI-Hypomania scores), trait-like sleep, coping, state sleep, and mood/wellbeing variables, named the BD, Coping, and Sleep models. Analyses also investigated the interactions of personality and motivation trait variables, and found the factors of which both capture impulsivity—namely Neuroticism and BAS Fun Seeking—to be significant and consistent correlates of vulnerability to BD. Trait-like sleep variables were found to be significant predictors of sGBI-Depression and sGBI-Hypomania scores in the context of personality and motivation traits. Neuroticism and trait-like sleep variables could be employed as valid correlates of vulnerability to BD. The conceptual separation of sleep into state and trait-like was supported and so trait-like sleep was considered an important construct for future research into BD, and recommended to be included as a screening measure in individuals who may be prone to a BD diagnosis. Both state sleep and coping styles were found to act as partial mediators between trait-sleep variables, vulnerability to BD (sGBI-Depression and sGBI-Hypomania scores), and mood/wellbeing variables. A novel finding regarding the partial mediators was that state sleep influenced coping, but coping did not influence state sleep. The present study demonstrated that there are multiple factors to consider when working with someone who may be predisposed to a BD diagnosis, specifically sleep (both trait-like and state features) and coping styles. Ultimately, the present project showed that both state and trait-like sleep are important variables to consider when investigating mood and wellbeing outcomes, not just in the general population, but also in individuals who may
be at risk of a BD diagnosis at a later point in time. And finally, the present project emphasises the key role of coping styles and sleep quality in mood and mental health generally, and encourages clinicians to attend to these variables.
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Appendix A

Informed Consent Form

THE RELATIONSHIP BETWEEN BIPOLAR-VULNERABILITY, COPING AND OUTCOME

Simmone Poulios – Doctoral Candidate
Dr Greg Murray and Dr Ben Williams- Supervisors

The aim of the present study is to investigate a broad range of factors that may influence mood variability. Extremes in mood variability have been associated with mental disorders such as anxiety and depression. The present study is interested in the factors of sleep, personality, mood, behavioural inhibition and behavioural activation systems, quality of life, and vulnerability to mood variability.

Participation involves completing a series of questionnaires that will ask questions about your sleep, your personality, general behaviour and mood, your current mood, how you cope with stressful events, and your quality of life. Participation will take no more than 45 minutes. There are no right or wrong answers. To ensure that results remain valid, please answer each question honestly, and only if you speak English. Participation is also completely anonymous and confidential. If you would like to participate in the following research please be alert to any local or government restrictions involved in foreign research if you reside in a country other than Australia.

Completing the following questionnaire will be viewed as informed consent. Your participation is voluntary. If however you find any of the questions distressing or confronting, you may withdraw your participation at any time. If participation in this research causes any discomfort or distress please speak to your local general practitioner. Australian participants can also contact the Swinburne Psychology Clinic on (03) 9214 8653 (a low-cost service), Swinburne Counselling Services on: 03 9214 8025 (Hawthorn) or 03 9215 7101 (Lilydale), or Lifeline on 13 11 14. International participants can contact the Lifeline International 24 hour telephone counselling service, details regarding this service in your home country can be found by accessing their website: http://www.lifeline-international.org/looking_for_help
The completion of this research project is part of a Doctor of Clinical Psychology degree. Should results from the research be published, data will be published in group format, not examining individual responses, thus maintaining anonymity. If you have any general enquiries about this study, please contact:

Simmone Poulios
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This project has been approved by Swinburne’s Human Research Ethics Committee (SUHREC) in line with the National Statement on Ethical Conduct in Human Research. If you have any complaints or concerns about the conduct of this project, you can contact:

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Thank you for your participation, time, and assistance in this research.
Appendix B

Measurement of vulnerability to Bipolar Disorder: Psychometric properties of the short General Behaviour Inventory.

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Bipolar disorder (BD, also known as Manic-Depressive Illness) has a lifetime prevalence estimated at 4.4% (Merikangas et al., 2007). Suicide rates for those with BD are 27.6 times higher than the international base rate of suicide found in the general population (Tondo, Isacsson, & Baldessarini, 2003). Early detection and intervention are two very important factors in the treatment of BD. This leads to the vital task of developing tools that screen for symptoms of, or vulnerability to, BD, giving clinicians a chance to intervene before a full episode can occur (Lam & Wong, 2005). One way that clinicians can ascertain if someone is vulnerable to BD is by administering tools such as questionnaires. Questionnaires enable the collection of precise information in a short time frame.

Numerous scales measuring bipolar symptoms have been developed over the years. They come in different forms such as observer-rated, clinician-administered, interview, and self-report. For an in depth review of the different screening tools available for the detection of BD see Goodwin and Jamison (2007) or Livianos-Aldana and Rojo-Moreno (2001). The current project will focus on self-report measures. Typically, researchers investigating BD have developed scales that measure only manic or hypomanic states. An example of such a measure is the Mood Disorders Questionnaire (MDQ) (Hirschfeld et al., 2000). Specificity and sensitivity of the MDQ has been found to comparable to instruments that screen for different psychiatric disorders (Hirschfeld et al., 2000). The MDQ is quick to administer, comprised of 15 items, and is easy to score (Altman, 2004). It does contain an item that questions the participant if they have experienced symptoms at the same time, screening for a history of a manic episode.

A disadvantage in using tools that concentrate solely on manic or hypomanic symptoms is that individuals who exhibit high levels of depression but only very mild
symptoms of hypomania go undetected or even overlooked, particularly when analysing
vulnerability to BD. Self-report instruments that measure the two poles of BD, that is,
hypo/mania and depression, have an advantage over unipolar scales, such as those that
measure either depression or mania. Examples of these scales include the Internal State
Scale (ISS; Bauer et al., 1991), the Chinese Polarity Index (CPI; Zheng & Lin, 1994),
and the General Behavior Inventory (GBI; Depue et al., 1981). An advantage of these
scales over other self-report scales developed for BD is that they take into account the
bi-phasic nature of BD.

Research demonstrates that there is substantial genetic influence in BD as
heritability amongst twins has been found to be as high as 85% (McGuffin, et al., 2003).
First-degree relatives of individuals with BD have a 5 to 10 % risk of developing BD
(Maier, Höfgen, Zobel, & Rietschel, 2005). As BD has been found to be highly genetic,
taking one’s trait-vulnerability into consideration appears to be a more accurate way of
assessing one’s vulnerability to BD. Therefore, employing a scale that measures the
biphasic nature and trait-vulnerability of BD is highly desirable. There is one such scale
available, namely the General Behavior Inventory (GBI, Depue et al., 1981).

The GBI was primarily developed to identify individuals in the nonclinical
population who are at risk of bipolar or unipolar mood disorders (Depue, Krauss,
Spoont, & Arbisi, 1989). It has been found to have sufficient sensitivity, high
prognostic power and specificity, and does an adequate job of selecting individuals who
are at risk of affective disorders in a nonclinical population for the purpose of research
(Depue et al., 1989). The GBI captures the biphasic nature of BD as it is made up of
three different types of items: 19 hypomanic items, 46 depression items, eight biphasic
items, and when looking at 4 of the biphasic items, mood lability can also be analysed.
The GBI has a total of 73 items. It has been found to identify a range of symptomatic
intensities, from sub-syndromal to full-syndromal, and shows adequate sensitivity and high specificity (Depue et al., 1989).

In the world of research, response rate must also be considered, as the length of a questionnaire can affect positive or negative responses (Blount, Evans, Birch, Warren, & Norton, 2002). Scales already employed in bipolar research have been shortened for the sake of efficiency (Oliver & Simons, 2004). The aim of the present study was to compare the psychometric properties of the shortened version of the GBI with the full version of the GBI. This was completed in two stages; firstly, an exploratory factor analysis was performed to identify the highest loading items on the GBI, and then correlations with known external variables were analysed.

Study 1

Method

Participants

The sample used to identify the top 10 loading items for each of the factors of depression and hypo/mania consisted of 102 males and 377 females (5 missing values for gender). Participant age ranged from 16 to 86, with an average age of 29 years with a standard deviation of 12.83 years. Various avenues of recruitment were pursued, namely, internal University e-noticeboards, press releases in local newspapers, and mental health websites.

Measures

The data set contained scores obtained for the Mood Disorders Questionnaire (MDQ) (Hirschfeld et al., 2000), the Temperament Evaluation of Memphis, Pisa, Paris and San Diego-auto questionnaire version (TEMPS-A, Akiskal et al., 2005), the short version of the NEO-60 (Aluja, Garcia, Rossier, & Garcia, 2005) and the GBI (Depue et al., 1981).
**Mood Disorders Questionnaire (MDQ):** The MDQ is a self-report questionnaire employed to identify Bipolar Spectrum Disorders. The first 13 items screen for lifetime history of hypomanic and manic criteria employed by the Diagnostic and Statistic Manual of Mental Disorders (DSM-IV), respondents answer yes/no to these items. A further two items ask if symptoms checked occurred during the same time frame, and how much these symptoms affected their occupational, social and financial functioning.

**Temperament Evaluation of Memphis, Pisa, Paris and San Diego-auto questionnaire version (TEMPS-A):** The TEMPS-A is a self-report questionnaire comprised of 39 yes/no items. It is based on the five factors of temperament, measuring cyclothymic, dysthymic, anxious, hyperthymic and irritable. The TEMPS-A has been standardised in mood disorder populations, relatives of individuals with BD, and normal controls and has been found to have internal consistency and is psychometrically valid (Akiskal et al., 2005).

**NEO-60** is a self-report questionnaire employed to identify the five factors of personality, Neuroticism (N), Extraversion (E), Openness to Experience (O), Agreeableness (A) and Conscientiousness (C). There are 60 items and they are scored using a 5-point Likert-type scale ranging from strongly agree (4 points) to strongly disagree (0 points) (Aluha et al., 2005).

**GBI:** The GBI is a self-report scale used to measure vulnerability to BD. There are 73 items that are divided into three measures; they are depression, hypomania and biphasia. Items are scored using a 4-point Likert-type scale ranging from never/hardly ever (1 point) to very often/almost constantly (4 points).

**Procedure for the development of the sGBI**

An exploratory factor analysis was performed to identify the top twenty loading items for the depression, hypomaniac and biphasic items of the GBI. Statistical analyses
were carried out with SPSS Version 16.0 for Windows. For the final 20 items selected to constitute the sGBI see Appendix B.

Results

Principal component analysis was performed to identify the latent factor structure of the scales. The 73 GBI, 60 NEO-FFI, 13 MDQ, and 39 TEMPS-A items were submitted for analysis. The minimum correlation value for meaningful interpretation is 0.3 (Tabachnick & Fidell, 2007). Multiple inter-item correlations greater than 0.3 were identified in the initial analysis. The data also exhibited good factorability, as evidenced by a significant test of sphericity (Bartlett’s) and a prominent Kaiser-Meyer-Olkin, a measure of sampling adequacy, of 0.936.

There were 40 extractable factors identified by the un-rotated factor solution. These factors had eigenvalues greater than 1 and accounted for 68.49 % total item-score variance. The screen plot was consulted, as interpretation of factors was problematic. Further investigation indicated that a 2-factor explanation was most salient, explaining 30.59 % of total variance. An oblique rotation, Direct Oblimin, was then applied. The Direct Oblimin was selected as the ideal method because there is overlap between the two factors (mania and depression; see Depue et al., 1989) rendering orthagonality as unjustified.

The oblique rotation solution produced a sizeable amount of items loading on each of the two factors, that is, many items loaded above 0.32. Good contributors to factors are those with factor loadings greater than 0.55 (Comrey & Lee, 1992), and so only those that loaded more than 0.55 are shown here. Item composition of the rotated and extracted factor can be viewed in Table 1.

As can be seen in Table 1, the 2-factor solution differentiates the experience of depression (Factor 1) and the experience of hypo/mania (Factor 2). Factor 1 items are
predominated by GBI-depression items, accounting for 68.8 % of items loading above 0.55, with the remaining 31.3 % of Factor 1 loading items derived from the Neuroticism items of the NEO-FFI, low Extraversion scores from the NEO-FFI, and the Dysthymic items from the TEMPS-A. Again, the GBI predominated items loading on Factor 2. GBI items (11 Hypomania and 1 Biphasic) constituted 80 % of items loading on Factor 2, and the remaining 20 % from mania items on the MDQ.

Table 1

Percent Variance and Item Loadings for the 2-factor Solution post-Oblique Rotation

<table>
<thead>
<tr>
<th>Item</th>
<th>Factor 1</th>
<th>Factor 2</th>
<th>Label</th>
</tr>
</thead>
<tbody>
<tr>
<td>GBI34</td>
<td>.83</td>
<td>.00</td>
<td>Depression</td>
</tr>
<tr>
<td>GBI56</td>
<td>.82</td>
<td>.00</td>
<td>Depression</td>
</tr>
<tr>
<td>GBI16</td>
<td>.82</td>
<td>.00</td>
<td>Depression</td>
</tr>
<tr>
<td>GBI63</td>
<td>.81</td>
<td>.00</td>
<td>Depression</td>
</tr>
<tr>
<td>GBI32</td>
<td>.81</td>
<td>.00</td>
<td>Depression</td>
</tr>
<tr>
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<td>.00</td>
<td>Depression</td>
</tr>
<tr>
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<td>.78</td>
<td>.00</td>
<td>Depression</td>
</tr>
<tr>
<td>GBI23</td>
<td>.77</td>
<td>.00</td>
<td>Depression</td>
</tr>
<tr>
<td>NEO26</td>
<td>.77</td>
<td>.00</td>
<td>Neuroticism</td>
</tr>
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<td>Depression</td>
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<td>.00</td>
<td>Depression</td>
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<td>GBI72</td>
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<td>.00</td>
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<tr>
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Table 1 continued

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<th>Factor</th>
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<td>NEO56</td>
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<td>.00</td>
<td>Neuroticism</td>
</tr>
<tr>
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<td>.00</td>
<td>Dysthymic</td>
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<td>.00</td>
<td>Depression</td>
</tr>
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<td>.00</td>
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</tr>
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<td>.00</td>
<td>Neuroticism</td>
</tr>
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<td>Neuroticism</td>
</tr>
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<td>.00</td>
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</tr>
<tr>
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<td>.00</td>
<td>Extraversion</td>
</tr>
<tr>
<td>NEO17</td>
<td>-.57</td>
<td>.00</td>
<td>Extraversion</td>
</tr>
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<td>Hypomania</td>
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<td>Hypomania</td>
</tr>
<tr>
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<td>.63</td>
<td>Mania – Energy</td>
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<td>MDQ9</td>
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<tr>
<td>GBI43</td>
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<td>.56</td>
<td>Hypomania</td>
</tr>
<tr>
<td>% Variance</td>
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<td>11.53</td>
<td></td>
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<tr>
<td>Cumulative Variance</td>
<td>19.05</td>
<td>30.58</td>
<td></td>
</tr>
</tbody>
</table>

Note. Factor loadings above .55 are presented in the table.

The 10 highest loading items for each depression and hypomania/mania/biphasia were then re-labelled as the sGBI. Table 2 presents means, standard deviations, and measures of normality, skewness and kurtosis, for the sGBI and the full-GBI.
Table 2

*Statistical descriptives for total scales and subscale measures of the short- and full-GBI.*

<table>
<thead>
<tr>
<th></th>
<th>M</th>
<th>SD</th>
<th>K</th>
<th>S</th>
</tr>
</thead>
<tbody>
<tr>
<td>sGBI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>17.43</td>
<td>12.21</td>
<td>-0.52</td>
<td>.53</td>
</tr>
<tr>
<td>Depression</td>
<td>11.18</td>
<td>8.77</td>
<td>-0.66</td>
<td>.60</td>
</tr>
<tr>
<td>Hypo/mania</td>
<td>6.25</td>
<td>5.56</td>
<td>0.43</td>
<td>0.93</td>
</tr>
<tr>
<td>Full-GBI</td>
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<td></td>
</tr>
<tr>
<td>Total</td>
<td>137.95</td>
<td>42.42</td>
<td>-0.24</td>
<td>0.61</td>
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<tr>
<td>Depression</td>
<td>91.75</td>
<td>31.00</td>
<td>-0.34</td>
<td>0.64</td>
</tr>
<tr>
<td>Hypo/mania</td>
<td>46.20</td>
<td>14.32</td>
<td>0.62</td>
<td>0.92</td>
</tr>
</tbody>
</table>

*Note.* K = Kurtosis; S = Skewness

Skewness and kurtosis values for each subscale of the full-GBI and the sGBI were very similar.

Study 2

Method

Participants

Two different data sets were employed to identify if the sGBI produced equivocal correlations as the full-GBI. The first data set consisted of 175 participants. There were 124 females (70.9% of the sample) and 51 males (29.1% of the sample).

There were 92 females between 18 and 25 years; 12 females between 26 and 35 years; 7 females between 36 and 45 years; and 13 females between 46 and 55 years. There were 29 males aged between 18 and 25 years; 7 males between 26 and 35 years; 4 males between 36 and 45 years; 9 males between 46 and 55 years and 2 males 56 years and older.

The second data consisted of 343 participants. There were 269 females (78.4% of the sample) and 67 males (19.9% of the sample), 7 participants did not specify their gender. There were 236 females aged between 18 and 25 years; 21 females between 26 and 30 years; 6 females between 31 and 40 years; and 6 females over the age of 40
years. There were 47 males aged between 18 and 25 years; 12 males between 26 and 30 years; 5 males between 31 and 40 years; and 2 males over the age of 40 years.

Measures

The first data set employed the Positive and Negative Affect Schedules (PANAS; Watson, Clark, & Tellegen, 1988) and the Revised NEO-Personality Inventory (NEO-PI-R; Costa & McCrae, 1992). The second data set employed the PA scale of the PANAS, the Mood Survey (Underwood & Froming, 1980), and the Affective Lability Scale-Short form (ALS-SF; Oliver & Simons, 2004). Both sets of data had also employed the GBI.

**PANAS:** The PANAS is a 20-item scale, comprised of a 10-item Positive Affect (PA) scale and a 10-item Negative Affect (NA) scale. Respondents are asked to what extent they feel a particular affect using a 5-point scale ranging from *very slightly or not at all* to *extremely*. It has been found to be internally consistent, and stable over time (Watson, et al., 1988).

**Revised NEO-Personality Inventory:** The Revised NEO-Personality Inventory (NEO-PI-R; Costa & McCrae, 1992) is a 240 item measure of the five-factor model of personality, these being *Neuroticism* (N), *Extraversion* (E), *Openness to experience* (O), *Agreeableness* (A) and *Conscientiousness* (C). Items are scored using a five-point Likert scale, ranging from *strongly disagree* to *strongly agree*. The NEO-PI-R has been found to be a reliable and valid measure of personality (McCrae & Costa, 2007).

**Mood Survey:** The reactivity subscale of the Mood Survey (MS; Underwood & Froming, 1980) was employed. The reactivity subscale has been found to measure an individual’s reactivity to experience of moods, that is the intensity and frequency by which they experience mood on the happy-sad continuum. It was originally designed as a trait measure of mood, however, the items were reworded so that they measured mood.
over the past week. Of the eight items, two items were excluded as reliability has been found to be improved when done so (Murray, 2003). The six items were scored using a six-point Likert scale ranging from *strongly disagree* to *strongly agree*.

*Affective-Lability Scale-short form:* The Affective Lability Scale- short form (ALS-SF; Oliver & Simons, 2004) is an 18-item measure of affect lability. Affect lability is best described as the range, speed and frequency of change in one's affective state (Oliver & Simons, 2004). Items were reworded from their trait form to a format that asked about *state* affect lability *over the past week*, for example, the first item was reworded from “*At times I feel just as relaxed as everyone else and then within minutes I become so nervous that I feel light-headed and dizzy*” to “*At times I would feel just as relaxed as everyone else and then within minutes I would become so nervous that I felt light-headed and dizzy*”. Items were scored using a four-point frequency scale, ranging from *very undescriptive* to *very descriptive*.

*Procedure*

Once the highest loading items for both depression and hypo/mania were identified, correlations were carried out between external variable and the sGBI and external variables and the full-GBI using the two data sets. These correlations were then compared using Steiger’s statistic (1980). Internal consistency of the sGBI was measured using Cronbach’s alpha.

*Results*

The table below presents the correlations between external variables and Total-, Mania-, and Depression-scores for both the full-GBI and the short-.
Table 3

Correlations between Total, Mania, and Depression subscale scores of both the full-GBI and the sGBI and external variables

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<th>Mania</th>
<th>Depression</th>
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<td>sGBI</td>
<td>GBI</td>
</tr>
<tr>
<td>DATA SET 1</td>
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<tr>
<td>Positive Affect</td>
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<tr>
<td>Neuroticism</td>
<td>0.664</td>
<td>0.634</td>
<td>0.495</td>
</tr>
<tr>
<td>Extraversion</td>
<td>-0.083</td>
<td>-0.048</td>
<td>0.075</td>
</tr>
<tr>
<td>Openness</td>
<td>0.124</td>
<td>0.120</td>
<td>0.117</td>
</tr>
<tr>
<td>Agreeableness</td>
<td>-0.292</td>
<td>-0.270</td>
<td>-0.347</td>
</tr>
<tr>
<td>Conscientiousness</td>
<td>-0.397</td>
<td>-0.401</td>
<td>-0.320</td>
</tr>
<tr>
<td>DATA SET 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive Affect</td>
<td>-0.232</td>
<td>-0.195</td>
<td>-0.033</td>
</tr>
<tr>
<td>Mood Survey</td>
<td>0.553</td>
<td>0.512</td>
<td>0.451</td>
</tr>
<tr>
<td>Altman Lability Scale</td>
<td>0.698</td>
<td>0.658</td>
<td>0.664</td>
</tr>
</tbody>
</table>

As can be seen in Table 3 the $r$ values of the sGBI and external variables and the $r$ values of the full-GBI and external variables were very similar.

Statistical analyses were then performed to identify if the correlations achieved by the sGBI were not significantly different to the correlations of the full-GBI. This was done using Steiger’s statistic (1980), and z-scores can be viewed in Table 4.
Table 4

*Steiger’s statistic, comparing the correlations between external variables and full-GBI and sGBI.*

<table>
<thead>
<tr>
<th></th>
<th>Z score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DATA SET 1</strong></td>
<td></td>
</tr>
<tr>
<td>Positive Affect</td>
<td>-0.812</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>0.8696</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>1.410</td>
</tr>
<tr>
<td>Extraversion</td>
<td>-1.236</td>
</tr>
<tr>
<td>Openness</td>
<td>0.142</td>
</tr>
<tr>
<td>Agreeableness</td>
<td>-0.810</td>
</tr>
<tr>
<td>Conscientiousness</td>
<td>0.155</td>
</tr>
<tr>
<td><strong>DATA SET 2</strong></td>
<td></td>
</tr>
<tr>
<td>Positive Affect</td>
<td>2.411*</td>
</tr>
<tr>
<td>Mood Survey</td>
<td>3.078*</td>
</tr>
<tr>
<td>Altman Lability Scale</td>
<td>-3.466*</td>
</tr>
</tbody>
</table>

*indicates significant difference between the correlations.

The sGBI was found to have good internal consistency, Cronbach’s alpha coefficient of 0.93.

Discussion

The aim of the present study was to compare the psychometric properties of the sGBI to the full-GBI with external known correlates. This was done in two stages. Firstly, an exploratory factor analysis was performed to identify the top twenty loading items for the depression, hypomanic and biphasic items of the GBI. The top ten loading items for each depression and hypomania/mania items of the GBI were then taken to constitute the sGBI.

The second stage of analysis compared the correlations produced by the full-GBI to known external correlates and the correlations between the sGBI and the same known external variables. Statistical analyses found that for the first data set, correlations produced by the sGBI were almost identical to those produced by the full-
GBI. Statistical analysis comparing correlations found no statistically significant difference between the correlations produced by the sGBI and those of the full-GBI. The second data set \((n = 343)\) employed in the second study was considerably larger than the first data set \((n = 175)\). Again correlations between the external variables and either the sGBI of the full-GBI were similar. However, when comparing the correlations it was found that there was statistical difference between the correlations produced by the sGBI to those of the full-GBI. The statistical significance produced by the larger data set when comparing correlations could be due to the size of the data set rather than the correlation values themselves (Pallant, 2007).

There were a few limitations to the present study. The present study was a confirmatory investigation of the sGBI to external correlates only. Also, each of the data sets had employed different measures and so the external correlates of BD vulnerability could not be compared across the two data sets. The effect of the sample size in the second data set also proved to be problematic as it unknown if this was due to the large sample size of the data set, or if in fact there was a difference in the correlations comparing the short- and full-GBI with external variables.

Further investigations confirming internal correlates need to be carried out, to ensure that the sGBI is a psychometrically sound measure that does an equivocal job to the full-GBI. Also, like Depue et al., future research should also conduct analyses that will determine if specific mood disorders can be identified from the sGBI. To demonstrate consistency, another suggestion for future research is to test the sGBI against other external variables such as temperament and personality, across different data sets that have employed the same measures.

The GBI is a widely employed screening tool used to identify individuals at risk of BD (Depue et al., 1989). As the full-GBI does take some time to administer and may
be somewhat repetitive to the individual completing it, the authors encourage the use of the sGBI. The sGBI has been found to be internally consistent, and correlates well with the known external correlates of BD. This paper by no means suggests that clinicians or researchers should stop using the full-GBI; merely that researchers and clinicians now have a quicker way to identify individuals vulnerable to BD.
References


Merikangas, K. R., Akiskal, H. S., Angst, J., Greenberg, P. E., Hirschfeld, R. M. A.,


### Appendix C

The sGBI

Here are some questions about behaviours that occur in the general population. Think about how often they occur for you. Using the scale below, select the response that best describes how often you experience these behaviours.

<table>
<thead>
<tr>
<th>Item</th>
<th>Question</th>
<th>Never or hardly ever</th>
<th>Sometimes</th>
<th>Often</th>
<th>Very often or almost constantly</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Have you experienced periods of several days or more when, although you were feeling unusually happy and intensely energetic (clearly more than your usual self), you also were physically restless, unable to sit still, and had to keep moving or jumping from one activity to another?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Have there been periods of several days or more when your friends or family told you that you seemed unusually happy or high, clearly different from your usual self or from a typical good mood?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Have there been times of several days or more when you did not feel the need for sleep and were able to stay awake and alert for much longer than usual because you were full of energy?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Have you had long periods in which you felt you couldn’t enjoy life as easily as other people?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Have you had periods of extreme happiness and intense energy lasting several days or more when you also felt much more anxious or tense (jittery, nervous, uptight) than usual (other than related to the menstrual cycle)?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Have there been times of several days or more when you were so sad that it was quite painful or you felt that you couldn’t stand it?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>7</strong></td>
<td>Have there been times lasting several days or more when you felt you must have lots of excitement, and you actually did a lot of new or different things?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>8</strong></td>
<td>Have you had periods of extreme happiness and intense energy (clearly) more than your usual self) when, for several days or more, it took you over an hour to get to sleep at night?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>9</strong></td>
<td>Have there been times when you looked back over your life and could see only failures or hardships?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>10</strong></td>
<td>Have there been long periods in your life when you flat sad, depressed, or irritable most of the time?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>11</strong></td>
<td>Have you had periods of extreme happiness and high energy lasting several days or more when what you saw, heard, smelled, tasted, or touched seemed vivid or intense?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>12</strong></td>
<td>Have there been times of several days or more when you were so down that nothing (not even friends or good news) could cheer you up?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>13</strong></td>
<td>Have there been times of a couple days or more when you felt that you were a very important person or that your abilities or talents were better than most other people’s?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>14</strong></td>
<td>Have there been times when you have hated yourself or felt that you were stupid, ugly, unlovable, or useless?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>15</strong></td>
<td>Have you had periods lasting several days or more when you flat depressed or irritable, and then other periods of several days or more when you felt extremely high, elated, and overflowing with energy?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>16</strong></td>
<td>Have there been periods when, although you were feeling unusually happy and intensely energetic, almost everything got on your nerves and made you irritable or angry (other than related to the menstrual cycle)?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Description</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>--------------------------------------------------------------------------------------------------------</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>Have there been times of several days or more when you really got down on yourself and felt worthless?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>Have you had periods when it seemed that the future was hopeless and things could not improve?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>Have there been periods lasting several days or more when you were so down in the dumps that you thought you might never snap out of it?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>Have there been times when you have felt that you would be better off dead?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
# Appendix D

The Composite Scale of Morningness (Smith, Reilly & Midkiff, 1989)

Please check the response for each item that best describes you.

1. Considering only your own “feeling best” rhythm, at what time would you get up if you were entirely free to plan your day?

<table>
<thead>
<tr>
<th>Time</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>5:00-6:30 a.m.</td>
<td></td>
</tr>
<tr>
<td>6:30-7:45 a.m.</td>
<td></td>
</tr>
<tr>
<td>7:45-9:45 a.m.</td>
<td></td>
</tr>
<tr>
<td>9:45-11:00 a.m.</td>
<td></td>
</tr>
<tr>
<td>11:00 a.m.- 12:00 (noon)</td>
<td></td>
</tr>
</tbody>
</table>

2. Considering your only “feeling best” rhythm, at what time would you go to bed if you were entirely free to plan your day?

<table>
<thead>
<tr>
<th>Time</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>8:00-9:00 p.m.</td>
<td></td>
</tr>
<tr>
<td>9:00-10:15 p.m.</td>
<td></td>
</tr>
<tr>
<td>10:15-12:30 a.m.</td>
<td></td>
</tr>
<tr>
<td>12:30-1:45 a.m.</td>
<td></td>
</tr>
<tr>
<td>1:45-3:00 a.m.</td>
<td></td>
</tr>
</tbody>
</table>

3. Assuming normal circumstance, how easy do you find getting up in the morning? (Check one.)

<table>
<thead>
<tr>
<th>Difficulty</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Not at all easy</td>
<td></td>
</tr>
<tr>
<td>Slightly easy</td>
<td></td>
</tr>
<tr>
<td>Fairly easy</td>
<td></td>
</tr>
<tr>
<td>Very easy</td>
<td></td>
</tr>
</tbody>
</table>

4. How alert do you feel during the first half hour after having awakened in the morning? (Check one)

<table>
<thead>
<tr>
<th>Alertness</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Not at all alert</td>
<td></td>
</tr>
<tr>
<td>Slightly alert</td>
<td></td>
</tr>
<tr>
<td>Fairly alert</td>
<td></td>
</tr>
<tr>
<td>Very alert</td>
<td></td>
</tr>
</tbody>
</table>

5. During the first half hour after having awakened in the morning, how tired do you feel? (Check one)
Very tired    __
Fairly tired    __
Fairly refreshed __
Very refreshed  __

6. You have decided to engage in some physical exercise. A friend suggests that you do this one hour twice a week and the best time for him is 7:00-8:00 a.m. bearing in mind nothing else but your own “feeling best” rhythm, how do you think you would perform?

Would be in good form __
Would be in reasonable form __
Would find it difficult   __
Would find it very difficult __

7. At what time in the evening do you feel tired and, as a result, in need of sleep?

8:00-9:00 p.m. __
9:00-10:15 p.m. __
10:15-12:30 a.m. __
12:30-1:45 a.m. __
1:45-3:00 a.m. __

8. You wish to be at your peak performance for a test which you know is going to be mentally exhausting and lasting for two hours. You are entirely free to plan your day, and considering only your own “feeling best” rhythm, which ONE of the four testing times would you choose?

8:00-10:00 a.m. __
11:00 a.m.-1:00 p.m. __
3:00-5:00 p.m. __
7:00-9:00 p.m. __

9. One hears about “morning” and “evening” types of people. Which ONE of these types do you consider yourself to be?

Definitely a morning type __
More a morning than and evening type __
More an evening than a morning type __
Definitely an evening type __
10. When would you prefer to rise (provided you have a full day’s work—8 hours) if you were totally free to arrange your time?

- Before 6:30 a.m. __
- 6:30-7:30 a.m. __
- 7:30-8:30 a.m. __
- 8:30 a.m. or later __

11. If you always had to rise at 6:00 a.m., what do you think it would be like?

- Very difficult and unpleasant __
- Rather difficult and unpleasant __
- A little unpleasant but no great problem __
- Easy and not unpleasant __

12. How long a time does it usually take before you “recover your senses” in the morning after rising from a night’s sleep?

- 0-10 minutes __
- 11-20 minutes __
- 21-40 minutes __
- More than 40 minutes __

13. Please indicate to what extent you are a morning or evening active individual.

- Pronounced morning active (morning alert and evening tired) __
- To some extent, morning active __
- To some extent, evening active __
- Pronounced evening active (morning tired and evening alert) __
### Appendix E

The Adult Sleep Wake Scale (Fortunato, LeBourgeois, & Harsh, 2008)

**Modified Version**

Using the choices below select *how often* the following things have happened *for most of your life.*

- **Never** - has not happened
- **Once in a while**- happened 20% of the time
- **Sometimes**- happened 40% of the time
- **Quite often**- happened 60% of the time
- **Frequently, if not always**- happened 80% of the time
- **Always**- happened 100% of the time

Questions 1 – 5 are only about you

**Going to Bed** at bedtime

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>O</th>
<th>S</th>
<th>Q</th>
<th>F</th>
<th>A</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. …I want to stay up and do other things (for example: read, work, or watch</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
In general...

2. …I have to make myself go to bed.  
3. …It is very hard for me to go to bed on time.  
4. …I “put off” or delay going to bed.  
5. How long do you usually “put off” or delay going to bed?  
   (a) < 15 min  (b) 15-30 min  (c) 30-45 min  (d) 45-60 min  (e) 60-90 min  (f) > 90 min

Remember: Think about most of your life.

Questions 6 - 10 are only about you

Falling Asleep after “lights-out”

When I’m in bed and it is time to fall asleep...

6. …I am not sleepy.  
7. …I am unable to settle down.

In general...

8. …I try to make myself go to sleep.  
9. …I fall asleep quickly.
10. How long does it *usually* take you to fall asleep after “lights out”?

(a) < 15 min (b) 15-30 min (c) 30-45 min (d) 45-60 min (e) 60-90 min (f) > 90 min

**Questions 11 - 15 are only about how you *Sleep* during the night**

(someone else could have told you these things)

---

**After I fall asleep, but during the night…**

11. …I toss and turn in bed.  
12. …I am very restless.  
13. …I awaken more than once.

**In general…**

14. …I sleep without arousals or awakenings.

15. How often do you *usually* wake up during the night?

(a) Never (b) Once (c) twice (d) 3 times (e) 4 times (f) more than 4 times

**Remember: Think about most of your life.**

Questions 16 – 20 are *only* about you

*Going back to sleep* after waking up during the night

<table>
<thead>
<tr>
<th>Always</th>
<th>Frequently, if not Always</th>
<th>Quite Often</th>
<th>Sometimes</th>
<th>Once in Awhile</th>
<th>Never</th>
</tr>
</thead>
<tbody>
<tr>
<td>100 %</td>
<td>80 %</td>
<td>60 %</td>
<td>40 %</td>
<td>20 %</td>
<td>0 %</td>
</tr>
</tbody>
</table>

**After waking up during the night…**

16. …I have a hard time going back to sleep.
17. …I drift back off to sleep easily.  
18. …I am calm and relaxed.  
19. …I roll over and go right back to sleep.  
20. How long does it usually take you to go back to sleep after waking during the night?  
   (a) < 5 min  (b) 5-10 min  (c) 10-15 min  (d) 15-20 min  (e) 20-30 min  (f) > 30 min  

Questions 21 – 25 are only about you

Waking Up in the morning

In the morning, I wake up…

21. …and feel ready to get up for the day.  
22. …rested and alert.  
23. …and just can’t get going.  

In general…

24. …I am slow-to-start in the morning.  
25. …I find it difficult to get out of the bed in the morning.
Appendix F

The Australian Personality Inventory (Murray et al., 2009)

For the statements below please indicate how accurate these statements are about yourself, using 1 = Very accurate, 2 = Moderately inaccurate, 3 = Neither inaccurate nor accurate, 4 = Moderately accurate, and 5 = Very accurate.

<table>
<thead>
<tr>
<th></th>
<th>Statement</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Often feel blue.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Feel comfortable around people.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Do not like art.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Have a good word for everyone.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Am always prepared.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Dislike myself.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Make friends easily.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Have a vivid imagination.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Believe that others have good intentions.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Pay attention to details.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Am often down in the dumps.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>Am skilled in handling social situations.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Have a rich vocabulary.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>Respect others.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>Get chores done right away.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>Have frequent mood swings.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>Am the life of the party.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>Carry the conversation to a higher level.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>Accept people as they are.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>Carry out my plans.</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>21</td>
<td>Panic easily.</td>
<td></td>
<td></td>
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<tr>
<td>22</td>
<td>Know how to captivate people.</td>
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<tr>
<td>23</td>
<td>Enjoy hearing new ideas.</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>24</td>
<td>Make people feel at ease.</td>
<td></td>
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<tr>
<td>25</td>
<td>Make plans and stick to them.</td>
<td></td>
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<tr>
<td>26</td>
<td>Seldom feel blue.</td>
<td></td>
<td></td>
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<tr>
<td>27</td>
<td>Have little to say.</td>
<td></td>
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<tr>
<td>28</td>
<td>Am not interested in abstract ideas.</td>
<td></td>
<td></td>
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<tr>
<td>29</td>
<td>Have a sharp tongue.</td>
<td></td>
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<tr>
<td>30</td>
<td>Waste my time.</td>
<td></td>
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<tr>
<td>31</td>
<td>Feel comfortable with myself.</td>
<td></td>
<td></td>
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<tr>
<td>32</td>
<td>Keep in the background.</td>
<td></td>
<td></td>
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<tr>
<td>33</td>
<td>Enjoy wild flights of fantasy.</td>
<td></td>
<td></td>
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<tr>
<td>34</td>
<td>Cut others to pieces.</td>
<td></td>
<td></td>
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<tr>
<td>35</td>
<td>Find it difficult to get down to work.</td>
<td></td>
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<tr>
<td>36</td>
<td>Rarely get irritated.</td>
<td></td>
<td></td>
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<tr>
<td>37</td>
<td>Would describe my experiences as somewhat dull.</td>
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<tr>
<td>38</td>
<td>Avoid philosophical discussions.</td>
<td></td>
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<tr>
<td>39</td>
<td>Suspect hidden motives in others.</td>
<td></td>
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<tr>
<td>40</td>
<td>Do just enough work to get by.</td>
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<tr>
<td>41</td>
<td>Am not easily bothered by things.</td>
<td></td>
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<tr>
<td>42</td>
<td>Don’t like to draw attention to myself.</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>43</td>
<td>Do not enjoy going to art museums.</td>
<td></td>
<td></td>
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<tr>
<td>44</td>
<td>Get back at others.</td>
<td></td>
<td></td>
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<tr>
<td>45</td>
<td>Don’t see things through.</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>46</td>
<td>Am very pleased with myself.</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>47</td>
<td>Don’t talk a lot.</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>48</td>
<td>Rarely look for a deeper meaning in things.</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>49</td>
<td>Insult people.</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>50</td>
<td>Shirk my duties</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>
Appendix G

The BIS/BAS scale (Carver & White, 1994)

The following questions ask about various aspects of your behavior. Please indicate the extent to which you agree or disagree with each question. Work quickly and remember that there is no right or wrong answer. Please be as honest as possible.

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. If I think something unpleasant is going to happen I usually get pretty &quot;worked up&quot;</td>
<td>Strongly Disagree</td>
<td>Disagree</td>
<td>Agree</td>
<td>Strongly Agree</td>
</tr>
<tr>
<td>2. When I get something I want, I feel excited and energised</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3. I often act on the spur of the moment</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4. Criticism or scolding hurts me quite a bit</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5. When good things happen to me it affects me strongly</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6. I go out of my way to get things I want</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7. I have very few fears compared to my friends.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8. I'm always willing to try something new if I think it will be fun</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9. I crave excitement and new sensations</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10. Even if something bad is about to happen</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
happen to me, I rarely experience fear or nervousness

<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>11. When I want something, I usually go all-out to get it.</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>12. When I'm doing well at something, I love to keep at it</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>13. It would excite me to win a contest</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>14. I worry about making mistakes</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>15. I feel pretty worried or upset when I think or know somebody is angry at me</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>16. When I see an opportunity for something I like, I get excited right away</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>17. I feel worried when I think I have done poorly at something</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>18. When I go after something I use a &quot;no holds barred&quot; approach</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>19. If I see a chance to get something I want I move on it right away</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>20. I will often do things for no other reason than that they might be fun</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
Appendix H

The Brief COPE (Carver, 1997)

We are interested in how people respond when they confront difficult or stressful events in their lives. There are lots of ways to try to deal with stress. This questionnaire asks you to indicate what you generally do and feel, when you experience stressful events. Obviously, different events bring out somewhat different responses, but think about what you usually do when you are under a lot of stress.

Indicate what YOU usually do when YOU experience a stressful event.

1 = I usually don't do this at all
2 = I usually do this a little bit
3 = I usually do this a medium amount
4 = I usually do this a lot

1. I turn to work or other activities to take my mind off things.  
2. I concentrating my efforts on doing something about the situation I'm in.  
3. I say to myself "this isn't real.".  
4. I use alcohol or other drugs to make myself feel better.  
5. I get emotional support from others.  
6. I give up trying to deal with it.  
7. I take action to try to make the situation better.  
8. I refuse to believe that it has happened.
9. I say things to let my unpleasant feelings escape. 1 2 3 4

10. I get help and advice from other people. 1 2 3 4

11. I use alcohol or other drugs to help me get through it. 1 2 3 4

12. I try to see it in a different light, to make it seem more positive. 1 2 3 4

13. I criticise myself. 1 2 3 4

14. I try to come up with a strategy about what to do. 1 2 3 4

15. I get comfort and understanding from someone. 1 2 3 4

16. I give up the attempt to cope. 1 2 3 4

17. I look for something good in what is happening. 1 2 3 4

18. I make jokes about it. 1 2 3 4

19. I do something to think about it less, such as going to movies, watch TV, read, daydream, sleep, or shop. 1 2 3 4

20. I accept the reality of the fact that it has happened. 1 2 3 4

21. I express my negative feelings. 1 2 3 4
22. I try to find comfort in my religion or spiritual beliefs.

23. I try to get advice or help from other people about what to do.

24. I learn to live with it.

25. I think hard about what steps to take.

26. I blame myself for things that happened.

27. I pray or meditate.

28. I make fun of the situation.
Appendix I

Measurement Model of Three Coping Factors Employed

The original Brief COPE is comprised of 28-items, measuring 14 different types of coping styles by two items each. The data file was split and an exploratory factor analysis was run to see if subscales could be grouped into more general coping styles. Previous research has extracted either three five factors (Kimemias, Asner-Self, & Daire, 2011), seven factors (Miyazaki et al., 2008), eight factors (Kapsou et al., 2010), and nine factors (Snell, Siegert, Hay-Smith, & Surgenor, 2011). Both Snell et al., (2011) and Miyazaki et al. (2008) argued that despite seven or nine factors being identified by exploratory factor analyses, three factors were more meaningful (Snell et al., 2011) and that 18 of the 28 items made up the three factors (Miyazaki et al., 2008). These three scales were called approach coping, avoidant coping and help-Seeking coping (Snell et al., 2011) or positive coping, denial or support seeking (Miyazaki et al., 2008).

The principal components method was chosen as the extraction technique, and Direct Oblimin as the rotation technique. Interpretation of the output revealed that Kaiser-Meyer Olkin Measure of Sampling Adequacy was .77 (greater than .6, thus suitable for factor analysis) and Bartlett's Test of Sphericity was significant ($p = .<.001$), again indicating that the sample was suitable for factor analysis.

Examination of the Total Variance Explained table indicated that there were 8 factors with eigenvalues greater than 1, explaining a cumulative percentage of 67% of the variance. However, the scree plot 'elbow' appeared to be between factors 4 and 9. Examination of the Component Matrix indicated that most items loaded strongly on eight components. The Pattern Matrix showed that three components only consisted of two items, however as it is ideal for a factor to have three or more items (Pallant, 2007),
they were not considered. The first three components had loadings from at least five or more items. The first three components explained 39.7% of the variance. As previous studies also agreed on three factors, it was decided that three factors of coping styles that would be investigated.

The three factors were then analysed by confirmatory factor analysis using Amos (Arbuckle, 2012). The maximum likelihood method of estimation was applied. The final model and items included can be seen on the next page. The three factors were named Active Coping, Support Seeking, and Avoidant Coping.
All indices analysed to assess the goodness of fit of the above model all indicated good fit. Chi-square was 49.69 based on 36 degrees of freedom ($p = .06$, chi-square/df = 1.38). Fit statistics for the above model can be seen in the table below.

<table>
<thead>
<tr>
<th>Goodness of Fit</th>
<th>GFI</th>
<th>.986*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adjusted Goodness of Fit</td>
<td>AGFI</td>
<td>.974*</td>
</tr>
<tr>
<td>Root mean-square of error of approximation</td>
<td>RMSEA</td>
<td>.025*</td>
</tr>
<tr>
<td></td>
<td>PCLOSE</td>
<td>.997*</td>
</tr>
<tr>
<td></td>
<td>LO 90</td>
<td>.000*</td>
</tr>
<tr>
<td>Tucker-Lewis Index</td>
<td>TLI</td>
<td>.992*</td>
</tr>
</tbody>
</table>

Note. * = good fit as described by Kline (2011).
Appendix J

The Pittsburgh Sleep Quality Index (Buysse et al., 1989)

The following questions relate to your usual sleep habits during the past month only.

Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

1. During the past month, what time have you usually gone to bed at night?
   BED TIME_____________

2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?
   NUMBER OF MINUTES ______________

3. During the past month, what time have you usually gotten up in the morning?
   GETTING UP TIME_____________________

4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.)
   HOURS OF SLEEP PER NIGHT________________

For each of the remaining questions, check the one best response. Please answer all questions.

5. During the past month, how often have you had trouble sleeping because you…
   a) Cannot get to sleep within 30 minutes
      Not during the past _______ Less than a week____ Once or twice a week____ Three or more times a week __
   b) Wake up in the middle of the night or early morning
      Not during the past _______ Less than a week____ Once or twice a week____ Three or more times a week __
c) Have to get up to use the bathroom
Not during the past___ Less than a week___ Once or twice a week___ Three or more times a week __

d) Cannot breathe comfortably
Not during the past___ Less than a week___ Once or twice a week___ Three or more times a week __

e) Cough or snore loudly
Not during the past___ Less than a week___ Once or twice a week___ Three or more times a week __

f) Feel too cold
Not during the past___ Less than a week___ Once or twice a week___ Three or more times a week __

g) Feel too hot
Not during the past___ Less than a week___ Once or twice a week___ Three or more times a week __

h) Had bad dreams
Not during the past___ Less than a week___ Once or twice a week___ Three or more times a week __

i) Have pain
Not during the past___ Less than a week___ Once or twice a week___ Three or more times a week __

j) Other reason(s), please describe____________________________________________________
____________________________________________________________________________________

How often during the past month have you had trouble sleeping because of this?
Not during the past___ Less than a week___ Once or twice a week___ Three or more times a week __

6. During the past month, how would you rate your sleep quality overall?
Very good _____
Fairly good ___
Fairly bad ___
Very bad ___

7. During the past month, how often have you taken medicine to help you sleep (prescribed or ‘over the counter’)?

Not during the past _______ Less than a week___ Once or twice a week___ Three or more times a week ___

8. During the past month, how often have you had trouble staying awake while, driving, eating meals, or engaging in social activity?

Not during the past _______ Less than a week___ Once or twice a week___ Three or more times a week ___

9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

No problem at all _______
Only a very slight problem _______
Somewhat of a problem _______
A very big problem _______

10. Do you have a bed partner or room mate?

No bed partner or room mate _______
Partner/room mate in other room _______
Partner in same room, but not same bed _______
Partner in same bed _______

If you have a roommate or bed partner, ask him/her how often in the past month you have had…

a) Loud snoring

Not during the past _______ Less than a week___ Once or twice a week___ Three or more times a week ___

b) Long pauses between breaths while asleep

Not during the past _______ Less than a week___ Once or twice a week___ Three or more times a week ___
c) Legs twitching or jerking while you sleep

Not during the past  Less than a week  Once or twice a week  Three or more times a week

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<tbody>
<tr>
<td>Not during the past</td>
<td>Less than a week</td>
<td>Once or twice a week</td>
<td>Three or more times a week</td>
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<tr>
<td>month</td>
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d) Episodes of disorientation or confusion during sleep

Not during the past  Less than a week  Once or twice a week  Three or more times a week

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<tbody>
<tr>
<td>Not during the past</td>
<td>Less than a week</td>
<td>Once or twice a week</td>
<td>Three or more times a week</td>
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<tr>
<td>month</td>
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e) Other restlessness while you sleep; please describe ________________________________

Not during the past  Less than a week  Once or twice a week  Three or more times a week

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<tbody>
<tr>
<td>Not during the past</td>
<td>Less than a week</td>
<td>Once or twice a week</td>
<td>Three or more times a week</td>
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<tr>
<td>month</td>
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Appendix K

The PANAS (Watson et al., 1988)

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

1 2 3 4 5
Very slightly or A little Moderately Quite a bit Extremely not at all

___ interested ___ irritable
___ distressed ___ alert
___ excited ___ ashamed
___ upset ___ inspired
___ strong ___ nervous
___ guilty ___ determined
___ scared ___ attentive
___ hostile ___ jittery
___ enthusiastic ___ active
___ proud ___ afraid
## Appendix L

The Satisfaction with Life Scale (Diener et al., 1985)

The following set of questions asks about your general levels of life-satisfaction. For each of the following, please indicate how you typically feel by selecting the relevant number.

<table>
<thead>
<tr>
<th></th>
<th>Strongly disagree</th>
<th>Disagree</th>
<th>Slightly disagree</th>
<th>Neither agree nor disagree</th>
<th>Slightly agree</th>
<th>Agree</th>
<th>Strongly agree</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>In most ways, my life is close to my ideal</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>2</td>
<td>The conditions of my life are excellent</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>3</td>
<td>I am satisfied with my life</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>4</td>
<td>So far I have got the important things I want in my life</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>5</td>
<td>If I could live my life over, I would change almost nothing</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>
Appendix M

Correspondence with SUHREC

To: Assoc Prof Greg Murray/Ms Simmone Poulios, FLSS

Dear Greg and Simmone

SUHREC Project 2009/002 The Impact of Sleep on Bipolar Vulnerability
Assoc Prof Gregory Murray, FLSS; Ms Simmone Poulios
Approved Duration: 30/03/2009 to 31/12/2011 [Modified April 2009; July/August/October/November 2009; February 2010; March 2010]
I refer to your request to further modify the above project as per your emails of 16, 25 and 26 March 2010, in particular concerning expanded recruitment arrangements. The request documentation was put to the Chair of SUHREC for consideration.
I am pleased to advise that, as submitted to date, the modified protocol has approval to proceed in line with standard on-going ethics clearance conditions previously communicated and reprinted below.
Please contact me if you have any queries about on-going ethics clearance, citing the SUHREC project number. A copy of this email should be retained as part of project record-keeping.
As before, best wishes for the continuing project.

Yours sincerely

Keith Wilkins
Secretary, SUHREC

*******************************************

Keith Wilkins
Research Ethics Officer
Swinburne Research (H68)
Swinburne University of Technology
P O Box 218
HAWTHORN VIC 3122
Tel +61 3 9214 5218
Fax +61 3 9214 5267

>>> Keith Wilkins 15/02/2010 6:26 PM >>>

To: Assoc Prof Greg Murray/Ms Simmone Poulios, FLSS

Dear Greg and Simmone

SUHREC Project 2009/002 The Impact of Sleep on Bipolar Vulnerability
Assoc Prof Gregory Murray, FLSS; Ms Simmone Poulios
Approved Duration: 30/03/2009 to 31/12/2011 [Modified April 2009; July/August/October/November 2009; February 2010]

I refer to your request to further modify the above project as per your email today with attachments. The request documentation was put to the Chair of SUHREC for consideration.

I am pleased to advise that, as submitted to date, the modified protocol has approval to proceed in line with standard on-going ethics clearance conditions previously communicated and reprinted below.

Please contact me if you have any queries about on-going ethics clearance, citing the SUHREC project number. A copy of this email should be retained as part of project record-keeping.

As before, best wishes for the project.

Yours sincerely

Keith Wilkins
Secretary, SUHREC

>>> Keith Wilkins 19/11/2009 11:32 AM >>>

To: Assoc Prof Greg Murray/Ms Simmone Poulios, FLSS

Dear Greg and Simmone

SUHREC Project 2009/002 The Impact of Sleep on Bipolar Vulnerability
Assoc Prof Gregory Murray, FLSS; Ms Simmone Poulios
Approved Duration: 30/03/2009 to 31/12/2011 [Modified April 2009; July/August/October/November 2009]

I refer to your request to further modify the above project as per your email of 11 November 2009 to expand further the pool of participants. The request, including text for an additional consent instrument, was put to the Chair of SUHREC for consideration.

I am pleased to advise that, as submitted to date, the modified protocol has approval to proceed in line with standard on-going ethics clearance conditions previously communicated and reprinted below.
Please contact me if you have any queries about on-going ethics clearance, citing the SUHREC project number. A copy of this email should be retained as part of project record-keeping.

As before, best wishes for the project.

Yours sincerely

Keith Wilkins

Secretary, SUHREC

>>> Keith Wilkins 30/10/2009 2:48 PM >>>

To: Assoc Prof Greg Murray/Ms Simmone Poulios, FLSS

Dear Greg and Simmone

SUHREC Project 2009/002 The Impact of Sleep on Bipolar Vulnerability

Assoc Prof Gregory Murray, FLSS; Ms Simmone Poulios

Approved Duration: 30/03/2009 to 31/12/2011 [Modified April 2009; July/August/October 2009]

I refer to your request to further modify the above project as per your email of 27 October 2009 to expand the pool of participants. The request was able to be put to the Chair of SUHREC for consideration today.

I am pleased to advise that, as submitted to date, the modified protocol has approval to proceed in line with standard on-going ethics clearance conditions previously communicated and reprinted below. Please be alert to due care needed when recruiting via "snowball email' to prevent identifiable material (including an individual's contact information) being communicated without prior consent of the individuals concerned.

Please contact me if you have any queries about on-going ethics clearance, citing the SUHREC project number. A copy of this email should be retained as part of project record-keeping.

As before, best wishes for the project.

Yours sincerely

Keith Wilkins

Secretary, SUHREC

>>> Keith Wilkins 24/08/2009 4:25 PM >>>

To: Assoc Prof Greg Murray/Ms Simmone Poulios, FLSS

Dear Greg and Simmone

SUHREC Project 2009/002 The Impact of Sleep on Bipolar Vulnerability

Assoc Prof Gregory Murray, FLSS; Ms Simmone Poulios
I refer to your request to further modify the above project as per your email of 18 August 2009 which was able to be put to a delegate of SUHREC for consideration.

I am pleased to advise that, as submitted to date, the modified protocol has approval to proceed in line with standard on-going ethics clearance conditions previously communicated and reprinted below. Please contact me if you have any queries about on-going ethics clearance, citing the SUHREC project number. A copy of this email should be retained as part of project record-keeping.

As before, best wishes for the project.

Yours sincerely

Keith Wilkins
Secretary, SUHREC

>>> Keith Wilkins 28/07/2009 6:09 PM >>>

To: Assoc Prof Greg Murray/Ms Simmone Poulios, FLSS

Dear Greg and Simmone

SUHREC Project 2009/002 The Impact of Sleep on Bipolar Vulnerability

Assoc Prof Gregory Murray,FLSS;Ms Simmone Poulios

Approved Duration: 30/03/2009 to 31/12/2011 [Modified April 2009; July 2009]

I refer to your request to further modify the above project as per your email of today which was able to be put to a delegate of SUHREC for consideration.

I am pleased to advise that, as submitted to date, the modified protocol has approval to proceed in line with standard on-going ethics clearance conditions previously communicated and reprinted below. Please contact me if you have any queries about on-going ethics clearance, citing the SUHREC project number. A copy of this email should be retained as part of project record-keeping.

As before, best wishes for the project.

Yours sincerely

Keith Wilkins
Secretary, SUHREC

>>> Keith Wilkins 24/04/2009 5:39 PM >>>

To: Assoc Prof Greg Murray/Ms Simmone Poulios, FLSS
Dear Greg and Simmone

SUHREC Project 2009/002 The Impact of Sleep on Bipolar Vulnerability

Assoc Prof Gregory Murray FLSS Ms Simmone Poulios

Approved Duration: 30/03/2009 to 31/12/2011 [Modified April 2009]

I refer to your request to modify the above project as per your email of 23 April 2009 which was put to a
delegate of SUHREC for consideration.

I am pleased to advise that, as submitted to date, the modified protocol has approval to proceed in line
with standard on-going ethics clearance conditions previously communicated and reprinted below.

Please contact me if you have any queries about on-going ethics clearance, citing the SUHREC project
number. Chief Investigators/Supervisors and student researchers should retain a copy of this email as part
of project record-keeping.

As before, best wishes for the project.

Yours sincerely

Keith Wilkins

Secretary, SUHREC

>>> Keith Wilkins 6/04/2009 1:09 pm >>>

To: Assoc Prof Greg Murray/Ms Simmone Poulios, FLSS

Dear Greg and Simmone

SUHREC Project 2009/002 The Impact of Sleep on Bipolar Vulnerability

Assoc Prof Gregory Murray FLSS Ms Simmone Poulios

Approved Duration: 30/03/2009 to 31/12/2011

I refer to the ethical review of the above project protocol by Swinburne's Human Research Ethics
Committee (SUHREC). Your responses to the review, as emailed on 25 March 2009, were put to a
SUHREC delegate for consideration as to sufficiency.

I am pleased to advise that, as submitted to date, the project may proceed in line with standard on-going
ethics clearance conditions here outlined.

- All human research activity undertaken under Swinburne auspices must conform to Swinburne and
external regulatory standards, including the National Statement on Ethical Conduct in Human Research
and with respect to secure data use, retention and disposal.
- The named Swinburne Chief Investigator/Supervisor remains responsible for any personnel appointed to or associated with the project being made aware of ethics clearance conditions, including research and consent procedures or instruments approved. Any change in chief investigator/supervisor requires timely notification and SUHREC endorsement.

- The above project has been approved as submitted for ethical review by or on behalf of SUHREC. Amendments to approved procedures or instruments ordinarily require prior ethical appraisal/ clearance. SUHREC must be notified immediately or as soon as possible thereafter of (a) any serious or unexpected adverse effects on participants and any redress measures; (b) proposed changes in protocols; and (c) unforeseen events which might affect continued ethical acceptability of the project.

- At a minimum, an annual report on the progress of the project is required as well as at the conclusion (or abandonment) of the project.

- A duly authorised external or internal audit of the project may be undertaken at any time.

Please contact me if you have any queries about on-going ethics clearance, citing the SUHREC project number. Chief Investigators/Supervisors and student researchers should retain a copy of this email as part of project record-keeping.

Best wishes for the project.

Yours sincerely

Keith Wilkins

Secretary, SUHREC

**********************************************************************
Appendix N

Website addresses where research was advertised

Online Psychology Research-UK:

http://www.onlinepsychresearch.co.uk/

Hanover college:

http://psych.hanover.edu/Research/exponnet.html

In Mind online magazine:

http://www.in-mind.org/online-research/index.php

Appendix O

Example of the Ladder and Gladder Command Outputs

sGBIdepresstotal

<table>
<thead>
<tr>
<th>Transformation</th>
<th>formula</th>
<th>chi2(2)</th>
<th>P(chi2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>cubic</td>
<td>sGBI-de^3</td>
<td>.</td>
<td>0.000</td>
</tr>
<tr>
<td>square</td>
<td>sGBI-de^2</td>
<td>.</td>
<td>0.000</td>
</tr>
<tr>
<td>identity</td>
<td>sGBI-de^1</td>
<td>58.98</td>
<td>0.000</td>
</tr>
<tr>
<td>square root</td>
<td>sqrt(sGBI-de^1)</td>
<td>2.75</td>
<td>0.252</td>
</tr>
<tr>
<td>log</td>
<td>log(sGBI-de^1)</td>
<td>.</td>
<td>.</td>
</tr>
<tr>
<td>1/(square root)</td>
<td>1/sqrt(sGBI-de^1)</td>
<td>.</td>
<td>.</td>
</tr>
<tr>
<td>inverse</td>
<td>1/sGBI-de^1</td>
<td>.</td>
<td>.</td>
</tr>
<tr>
<td>1/square</td>
<td>1/(sGBI-de^2)</td>
<td>.</td>
<td>.</td>
</tr>
<tr>
<td>1/cubic</td>
<td>1/(sGBI-de^3)</td>
<td>.</td>
<td>.</td>
</tr>
</tbody>
</table>

Quantile-Normal plots by transformation
### Appendix P

Sample description of research used for published means

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample</th>
<th>Scale and published mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poulios et al., 2010</td>
<td>N = 479; General population</td>
<td>Mean age = 29 years; sGBI-Depression = 11.18, sGBI-Hypomania = 6.25</td>
</tr>
<tr>
<td>Smith et al., 1989</td>
<td>N = 501; Undergraduate students</td>
<td>Age not reported; Composite Scale = 23.6</td>
</tr>
<tr>
<td>Fortunato et al., 2008</td>
<td>N = 166; Undergraduate students</td>
<td>Going to bed = 18.0; Falling asleep = 20.90, Staying asleep = 20.1, Reinitiating sleep = 21.1, Returning to wakefulness = 16.85</td>
</tr>
<tr>
<td>Murray et al., 2009</td>
<td>N = 27; General population and university students</td>
<td>Mean age = 31.4 years; Neuroticism = 25.7, Extraversion = 34.3, Openness = 36.4, Agreeableness = 37.7, Conscientiousness = 36.1</td>
</tr>
<tr>
<td>Jorm et al., 1999</td>
<td>N = 312; General population recruited from electoral role.</td>
<td>Age = 18-29 year old females; BAS Drive = 10.7, BAS Fun Seeking = 11.9, BAS Reward Responsiveness = 17.6, BIS = 20.0</td>
</tr>
<tr>
<td>Buysse et al., 1989</td>
<td>N = 148; Combination of healthy controls (n = 54, mean age = 59.9 years), poor sleepers (n = 34, mean age = 50.9 years), and physician referred outpatients (n = 45 diagnosed with Disorder of initiating and maintaining sleep and mean age = 44.8 years, and 17 diagnosed with Disorder of excessive somnolence and mean age = 42.2 years)</td>
<td>Pittsburgh Sleep Quality Index score = 7.4</td>
</tr>
<tr>
<td>Watson et al., 1988</td>
<td>N = 660; Undergraduate students</td>
<td>Temporal instruction: “right now”; Positive Affect = 29.7, Negative Affect = 31.31</td>
</tr>
<tr>
<td>Crawford &amp; Henry, 2004</td>
<td>N = 100; Non clinical, general adult UK population.</td>
<td>Mean age = 42.9 years; Positive Affect = 31.31, Negative Affect = 16.00</td>
</tr>
<tr>
<td>Diener et al., 1985</td>
<td>N = 176 Undergraduate students</td>
<td>Age not reported; Satisfaction with Life Scale = 23.5</td>
</tr>
</tbody>
</table>

*Note. N = sample size*
Appendix Q

Intercorrelations between scores on sGBI, BIS/BAS scales and API

<table>
<thead>
<tr>
<th>Measures</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. sGBIdep</td>
<td>.86**</td>
<td>.12**</td>
<td>.17**</td>
<td>.12**</td>
<td>.27**</td>
<td>.53**</td>
<td>-.12**</td>
<td>.10*</td>
<td>-.27**</td>
<td>-.31**</td>
</tr>
<tr>
<td>2. sGBIHyp</td>
<td>-.01</td>
<td>.01</td>
<td>.03</td>
<td>.40**</td>
<td>.72**</td>
<td>-.30**</td>
<td>.03</td>
<td>-.31**</td>
<td>-.41**</td>
<td></td>
</tr>
<tr>
<td>3. BASd</td>
<td>.41**</td>
<td>.50**</td>
<td>-.01</td>
<td>-.11**</td>
<td>.33**</td>
<td>.22**</td>
<td>-.05</td>
<td>.20**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. BASfs</td>
<td>.42**</td>
<td>-.15**</td>
<td>-.17**</td>
<td>.38**</td>
<td>.27**</td>
<td>.04</td>
<td>-.06</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. BASrr</td>
<td>.27**</td>
<td>-.05</td>
<td>.26**</td>
<td>.24**</td>
<td>.18**</td>
<td>.16**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. BIS</td>
<td>.58**</td>
<td>-.22**</td>
<td>-.07</td>
<td>-.03</td>
<td>-.22**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Neuroticism</td>
<td>-.41**</td>
<td>-.06</td>
<td>-.36**</td>
<td>-.43**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Extraversion</td>
<td>.30**</td>
<td>.24**</td>
<td>.31**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Openness</td>
<td>.10*</td>
<td>.17**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Agreeableness</td>
<td>.37**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Conscientiousness</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. BASd = BAS Drive; BASrr = BAS Reward Responsiveness; BASfs = BAS Fun Seeking.
* p < .05. ** p < .01.

N = 608
## Appendix R

Intercorrelations between scores on the sGBI, modified ASWS, and PSQI

<table>
<thead>
<tr>
<th>Measures</th>
<th>Correlation Coefficients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2.</td>
</tr>
<tr>
<td>1. sGBI depression</td>
<td>.86**</td>
</tr>
<tr>
<td>2. sGBI Hypomania</td>
<td>-.21**</td>
</tr>
<tr>
<td>3. Morningness</td>
<td>.56**</td>
</tr>
<tr>
<td>4. GTB</td>
<td>.54**</td>
</tr>
<tr>
<td>5. FA</td>
<td>.44**</td>
</tr>
<tr>
<td>6. SA</td>
<td>.54**</td>
</tr>
<tr>
<td>7. RS</td>
<td>.20**</td>
</tr>
<tr>
<td>8. WU</td>
<td>.</td>
</tr>
</tbody>
</table>

*N = 608

*Note. GTB = Going to Bed score; FA = Falling Asleep score; SA = Staying Asleep score; RS = Reinitiating Sleep score; WU = Waking Up score.*  

*p < .05. ** p < .01.
Appendix S

Standardised Direct and Indirect Effect Table Outputs from AMOS from BD, Sleep and Coping Model containing sGBI-Depression Scores

### Standardized Direct Effects (Group number 1 - Default model)

<table>
<thead>
<tr>
<th></th>
<th>SA</th>
<th>RS</th>
<th>FA</th>
<th>WU</th>
<th>GTB</th>
<th>Depression</th>
<th>Avoidant_Coping</th>
<th>Supportseeking</th>
<th>PSQItotal</th>
<th>Activecoping</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>-.135</td>
<td>-.209</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>-.299</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Avoidant_Coping</td>
<td>&lt;.001</td>
<td>-.088</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>-.088</td>
<td>&lt;.001</td>
<td>.377</td>
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<td>&lt;.001</td>
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<td>.112</td>
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<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PSQI</td>
<td>-.281</td>
<td>-.163</td>
<td>-.351</td>
<td>-.126</td>
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<td>.091</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Activecoping</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>.229</td>
<td>-.110</td>
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<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
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<tr>
<td>Morningness</td>
<td>&lt;.001</td>
<td>-.116</td>
<td>&lt;.001</td>
<td>.635</td>
<td>.292</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
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<tr>
<td>SWLS</td>
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<td>-.212</td>
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<tr>
<td>PA</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>.289</td>
<td>.099</td>
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<td>.228</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>.113</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

### Standardized Indirect Effects (Group number 1 - Default model)

<table>
<thead>
<tr>
<th></th>
<th>SA</th>
<th>RS</th>
<th>FA</th>
<th>WU</th>
<th>GTB</th>
<th>Depression</th>
<th>Avoidant_Coping</th>
<th>Supportseeking</th>
<th>PSQItotal</th>
<th>Activecoping</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
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<td>Avoidant_Coping</td>
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<td>&lt;.001</td>
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<tr>
<td>Supportseeking</td>
<td>.017</td>
<td>.026</td>
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<td>&lt;.001</td>
<td>.037</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
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<tr>
<td>PSQI</td>
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</tr>
<tr>
<td>Activecoping</td>
<td>.015</td>
<td>.023</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>.033</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Morningness</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
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<td>SWLS</td>
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<td>-.035</td>
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<td>&lt;.001</td>
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<td>-.040</td>
<td>-.039</td>
<td>-.105</td>
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Standardised Direct and Indirect Effect Table Outputs from AMOS from BD, Sleep and Coping Model containing sGBI-Hypomania Scores

### Standardized Direct Effects (Group number 1 - Default model)

<table>
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<tr>
<th></th>
<th>GTB</th>
<th>FA</th>
<th>SA</th>
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<th>RS</th>
<th>Morningness</th>
<th>Hypomania</th>
<th>Supportseeking</th>
<th>Activecoping</th>
<th>Avoidant_Coping</th>
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### Standardized Indirect Effects (Group number 1 - Default model)

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<th>RS</th>
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<th>Hypomania</th>
<th>Supportseeking</th>
<th>Activecoping</th>
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<th>PSQI Total</th>
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