Cogn Ther Res
DOI 10.1007/s10608-013-9541-4

ORIGINAL ARTICLE

Self Appraisals of Internal States and Risk of Analogue Bipolar Symptoms in Student Samples: Evidence from Standardised Behavioural Observations and a Diary Study

Alyson L. Dodd · Warren Mansell · Rosie A. Beck · Sara J. Tai

Abstract An integrative cognitive model proposed that individuals vulnerable to bipolar disorder (BD) assign extreme personal meaning to internal states. This research investigated the utility of the Hypomanic Attitudes and Positive Predictions Inventory as a cognitive risk measure for BD. Study 1 ($N = 64$; mean age 21.8 years, 42 female) explored whether students at cognitive risk had more extreme changes in mood and both self-reported and observer-rated bipolar-relevant symptoms during an interview task following a mood induction. The risk group did not respond differentially to the mood induction, but they spoke faster and dominated the conversation more during the interview task, self-reported greater activation, depression and negative affect, and scored higher on hypomanic personality, reward sensitivity, and dysfunctional attitudes. When controlling for other established cognitive measures, activation was still higher in the cognitive risk group at trend, and depression and negative affect were significantly higher. Activation, depression, and negative affect were still significantly higher in the cognitive risk group when controlling for reward sensitivity. Study 2 ($N = 30$; mean age 19.93 years, 21 female) complemented the experimental study with a 7 days diary study of everyday mood and behaviour. The risk group reported higher negative affect and bipolar-relevant symptoms. These results are consistent with the role of extreme appraisals of internal state in vulnerability to BD.

Keywords Mood induction · Bipolar disorder · Appraisals · Hypomania · Depression · Hypomanic personality

A. L. Dodd (✉)
Division of Health Research, Spectrum Centre for Mental Health Research, Lancaster University, Lancaster LA1 4YT, UK
e-mail: a.dodd@lancaster.ac.uk

W. Mansell · S. J. Tai
School of Psychological Sciences, University of Manchester, Manchester, UK

R. A. Beck
Department of Clinical Psychology, University of Liverpool, Liverpool, UK

Introduction

For the past two decades, growing evidence has highlighted the role of psychological processes in the development of mood swings and bipolar disorder (BD). Improving understanding of these processes gives us the potential to facilitate the identification of high risk individuals, and improve psychological interventions. Vulnerability to BD has been attributed to over-sensitivity of a motivational brain system labelled the behavioural activation system (BAS: Depue and Iacono 1989; Urosevic et al. 2008). The BAS is proposed to be sensitive to signals of reward (Depue and Iacono 1989). At the extreme, high-BAS activity has been linked to mania, and low-BAS activity to depression. Several researchers have found relationships between BAS sensitivity, as measured by the Behavioural Inhibition and Behavioural Activation Scales (BIS/BAS; Carver and White 1994) and the symptoms of BD in non-clinical and clinical samples (e.g., Alloy et al. 2012; Mansell et al. 2008; Meyer et al. 1999, 2001; Van der Gucht et al. 2009).

The BIS/BAS scales were designed to tap stable trait-like physiological, motivational processes relating to reward sensitivity rather than cognitive processes (Carver and White 1994), although BAS-relevant cognitions (e.g.,

Published online: 24 April 2013

goal-attainment beliefs) have come to the fore in theory and research more recently (Urosevic et al. 2008; Johnson et al. 2012). Research into cognitive processes underlying BD has also focussed on measures developed to examine depression-relevant cognitions, such as the Dysfunctional Attitudes Scale (DAS; Weissman and Beck 1978). Findings have been mixed, although it appears that individuals with BD show negative cognitive styles reminiscent of individuals with unipolar depression, even in the manic phase, including need for approval, dependency, self-criticism and perfectionism (for reviews, see Mansell et al. 2005; Mansell and Scott 2006). Interestingly, one study (Lam et al. 2004) found that remitted BD individuals differed significantly from remitted unipolar individuals on a Goal Attainment subscale of the DAS. Further studies have found an association between excessive, overly ambitious goal attainment beliefs and vulnerability to BD (for a review, see Johnson et al. 2012), as well as goal attainment life events and goal-striving (Alloy et al. 2012; Johnson et al. 2000). Further, during a goal-oriented task, students at risk of mania endorsed higher ratings of both positive and negative self-appraisals, relative to students at low risk (Taylor and Mansell 2008). Excessively optimistic goal beliefs are also consistent with the BAS dysregulation model of BD, such that these cognitions and life events would be considered BAS-relevant (Urosevic et al. 2008). This factor may form a particular set of extreme beliefs unique to BD, which drive moods upwards via engagement in activating behaviours, such as excessive spending. However, the BAS scales and DAS were respectively designed to measure sensitivity to reward and depressogenic cognitive styles characteristic of unipolar depression, and findings in relation to BD have been mixed (e.g., Mansell et al. 2008; Mansell and Scott 2006).

There has been an increasing focus on the role of cognitive appraisals in BD. Cognitive appraisals of intrusions into awareness, such as the catastrophic misinterpretation of bodily sensations in panic disorder (Clark 1986) or culturally unacceptable interpretations of intrusions such as auditory hallucinations (Morrison 2001), are transdiagnostic maintaining features for psychological disorders (Harvey et al. 2004). It is the content of appraisals that is 'disorder-specific'; in BD, there is evidence that excessively positive self-appraisals of hypomanic experiences, and negative self-appraisals of depression-relevant experiences, are associated with vulnerability to mania and diagnosis of BD (Jones and Day 2008; Jones et al. 2006). In particular, it is proposed that internal changes that arise from circadian rhythm disruptions are then appraised in a self-relevant way (Jones 2001).

The Hypomanic Attitudes and Positive Predictions Inventory (HAPPI; Mansell 2006) was designed to assess a wide scope of beliefs about internal states thought to be specific to BD including self-activating, self-critical and catastrophic beliefs about the consequences and losing control. The HAPPI was developed in tandem with an integrative cognitive model of BD (Mansell et al. 2007), which proposed that individuals vulnerable to BD hold multiple, extreme, positive and negative personalised appraisals of internal states. For example, assigning extreme positive meaning to increased activation (e.g., 'When I am excited I can do no wrong') would be expected to propel mood upwards, as the individual would respond by attempting to enhance activation via engaging in ascent behaviours (e.g., avoiding sleep). Conversely, a negative appraisal (e.g., 'When I feel excited, I am about to have a breakdown') would instigate attempts to control or decrease activation to avoid becoming too high, known as descent behaviours (e.g., reducing activity). Further fluctuations in activation triggered by engagement in these behaviours would also be appraised in an excessively self-relevant manner. At the extreme, this would result in escalating bipolar symptoms.

The HAPPI has distinguished individuals with a diagnosis of BD from non-clinical controls (Mansell 2006; Mansell and Jones 2006) and individuals with a diagnosis of unipolar depression (Alatiq et al. 2010; Mansell et al. 2011) even when controlling for current symptoms. In analogue samples (Mansell et al. 2008; Dodd et al. 2010; 2011a, b), the HAPPI has been found to predict past hypomanic experiences and bipolar symptomatology, and specific components were related to different clusters of bipolar symptoms. The HAPPI also predicted prospective bipolar symptoms, and lower functioning, after 4 weeks in individuals with diagnosable BD (Dodd et al. 2011c); in the same study, reward sensitivity was not associated with symptoms. Taken together, these results suggest that stronger conviction in both positive and negative self-referent appraisals of internal states put individuals at risk of developing bipolar-relevant symptomatology.

Previous research has assigned students at high or low cognitive risk for depression based on attributional style and dysfunctional attitudes (e.g., Alloy et al. 2006; Bentall et al. 2011). Given the evidence for extreme appraisals and vulnerability to BD (Dodd et al. 2010; 2011a, b, c), students with elevated scores on the HAPPI could be considered at cognitive risk to BD. The key aim of the current research was to investigate, via several means, whether extreme appraisals conferred cognitive risk of BD in a non-clinical population. Study 1 is the first experimental investigation of whether students with more positive and negative self-appraisals are (1) at greater risk of developing BD as determined by existing measures of vulnerability to BD and (2) susceptible to more exaggerated mood responses to success and failure. Eisner et al. (2008) have previously reported that students at risk of developing mania were more likely to engage in positive

over-generalisation *after* a success, whereas this research was designed to assess if cognitive style measured *before* a mood induction conferred risk of a more extreme response to success. Study 1 adopted a cross-sectional design to explore the following objectives:

1. Is heightened cognitive risk of mood swings related to higher rates of (1) bipolar risk and (2) psychological processes putatively associated with vulnerability to BD? It was predicted that the cognitive risk group would display higher rates of bipolar risk, reward sensitivity, dysfunctional attitudes, and positive self-dispositional appraisals relative to controls. These were assessed by self-report measures.
2. Does heightened cognitive risk of mood swings make individuals more susceptible to analogue bipolar symptoms? It was predicted that the cognitive risk group would (1) display higher bipolar-relevant symptoms overall relative to a control group and (2) display greater changes in positive and negative affect and bipolar-relevant symptoms in immediate response to a mood induction. Affect and bipolar-relevant symptoms were assessed using a combination of self-report measures and objective, observed behavioural measure of pressured speech.
3. Does heightened cognitive risk make a unique contribution to bipolar-relevant symptoms? When controlling for other psychological processes known to be associated with vulnerability to BD, it was predicted that the cognitive risk group would (1) display higher bipolar-relevant symptoms overall relative to a control group and (2) display greater changes in positive and negative affect and bipolar-relevant symptoms in immediate response to a mood induction.

Study 2 examined prospective relationships between cognitive risk, bipolar-relevant symptoms and mood using a diary in everyday life and therefore complemented the experimental study to explore the sequalae of cognitive risk over a longer period within an ecologically valid setting. The objectives of Study 2 were to:

1. Investigate whether cognitive risk predicted greater prospective bipolar-relevant symptoms and mood states. It was hypothesised that the cognitive risk group would demonstrate higher scores on self-report measures of bipolar-relevant symptoms and both positive and negative affect over a 1 week period, relative to a control group.
2. Investigate whether cognitive risk predicted greater variability in bipolar-relevant symptoms and mood. It was predicted that the cognitive risk group would demonstrate greater variability in bipolar-relevant and positive and negative affect over a 1 week period.

Study 1

Introduction

Mood induction procedures (MIPs) have been used to examine relationships between psychological processes and mood in a controlled setting (for a review, see Martin 1990). It has been reported that mood manipulation is most effective when the participant is explicitly told, or can easily guess, the mood being induced (Gerrards-Hesse et al. 1994; Martin 1990; Westermann 1996). However, the success of these techniques may result from experimenter demand effects. Success/failure manipulations are an ecologically valid alternative (e.g., Eisner et al. 2008), as participants are not merely passive audiences, but are actively involved in the MIP (Westermann 1996). In a previous study, a success/failure paradigm (Krohne et al. 2002) significantly induced positive and negative affect after experiencing success and failure, respectively. This paradigm was adopted in the current study for several reasons. Firstly, the implicit nature of the task controlled for demand effects. Task difficulty was manipulated to fix success/failure outcomes, as opposed to giving the participants false feedback, so suspicion regarding the true purpose of the study was unlikely (see Westermann 1996). Secondly, the MIP was deemed ecologically valid, as participants were told that their score would be predictive of academic success, which was assumed to have personal meaning for the participants, who were all students. It has been demonstrated that manic symptoms are related to goal-attainment life events among individuals with BD (Johnson et al. 2000), and with a goal-striving event (final exams) among students with softer symptom expression 'bipolar spectrum disorder' (Nusslock et al. 2007). In the current study, achieving a high score on the task could be construed as a goal for students, as it was supposedly relevant to their future academic achievements.

We examined whether the cognitive risk group displayed heightened scores on hypomanic personality, reward sensitivity, dysfunctional attitudes, positive self appraisals, and history of hypomanic symptoms. In addition, it was predicted that the cognitive risk group would show a greater increase in analogue hypomanic symptoms and positive affect after the success mood induction, as compared to individuals in the control group. Further, two behavioural indices were taken as objective measures of hypomanic symptoms. In line with clinical presentations, these were pressure of speech, and the extent to which the participant dominated the conversation. Pressure of speech is included in the criteria of the Diagnostic and Statistical Manual for Mental Disorder [DSM-IV-TR; APA 2000], as an observable symptom of hypomania. The unbiased nature of these measures increased the validity of the current

study. In the failure condition, it was predicted that the cognitive risk group would show a greater increase in analogue depressive symptoms and negative affect after mood induction.

Method

Participants

At the screening stage of the study, students from the University of Manchester were invited to complete the HAPPI online via an advertisement on the university research volunteering website and posters throughout campus. Based on the distribution of HAPPI scores within the initial sample, individuals were split into groups; those with a mean HAPPI score ≥47 (90th percentile of HAPPI scores) were included in the cognitive risk group, and individuals with a mean HAPPI ≤41.65 (0.5 SD above the mean) were controls. The experimenter was blind to group to reduce bias in observer-rated assessments. Group allocation information was held by a co-author, and participants were invited to meet with the experimenter on a first-come-first-served basis from a list without group details. This control was necessarily dropped towards the end, as the groups were unbalanced in favour of the control group and participants were purposefully selected from the high risk group at this point. On the day of testing, participants were screened for current manic or depressive symptoms using the Bech–Rafaelsen Mania Rating Scale (BMRS; Bech et al. 1978) and Beck Depression Inventory-II (BDI-II; Beck 1996). In line with recommendations (Cox et al. 2001), anyone scoring ≥16 on the latter was excluded, as it was deemed inappropriate to manipulate mood in an individual who was experiencing depressive symptoms. It was originally intended that individuals scoring ≥6 on the BMRS would also be excluded, but none of the participants exceeded this score. In the final sample, there were 32 participants in each group. The mean age of the overall sample ($N = 64$) was 21.8 years (SD = 3.53), and 42 were female. A one-way ANOVA found that the HAPPI groups did not differ with respect to age, $F(1, 63) = 2.98$, *ns*. The gender ratio was identical in both groups. 2nd year Psychology undergraduates were awarded course credits for taking part. All other participants were awarded an incentive of £5.

Materials

Screening Measures

HAPPI (Mansell 2006; Dodd et al. 2010). The extended 61-item version (Dodd et al. 2010) was administered in this study. Participants completed the HAPPI online to determine whether they were in the cognitive risk or control group, and then in paper format on the day of testing to assess test–retest reliability ($r = .89$, $p < .01$, after an average of 2 weeks). In the online version, participants indicated to what extent they agreed with each statement using a drop-down menu, whereas the paper version utilised a visual analogue scale. The rating scale was divided into intervals of 10 from 0 ('I don't believe this at all') to 100 ('I believe this completely'). The HAPPI has a high internal consistency of Cronbach's $\alpha = .97$ (Dodd et al. 2010).

BMRS (Bech et al. 1978). The BMRS is an 11-item observer-rated scale which covers a range of manic symptoms, including motor activity, racing thoughts, and self-confidence. Each item was scored according to 5 categories regarding to what extent that symptom was present. High inter-rater reliability has been reported (r = .80 to .95; Bech et al. 1978).

BDI-II (Beck 1996). The BDI-II is a 21-item self-report measure based around the diagnostic criteria for major depression (e.g., low mood, sleep disturbances, appetite change, concentration difficulties; APA 2000). Participants rated to what extent they had experienced each depressive symptom over the previous fortnight.

Reward Sensitivity

BIS/BAS (Carver and White 1994). This study utilised a revised version of the BIS/BAS scales, which incorporated a dimension reflecting instability of BAS activity, labelled BAS Dysregulation (Holzwarth and Meyer 2006). In the current study, the four highest loading items of the BAS Dysregulation subscale, as found through factor analysis (Holzwarth and Meyer 2006) were used, to reflect the length of the original BAS subscales (Carver and White 1994); Reward Responsiveness (e.g., 'It would excite me to win a contest'), Drive (e.g., 'When I go after something I use a "no holds barred" approach'), and Fun-Seeking (e.g., 'I'm always willing to try something new if I think it will be fun'; Carver and White 1994). In addition, the BIS (e.g., 'Criticism or scolding hurts me quite a bit') was included. The BAS scales were included to assess reward sensitivity. Internal consistencies for the subscales were Cronbach's $\alpha = .54$ for Reward Responsiveness, .76 for Drive, .77 for Fun-Seeking, .85 for both BIS and BAS Dysregulation, largely in keeping with previously reported internal consistencies (Cronbach's alpha from .60 to .84; Holzwarth and Meyer 2006). Participants ascribed a number 1–4 to each statement, where 1 = 'Very false for me' and 4 = 'Very true for me'.

Positive and Negative Cognitive Style

DAS (Weissman and Beck 1978). The 24-item version (Power et al. 1994) was used in this study. Among individuals diagnosed with BD and unipolar depression, a principal components analysis (Lam et al. 2004) identified 3 subscales, which were used in the current study to assess

bipolar-relevant dysfunctional attitudes: Goal-Attainment (e.g., 'I should always have complete control over my feelings'; Cronbach's $\alpha = .79$), Dependency (e.g., 'If others dislike you, you can not be happy'; Cronbach's $\alpha = .78$) and Achievement (e.g., If I don't set high standards I will end up being second rate'; Cronbach's $\alpha = .80$). Participants rated the extent they agreed with each statement on a Likert scale from 1 to 7, where 1 = 'Totally agree' and 7 = 'Totally disagree'.

HIQ (Jones et al. 2006). Ten hypomania-relevant situations were described (e.g., 'If I felt impulsive I would probably think it was because…'). For each situation, two possible explanations are given: a positive self-dispositional appraisal (HIQ-Hypomania; e.g., '…I could make rapid decisions and good choices') and a normalising appraisal (HIQ-Normalising; e.g., '…something had disrupted my routine'). Participants were asked to rate how much each appraisal would explain the situation for them from A = 'Not at all' to D = 'A great deal'. In an analogue sample (Jones et al. 2006), internal consistencies for HIQ-Hypomania and HIQ-Normalising were adequate; Cronbach's $\alpha = .83$ and .73. Test–retest correlations were $r = .56$ for HIQ-Hypomania and $r = .59$ for HIQ-Normalising.

Risk of Bipolar Disorder

HIQ (Jones et al. 2006). In addition to the cognitive subscales of the HIQ above, participants indicated whether they had experienced the situation described within the previous 3 months (HIQ-Experience). As such, HIQ-Experience was included as a measure of history of hypomanic symptoms.

Hypomanic Personality Scale (HYP; Eckblad and Chapman 1986). Hypomanic personality is a gregarious, grandiose set of traits proposed to fall on the 'bipolar spectrum' (Angst and Gamma 2002). The HYP has been associated with the symptoms of BD (Eckblad and Chapman 1986; Meyer 2002; Meyer and Hautzinger 2003). At 13-year follow-up (Kwapil et al. 2000), high scorers on the HYP had a higher occurrence of BD diagnoses compared to controls. Consequently, the HYP was included in this study as a measure of bipolar risk, in keeping with previous research (e.g., Eisner et al. 2008; Hofmann and Meyer 2006; Meyer and Hautzinger 2003).The HYP consists of 48 true/false statements (e.g., 'I often feel excited and happy for no reason'). HYP has good internal consistency (Cronbach's $\alpha = .87$) and test–retest reliability ($r = .81$ after 15 weeks; Eckblad and Chapman 1986).

Mood Disorders Questionnaire (MDQ; Hirschfeld et al. 2000). The MDQ is a self-report inventory designed to screen for past hypomanic symptoms (e.g., 'Has there ever been a period of time when you were not your usual self and you felt much more self-confident than usual?'), based DSM-IV-TR criteria (APA 2000). Participants answered 'Yes' or 'No' to each item. A further yes/no question asks if several of these symptoms have ever occurred simultaneously. The final question enquires about the severity of the symptoms ('No problem' through to 'Serious problem'). The MDQ was included as a measure of history of hypomanic symptoms. In the initial sample of patients with mood disorder, this measure had good internal consistency, Cronbach's $\alpha = .90$ (Hirschfeld et al. 2000).

Bipolar Relevant-Symptoms and Mood

Internal States Scale (ISS; Bauer et al. 1991). The ISS was developed to assess concurrent depressive and manic symptoms, and comprises 4 subscales representing different symptoms: ISS Activation (e.g., 'Today I feel "sped up" inside'), which measures cognitive and behavioural activation, such as racing thoughts and elevated energy; ISS Conflict (e.g., 'Today I feel argumentative'), which measures irritability; ISS Well-Being (e.g., 'Today I actually feel great inside'), which measures psychological well-being; and ISS Depression (e.g., 'Today it seems like nothing will work out for me'), which measures depressive symptoms. Internal consistencies ranged from Cronbach's $\alpha = .81$ to .92 (Bauer et al. 1991). The present state version of the ISS was used in this study, and asked participants rated how much they currently felt the way described (e.g., 'I feel "sped up" inside'), as adapted by Mansell and Lam (2006). The ISS present state was completed pre- and post-mood induction to assess bipolar-relevant symptoms.

Positive and Negative Affect Schedule (PANAS; Watson et al. 1988). Participants rated 10 adjectives describing Positive Affect (PANAS-PA; e.g., 'inspired') and 10 describing Negative Affect (PANAS-NA; e.g., 'irritable'). The version of the PANAS included in this study was the present state version. For the present state version, internal consistencies were Cronbach's $\alpha = .89$ for PANAS-PA, and $\alpha = .85$ for PANAS-NA. The present state PANAS has demonstrated sensitivity to within-subjects mood fluctuations (Watson et al. 1988). Participants were asked to rate to what extent they currently felt the way described on a 5-point scale from 'Very slightly or not at all' to 'Very much'. The measure was included to assess positive and negative affect pre- and post-mood induction.

Interview. The interview asked questions about the participant's academic experiences (e.g., 'What do you feel are your academic strengths and weaknesses?) and future aspirations (e.g., 'Where do you see yourself in 5 years time?). The questions were split into set (a) and set (b). Participants were randomly allocated to one of two conditions: Half were asked set (a) prior to the mood induction and the other half were asked set (b) prior to the mood induction. The alternative set of questions was administered post-mood induction. Interviews were filmed to allow for inter-rater agreement on behavioural measures. Recordings were made using a Sony Cyber Shot (DSC-W70)

digital camera (7.2 megapixels; MPEG = 640 × 480 with 30 frames per second).

The interview was used to assess objective behavioural indices of bipolar-relevant symptoms. Pressure of speech was calculated by transcribing the first 2 min of each interview and counting the number of words uttered by the participant. The number of words spoken by the experimenter in the first 2 min was also assessed. Proportion of conversation dominated by the participant was determined by calculating the percentage of overall words that were uttered by the participant. These independent behavioural measures were calculated for interviews pre- and post-mood induction. An independent rater performed a word count on a proportion of the recorded interviews, and inter-rater reliability was high; $r = .98$ and .99 for words said by the participant pre- and post-MIP, and $r = .96$ and .98 for words said by the experimenter pre- and post-MIP.

Mood Induction Procedure

Advanced Raven's Progressive Matrices (APM; Raven 1998). Selected items from the APM were used to induce mood. For each item, participants had to select the missing piece of a pattern matrix from eight possible choices. Two booklets of matrices were produced, one for the success condition and the other for the failure condition. Participants were told they were completing an intelligence test, and that this was predictive of academic ability. They were also told that the 'average' student should get at least 50 % correct. Both booklets began with an identical set of instructions, followed by 4 practice items (2 easy, 2 difficult). The success condition included predominately easy items, whereas the failure condition included a higher proportion of difficult items. In the success booklet, the 12 'test' items were comprised of 9 easy and 3 difficult items; in the failure booklet, there were 9 difficult and 3 easy items. A pilot study supported the use of the items originally used by Krohne et al. (2002) for the purposes of mood induction.

Design

Three aspects of the design were counterbalanced. Firstly, to reduce bias, half of the participants were randomly allocated to the success (positive) condition in the MIP, and the rest to the failure (negative) condition. To control for order effects, half the participants completed the self-report assessments before being interviewed, whereas half were interviewed first. The interview was split into two halves (question sets (a) and (b)) to allow for objective behavioural measurements (pressure of speech and conversation dominance) to be taken pre- and post-MIP. Half of the participants were asked questions (a) pre-MIP and questions (b) post-MIP, and half of the participants were asked questions (b) pre-MIP and (a) post-MIP.

Procedure

The study received full ethical approval from the School of Psychological Sciences at the university, in compliance with British Psychological Society guidelines. All participants gave informed consent before completing screening and again on the day of testing. After screening, eligible participants were issued with a battery of questionnaires: HAPPI, HYP, BIS/BAS, DAS, MDQ and HIQ. Depending on random allocation, participants were next given a brief interview regarding their academic experiences and ambitions, or asked to complete the present state versions of the ISS and PANAS. The alternative was then completed before the MIP. Participants were randomly assigned to the success or failure condition.

In both conditions, participants were told they were going to be given a brief intelligence test and given a verbal overview, before being presented with a booklet containing the relevant APMs. Further instructions were included at the beginning of the booklet. Both verbal and written instructions stated that the task was a validated intelligence test that was a reliable and valid predictor of academic success among students, and that the 'average' student would be expected to score at least 50 %. Participants then completed the 4 practice items, after which they were given feedback and asked to predict how many items they would get correct out of 12 test items. Participants were given 30 s to give a verbal answer to each test item, and received feedback after each. At the end, the participant was given their score out of 12, and their score was pointed out on a graph depicting a normal curve with a peak at 50 % correct.

Post-MIP, participants were requested to complete the present state ISS and PANAS, and asked the 2nd set of interview questions. Order of these tasks was again counterbalanced. Finally, all participants were fully debriefed. During this debrief, all participants were informed that the task was split into 'success' and 'failure' conditions, and that it was extremely difficult to obtain 50 % correct response in the 'failure' condition. They were told that the results of the task had no implication for their future academic achievement and given a support sheet.

Results and Discussion

Preliminary Analyses

Data were analysed using SPSS Version 15.0 and 16.0. Data were normally distributed, such that skewness and kurtosis were acceptable, with values not substantially greater than zero, and within the limits of skewness <2 and kurtosis <7 (Tabachnick and Fidell 2001). Examination of normal probability plots and detrended normal probability

plots indicated that most items followed a normal distribution (Tabachnick and Fidell 2001). MANOVA revealed that there were no significant effects of order of assessment (questionnaires first or interview first; $F(14, 35) = 0.87$, *ns*) or order of interview questions (set (a) first or set (b) first; $F(14, 35) = 1.67$, *ns*) for the symptom and mood measures pre- and post-MIP. MANOVA revealed that for participant's predictions of how they would perform on the intelligence test and their actual scores, there was no main effect of Group, $F(2, 59) = 0.76$, *ns*, or Group × Mood interaction, $F(2, 59) = .93$, *ns*. The main effect of Mood was significant, $F(2, 59) = 75.43$, $p < .001$. Follow-up univariate ANOVAs revealed a significant main effect of Mood for score on the intelligence test, $F(1, 60) = 103.93$, $p < .001$, such that the mean score out of 12 was 4.97 in the failure condition and 8.44 in the success condition. This provided further validation for the items used in the MIP, and indicated that the groups did not score differentially on the intelligence test, in either mood induction condition.

Table 1 Results from ANOVAs for group differences in bipolar risk measures and cognitive and motivational processes (Study 1)

Group	Control	Cognitive risk	*F*	η^2
Variable	*M* (SD)	*M* (SD)		
HIQ-hypomania	24.84 (3.96)	26.06 (4.66)	1.27	.20
HIQ-normalising	23.13 (4.25)	26.00 (5.05)	6.08*	.09
HIQ-experience	15.69 (2.10)	16.69 (2.46)	3.06	.05
HYP	13.97 (6.62)	25.03 (9.61)	28.78***	.32
DAS goal attainment	25.34 (5.69)	28.59 (4.90)	6.00*	.09
DAS dependency	13.94 (3.93)	18.72 (3.93)	23.71***	.28
DAS achievement	17.81 (5.87)	23.69 (4.28)	20.93***	.25
BIS	21.63 (3.94)	23.47 (3.59)	3.83	.06
BAS reward responsiveness	16.47 (1.93)	17.44 (1.70)	4.52*	.07
BAS drive	10.25 (1.90)	11.28 (2.57)	3.33	.05
BAS fun seeking	11.53 (2.40)	12.06 (2.61)	0.72	.01
BAS dysregulation	10.69 (2.46)	12.75 (2.18)	12.60***	.17
MDQ	6.16 (3.31)	9.28 (3.04)	15.46***	.20

df = 1, 62; * $p < .05$; ** $p < .01$; *** $p < .001$

Main Analysis

Objective 1: Table 1 displays descriptive statistics and the results of a MANOVA exploring differences between the groups on baseline measures. There were significant overall differences between the groups on these self-report measures, $F(15, 48) = 4.18$, $p < .001$, partial μ^2 (hereafter referred to as μ^2) = .57. The cognitive risk group displayed significantly higher scores on HIQ-Normalising, positive and negative cognitive styles (DAS Goal Attainment, Dependency, and Achievement), reward sensitivity (BAS Reward Responsiveness and Dysregulation), and risk of BD (HYP and MDQ).

As predicted, the cognitive risk group had elevated scores on several measures that have previously been associated with BD, as well as bipolar risk (hypomanic personality) and a history of DSM-IV-TR (APA 2000) hypomanic symptoms as measured by the MDQ. The HYP and MDQ are both established measures of bipolar risk (Eckblad and Chapman 1986; Hirschfeld et al. 2003a, b; Hirschfeld et al. 2000; Kwapil et al. 2000). Further, the DAS subscales, BAS Reward Responsiveness, and to some extent BAS Dysregulation, have all been demonstrated as associated with bipolar disorder in past research (e.g., Holzwarth and Meyer 2006; Meyer et al. 1999; Scott and Pope 2003). The finding that the high group scored higher on these multiple measures provides further validation of the HAPPI as a vulnerability marker for BD, in support of Mansell et al.'s (2007) model.

Objective 2: Table 2 displays the mean scores on the main symptom (ISS Activation and Depression) and mood (PANAS-PA and PANAS-NA) measures both pre- and post-mood induction for both HAPPI groups by mood induction condition.

A series of 2 (Time: Pre- vs. post-MIP) × 2 (Group: control vs. cognitive risk) × 2 (Mood: Failure vs. success) mixed design ANOVAs were conducted. Analyses were run with pre- and post-mood induction ISS Activation and Depression, PANAS-PA and PANAS-NA as repeated measures. Finally, analyses were run with the objective behavioural measures (pressure of speech and proportion of conversation dominated by participant pre- and post-mood induction). Mauchly's test indicated that the assumption of sphericity was tenable in each ANOVA.

ISS Activation: The main effect of Group was significant, $F(1, 60) = 18.29$, $p < .001$, $\eta^2 = .23$. Bonferroni adjusted pairwise comparisons showed that the cognitive risk group scored significantly higher than the control group regardless of time point and mood induction condition. The predicted Time × Mood × Group interaction was not significant, $F(1, 60) = 1.40$, *ns*.

ISS Depression: The Time × Mood × Group interaction approached significance, $F(1, 60) = 2.86$, $p = .09$, $\eta^2 = .05$. There was a main effect of Group, $F(1, 60) = 23.82$, $p < .001$, $\eta^2 = .28$, but this was qualified by a Group × Mood interaction, $F(1, 60) = 5.72$, $p < .05$, $\eta^2 = .09$. Follow-up paired samples *t* tests using an average across the time points to give a general ISS Depression score found that there was a significant difference between the groups in the failure condition only, $t(30) = 5.16$, $p < .01$, such that the cognitive risk group had greater depression scores. The Time × Mood interaction was also significant, $F(1, 60) = 10.37$, $p < .01$, $\eta^2 = .15$.

Table 2 Descriptive statistics for group differences pre- and post-MIP, by mood induction condition (Study 1)

Variable	MIP	Control		Cognitive risk	
		Pre *M* (SD)	Post *M* (SD)	Pre *M* (SD)	Post *M* (SD)
Activation	Success	103.00 (84.17)	88.50 (87.60)	188.38 (108.39)	188.75 (131.39)
	Failure	95.62 (11.03)	94.06 (84.11)	218.44 (102.77)	195.88 (79.46)
Depression	Success	20.00 (35.96)	11.31 (23.83)	36.25 (22.28)	27.31 (24.04)
	Failure	6.56 (15.13)	6.69 (10.81)	44.06 (39.25)	63.44 (40.20)
Pressure of speech	Success	96.25 (22.85)	97.88 (32.43)	113.88 (30.38)	118.19 (30.40)
	Failure	91.31 (31.01)	93.69 (27.98)	100.50 (22.69)	100.44 (32.65)
Speech dominance	Success	67.64 (8.87)	65.31 (13.62)	68.03 (7.99)	71.35 (6.53)
	Failure	62.28 (13.73)	61.79 (11.94)	69.83 (9.84)	68.35 (11.74)
PANAS-PA	Success	30.31 (8.47)	31.63 (10.36)	29.50 (7.95)	30.69 (10.36)
	Failure	25.25 (8.39)	21.88 (7.66)	29.63 (7.59)	26.00 (6.53)
PANAS-NA	Success	11.38 (2.19)	10.75 (1.44)	14.38 (5.67)	13.63 (5.89)
	Failure	11.43 (1.86)	12.75 (3.30)	17.00 (5.89)	18.50 (6.51)

Follow-up paired samples *t* tests for both mood induction conditions revealed that all participants had significantly lower ISS Depression scores at pre-MIP compared to post-MIP in the success condition only ($t(31) = 3.33$, $p < .001$).

PANAS-PA: The predicted Time × Mood × Group interaction was non-significant, $F(1, 60) = .00$, *ns*. There was a significant main effect of Mood, $F(1, 60) = 6.40$, $p < .05$, $\eta^2 = .10$, and this was qualified by a significant Time × Mood interaction, $F(1, 60) = 11.30$, $p < .001$, $\eta^2 = .16$. This finding was further examined using paired samples t tests; PANAS-PA was significantly lower post-MIP in the failure condition only, $t(31) = -3.60$, $p < .001$.

PANAS-NA: There was a significant Group difference, $F(1, 60) = 16.95$, $p < .001$, $\eta^2 = .22$. Bonferroni pairwise comparisons revealed higher levels of PANAS-NA in the cognitive risk group relative to the control group. There was a significant effect of Mood, $F(1, 60) = 5.25$, $p < .05$, $\eta^2 = .08$, but this was succeeded by a Time × Mood interaction, $F(1, 60) = 5.48$, $p < .05$, $\eta^2 = .08$. Follow-up paired samples *t* tests revealed that PANAS-NA was higher in the failure condition post-MIP compared to pre-MIP, but only at trend, $t(31) = 1.96$, $p = .06$.

Pressure of Speech: The predicted Time × Mood × Group interaction was non-significant, $F(1, 60) = 0.12$, *ns*. There was a main effect of Group, $F(1, 60) = 4.63$, $p < .05$, $\eta^2 = .07$. Bonferroni pairwise comparisons indicated that the cognitive risk group used more words than the control group, regardless of mood induction condition or time point.

Proportion of Conversation: The predicted Time × Mood × Group interaction was non-significant, $F(1, 60) = 1.11$, *ns*. Again, there was a significant effect of Group, $F(1, 60) = 5.46$, $p < .05$, $\eta^2 = .08$. The cognitive risk group dominated the conversation more than the control group, irrespective of mood induction condition or time point.

These results show significant differences between the risk and control groups on self-reported activation and objective behavioural measures of hypomanic symptoms, regardless of mood induction condition or time point. This finding suggests that individuals who hold more of the extreme, personal, positive and negative beliefs as measured by the HAPPI have elevated analogue hypomanic symptoms. Depression was only significantly higher in the cognitive risk group in the failure condition, and there was a trend towards this being post-MIP only. This provides tentative evidence that individuals with extreme personalised positive and negative appraisals of activated states are more affected by failure than those who hold less of these beliefs.

In terms of mood manipulation, only in the failure condition did positive and negative affect change from pre- to -post MIP, such that positive affect decreased and negative affect increased. For the success condition, participants reported less depressive symptoms from pre- to post-mood manipulation. These results indicate that the success/failure paradigm was successful at manipulating mood in both groups, to some extent.

The integrative cognitive model (Mansell et al. 2007) proposed that having access to multiple, extreme appraisals of internal states, that are both self-relevant and conflicting, is the key factor driving poor mood regulation in individuals vulnerable to mood swings. It this vein, it was predicted that the cognitive risk group would respond differentially to an MIP compared to the control group. However, the risk group did not demonstrate a significantly greater increase in activation, pressure of speech,

dominance of conversation and positive affect after the mood induction in the success condition, relative to the control group.

Exploratory Analyses: Controlling for Other Measures of Cognitive Style

Objective 3: To explore the independent contribution of cognitive risk (as measured by the HAPPI) to affect and symptoms, a series of 2 (Time) × 2 (Group) × 2 (Mood) mixed ANCOVAs were run for those dependent variables where there were significant group differences. HIQ-Normalising, DAS Goal Attainment, DAS Dependency, and DAS Achievement as covariates, as they were significantly different between the cognitive risk group and control group.

ISS Activation: The main effect of Group was reduced to trend, $F(1, 56) = 3.69$, $p = .06$.

ISS Depression: The main effect of Group retained significance, $F(1, 56) = 5.91$, $p < .05$. The Group × Mood interaction was also still significant, $F(1, 56) = 4.88$, $p < .05$, as was the Time × Mood interaction, $F(1, 56) = 10.37$, $p < .01$. Additionally, DAS Dependency was a significant covariate, $F(1, 56) = 4.49$, $p < .05$.

PANAS-NA: The main effect of Mood remained significant, $F(1, 56) = 4.88$, $p < .05$. However, the main effect of Group was reduced to trend, $F(1, 56) = 3.48$, $p = 0.06$. The Time × Mood interaction was still significant, $F(1, 56) = 5.48$, $p < .05$.

For both Pressure of speech and Proportion of Conversation, when the potentially confounding cognitive measures were entered as covariates, there were no significant effects.

These results show that those scoring higher on the HAPPI still had higher scores on activation and negative affect when controlling for alternative cognitive measures, although these were reduced to trend. There was still a significant difference between groups on depression, qualified by an interaction with mood condition as before. DAS Dependency was also associated with depression. These findings highlight the importance of conviction in extreme beliefs in the development of low mood.

Exploratory Analyses: Controlling for Reward Sensitivity

Objective 3: A series of 2 (Time) × 2 (Group) × 2 (Mood) mixed design ANCOVAs were run, with BAS Reward Responsiveness and BAS Dysregulation as covariates.

For ISS Activation, the main effect of Group retained significance, $F(1, 58) = 9.70$, $p < 0.01$. BAS Reward Responsiveness was a significant covariate, $F(1, 58) = 4.57$, $p < .05$. There were no other significant effects. For ISS Depression, the main effect of Group remained significant, $F(1, 58) = 15.0$, $p < .001$. Further, the Group × Mood interaction was significant, $F(1, 58) = 6.53$, $p < .05$. There was also a significant Time × Mood interaction, $F(1, 60) = 10.37$, $p < .01$. The Time × Mood × Group interaction was still at trend, $F(1, 60) = 2.86$, $p = 0.096$. For PANAS-NA, as before, there were significant main effects of Mood ($F(1, 58) = 4.98$, $p < .05$) and Group ($F(1, 58) = 12.95$, $p < .001$). The Time × Mood interaction retained significance, also, $F(1, 60) = 5.48$, $p < .05$. For both Pressure of speech and Proportion of Conversation, when the potentially confounding personality variables were entered as covariates, there were no significant effects.

These exploratory results show that when reward sensitivity was controlled for, the groups no longer differed on objective behavioural measures of pressured speech and conversation dominance. However, there was still a significant difference between groups on self-reported activation. BAS Reward Responsiveness was also significantly associated with activation, in line with previous research on reward sensitivity and mania risk (e.g., Alloy et al. 2008). The difference between groups also remained significant for depression, and was qualified by an interaction between group and mood induction condition.

Study 1 investigated associations between cognitive risk, mood, and bipolar-relevant symptoms in an artificial setting with a short time-frame. Mansell et al.'s (2007) model would suggest that the escalating cycle of symptoms would be instigated by a change in internal state and involve extreme appraisal of this change, with a reciprocal impact on behaviour and further changes to internal state. It is possible that this was not achievable in a short laboratory session; as such, Study 2 explored whether a cognitive risk group had higher rates of prospective bipolar-relevant symptoms and mood in a real world setting.

Study 2

Introduction

To complement the cross-sectional, experimental findings of Study 1, this study aimed to investigate real world differences between a group with high levels of cognitive appraisals and a group with low levels of cognitive appraisals on prospective bipolar symptoms, mood, and variability in these measures over 1 week.

Diary studies allow the examination of the variation in symptoms and mood on a day-to-day basis in a real world setting, providing ecological validity (see Bolger et al. 2003, for a review). This methodology has been previously used in BD research to explore relationships between vulnerability measures and fluctuations in mood over time

(Hofmann and Meyer 2006). Participants in the current study completed a 7-day diary comprised of analogue bipolar symptom and mood scales. It was predicted that individuals in the cognitive risk group would display significantly higher scores on symptom and mood measures, as well as variability in these measures, across 7 consecutive days.

Method

Participants

Participants were recruited as in Study 1, and again, groups were defined by the distribution of scores on the HAPPI, where the cognitive risk was defined by the 90th percentile (HAPPI mean $\geq$ 48) and the control group by $\leq$0.5 SD above the mean = 36). In the final sample, there were 15 participants in the cognitive risk group (10 female) with a mean age of 20.27 years (SD = 3.47) and the control group (11 female), with a mean age of 19.60 years (SD = 2.85). Independent *t* tests revealed no significant difference in age between groups, $t(28) = .58$, *ns*. Chi squared analysis indicated no significant difference in gender ratio, Pearson; $\chi^2(1) = .16$, *ns*, Yate's continuity correction; $\chi^2(1) = .00$, *ns*.

Measures

The HAPPI, ISS (Bauer et al. 1991), and PANAS (Watson et al. 1988) were all used, as in Study 1. Beyond the baseline ISS, the wording of the items was modified in the current study so that the scale began with 'Since I last completed this scale…', in order to assess symptoms between time points.

Procedure

Participants completed the HAPPI online for another study, and were asked if they would be willing to be contacted about related research in the future. Those scoring within the relevant ranges on the HAPPI were invited to meet with the experimenter to acquire a diary. Full research objectives were not initially communicated, to reduce the influence of demand effects. Participants were informed of the importance of adhering to the requested times when completing the diary and asked if they would like a reminder via text message or telephone call to continue completing the diary. This was optional but the majority of participants chose to do so.

After giving informed consent, participants were asked to complete a diary twice-daily, at 3 p.m. and 10 p.m., over 7 consecutive days. The ISS and PANAS required completion at all time points. Other measures not discussed in the current study were also taken. The order of these scales was counterbalanced between participants to control for order effects. A meeting was arranged for the participants to return completed diaries, allowing any questions or concerns to be communicated and addressed, and the necessary information or support provided. In addition, a debriefing e-mail, including details of available support services, was sent out 1 week after data collection was completed.

Results and Discussion

Objective 1: Mean scores were calculated over the 14 time points for the ISS and PANAS. Missing items were attained by pro-rating. MANOVA was conducted to investigate group differences on mean mood (PANAS) and analogue bipolar symptoms (ISS Activation, Conflict, Depression and Well-being) across the 14 time points. There were significant overall differences between the groups, $F(6, 23) = 5.66$, $p < .001$, $\mu^2 = .60$. Follow-up univariate ANOVAs were significant for PANAS-NA, ISS Activation, ISS Conflict, and ISS Depression (see Table 3). The cognitive risk group had higher scores, as would be expected in line with Mansell et al.'s (2007) model, which proposed that having greater conviction in the extreme personalised appraisals measured by the HAPPI would confer greater vulnerability to bipolar symptomatology.

Objective 2: Variability in ISS and PANAS scores was calculated as the mean absolute change from one time point to the next, across all 14 time points. This was considered a sensitive measure of fluctuation over time as it assessed the average moment-to-moment change in participants' mood and symptoms changed across the 7 days as used in diary studies exploring self-esteem fluctuation (e.g., Thewissen et al. 2008). MANOVA revealed no significant differences in symptom or mood variability between groups, $F(6, 23) = 1.41$, *ns*.

Table 3 Results from ANOVAs for group differences in ISS scores and mood (Study 2)

Variable	Group		*F*	η^2
	Control *M* (SD)	Cognitive risk *M* (SD)		
PANAS-PA	26.34 (6.19)	26.07 (7.33)	0.01	.00
PANAS-NA	13.92 (2.77)	19.23 (7.35)	6.85*	.20
ISS activation	108.58 (65.5)	196.64 (92.19)	9.10**	.25
ISS conflict	96.54 (50.32)	166.77 (76.60)	8.65**	.24
ISS depression	25.47 (20.93)	65.96 (30.42)	18.03***	.39
ISS well-being	157.38 (51.59)	133.32 (54.05)	1.56	.05

df = 1, 28; * $p < .05$; ** $p < .01$; *** $p < .001$

General Discussion

Study 1 investigated whether students at cognitive risk of BD (1) had higher scores on measures of reward sensitivity, bipolar risk, and cognitive styles associated with BD, (2) were more susceptible to symptom and mood changes after a mood induction, and (3) displayed higher rates of bipolar-relevant symptoms and mood states, relative to a control group. Study 2 investigated whether students at cognitive risk of BD demonstrated (1) higher bipolar-relevant symptoms and mood states over 1 week and (2) greater variability in symptoms and mood over 1 week, relative to a control group.

In Study 1, individuals with a putative cognitive vulnerability to BD displayed elevated scores on established risk measures (hypomanic personality and history of hypomanic symptoms), as well as other constructs associated with vulnerability to BD including reward sensitivity and dysfunctional attitudes. That those with higher HAPPI scores also had elevated scores on several vulnerability measures, pertaining to motivational and cognitive processes as well as a risk of BD, provides support for the construct validity of the HAPPI and provides tentative support for Mansell et al.'s (2007) model. However, there were no group differences on positive self appraisals, as would be expected given the similarity between the positive appraisals of hypomania-relevant appraisals measured by the HIQ-Hypomania subscale and the HAPPI.

Further, the cognitive risk group had heightened negative affect and hypomania. The latter was true for self-reported symptoms, as assessed by the activation subscale of the ISS, and objective behavioural measures of pressured speech and conversation dominance. The behavioural measures represent unbiased indices of hypomanic symptoms to complement the self-report measure, which is a strength of the current research. Additionally, the self-report measure ISS Activation has been associated with clinician-made ratings of mania (Bauer et al. 1991, 2000). It has been argued that activation is a core feature of hypomania (Benazzi 2007), and DSM-IV-TR criteria (APA 2000) includes increased rate and quantity of speech as symptoms of hypomania.

It was predicted that, compared to control individuals, the cognitive risk group would experience a greater increase in hypomania and positive affect in response to success in the mood induction condition. In this study, students with more conviction in these appraisals did not respond differentially to mood manipulation, compared to the control group (students with relatively low belief in the appraisals). The HAPPI has previously been found to predict heightened symptoms prospectively in both student and clinical samples (Dodd et al. 2011a, c). It is possible that the HAPPI may not predict an instantaneous reaction to a mood induction but instead would predict reactions over a longer time period, where individuals develop high moods through a vicious cycle of appraisal, engagement in ascent behaviours, and re-appraisal of subsequent changes to internal state.

Further, relationships between (hypo)mania and goal-attainment or goal-striving life events have been established (e.g., Johnson et al. 2000; Nusslock et al. 2007), but the cognitive risk group did not have heightened expectancies of their own performance on the task relative to controls, in concordance with the finding that hypomania was unrelated to baseline success expectancy in a previous study (Johnson et al. 2005). Taking account of the fact that cognitive risk individuals did not differentially expect success on the task compared to the control group, it may be that the success experienced by participants in the current study was not sufficiently rewarding or relevant to personal goals to induce increased symptoms and elevated positive mood. Further, it may be that the HAPPI is associated with changes in symptoms over a longer temporal period, as opposed to immediately following a mood induction.

When controlling for other cognitive measures of dysfunctional attitudes and normalising appraisals, which were both also significantly raised in the cognitive group, group differences on activation and negative affect were reduced to trend, and for depression retained significance, although only in the failure condition as before. Group differences on activation, depression and negative affect remained significant when controlling for reward sensitivity. This is an important finding in keeping with previous research (Mansell et al. 2008; Dodd et al. 2010, 2011c) where the HAPPI has uniquely predicted bipolar-relevant symptoms over and above reward sensitivity and other cognitive measures among students and clinical samples. However, group differences were no longer significant for behavioural measures of pressured speech and conversation dominance when controlling for reward sensitivity or when controlling for depressogenic cognitive styles. Differences between the groups on these alternative motivational and cognitive measures may have accounted for the group differences on observed behavioural symptoms, in keeping with findings that vulnerability to BD is multifaceted and likely to arise from a combination of multiple psychological factors (Johnson and Jones 2009). This study is unique in its use of this particular way of assessing analogue bipolar symptomatology and it is worth investigating this in more detail in the future.

Study 2 reported that the cognitive risk group had greater levels of hypomania and depression, as well as negative affect, across the subsequent 7-day period. These results suggest that individuals with more multiple, extreme, self-relevant positive and negative beliefs about

internal states are more vulnerable to negative affect and analogue bipolar symptoms, measured prospectively. However, contrary to predictions, there were no significant differences between the groups on variability in mood or symptoms across the week. A previous study (Hofmann and Meyer 2006) found greater fluctuations in mood among individuals with high scores on the hypomanic personality scale, over a 28-day period. It may be that 1 week was not a long enough period to detect differences between the groups in mood fluctuation.

Taken together, the findings of these studies suggest that individuals with stronger conviction in extreme, personalised, positive and negative appraisals of internal states are more prone to bipolar-relevant symptoms and mood. The studies use complementary experimental and diary methodology to assess whether the appraisals measured by the HAPPI confer heightened cognitive risk for bipolar disorder relevant experiences. This supports the integrative cognitive model, which proposed that having extreme positive and negative appraisals of changes to internal states prompts counterproductive responses, driving activation levels upwards or downwards, depending on the activating or deactivating nature of the appraisal and subsequent response. This would reduce the individual's ability to reappraise internal states and respond in a normalising manner, leading to poor affect regulation. These results also provide experimental evidence that the HAPPI is a cognitive risk measure for BD that could be used in early intervention for to identify those vulnerable to BD.

Clinical Implications

The development of psychotherapeutic interventions for psychological disorders has been influenced by the development of cognitive models specific to these disorders, and the formulation of measures of beliefs central to the models (for example the interpretation of bodily sensations in cognitive therapy for panic disorder: Clark 1986; Clark et al. 1997, 1999). The Think Effectively About Mood Swings (TEAMS) approach to cognitive therapy for BD was developed from Mansell et al.'s (2007) cognitive model, and utilises the HAPPI as a clinical tool for formulation and measuring change. TEAMS is promising; in a clinical case series, clients displayed improvements in symptoms and functioning, and therapy was considered feasible and acceptable (Searson et al. 2012). That cognitive risk, as measured by the HAPPI, is associated with risk of BD and bipolar-relevant symptoms and mood in a student sample provides further support for its potential clinical utility. This has implications for early detection and intervention for problematic cognitive styles amongst those at risk of developing BD.

Limitations and Future Directions

No power calculations were undertaken, and sample size was based on previous similar research (e.g., Taylor and Mansell 2008). The research may therefore be limited by a lack of power, particularly in light of the number of variables analysed. However, we still found significant differences between the groups on several key measures conferring vulnerability to BD. The cognitive risk group also displayed higher bipolar relevant-symptoms and mood, and this was still the case when controlling for covariates including reward sensitivity and depressogenic cognitive styles; effects reduced to trend may be an artefact of the small sample size.

The mood induction appeared to successfully manipulate mood in both conditions, such that depression was significantly reduced after a success, while positive and negative affect changed in the expected direction after experiencing failure. However, the groups were not differentially affected by the mood induction. Future research could utilise MIPs of greater relevance for vulnerability to BD, for example incorporating a reward or a more personalised goal. In similar undergraduate samples, alternative MIPs have successfully manipulated mood. These included increased activation after a musical mood induction (Taylor and Mansell 2008), and increased happiness after false success feedback on a cognitive word task (Trevisani et al. 2008). The latter MIP was similar to that of the current study in that individuals were told the average student would score within a particular range, and participants were told at the end of the task they had far exceeded this average score. However, there was also a reward element to the task, where participants received a greater number of experiment credits for gaining a high score. Thus, it could be argued that the task adopted in the current study was not rewarding enough to induce increases in positive affect and hypomania-relevant symptoms such as activation. Further, while the success/failure paradigm was chosen in light of associations between symptom exacerbations and goal-attainment or goal-striving life events (Johnson et al. 2000; Nusslock et al. 2007), previous studies have involved individuals with a bipolar spectrum diagnosis and used more methodologically robust and ecologically valid longitudinal designs, whereas the current study recruited an analogue sample without conducting a research diagnostic interview and a mood induction under artificial, experimental conditions. Appraisals of changes to internal state (including mood) are a key driving factor in the development of symptoms according to the cognitive model (Mansell et al. 2007), and under the experimental conditions in this study, it is highly plausible that students were less likely to appraise any change in mood in an extreme, personalised way, as they knew they were taking

part in an experiment. Future studies should disentangle the role of appraisals in the development of mood changes more directly using diary methods to test whether appraisals predict subsequent mood changes, or experimentally by manipulating appraisals and assessing subsequent mood change. It would also be interesting to investigate whether experimentally manipulating appraisal style results in more engagement in activating or deactivating behaviours and increases in bipolar symptomatology.

In Study 1, the cognitive risk group had elevated scores on several measures representing bipolar risk, reward sensitivity, and cognitive vulnerability to BD. There is considerable overlap between the different measures employed and models of BD. For example, goal attainment beliefs such as those measured by the DAS could be considered BAS-relevant cognitive styles (Wright and Lam 2004), as could facets of the HAPPI (e.g., 'When I am excited, the world is full of unlimited opportunities for me'). Group differences on behavioural measures of hypomania were redundant when covarying for other vulnerability measures. However, self-reported activation, depression and negative affect were still higher in the cognitive risk group when controlling for these measures, suggesting that the HAPPI makes a unique contribution to these symptoms. Unlike these alternative measures, the HAPPI was developed specifically to assess a cognitive style associated with mood swings and BD, and these are amenable to change in cognitive therapy for BD (see Clinical Implications below). A fine-grained investigation of the longitudinal association between appraisals, ascent/descent behaviours, and symptoms is merited, alongside other measures of cognitive and response style known to be associated with vulnerability to bipolar disorder (Johnson and Jones 2009). This is because (1) bipolar disorder is a multifaceted condition and it is likely that a number of factors contribute to vulnerability to bipolar disorder (Johnson and Jones 2009) and (2) it may also be the case that extreme, personalised appraisals are only related to mood swings over time and in interaction with engaging in attempts to enhance or control internal states, and this study did not employ a measure of behavioural response style. Mansell et al.'s (2007) model suggests that a behavioural component is crucial for the development of bipolar symptoms, and further work suggests a key role for ruminative response styles to both positive and negative affect (e.g., Johnson et al. 2008).

Although the results of Study 2 were promising with respect to validation of the HAPPI as a risk measure of developing bipolar symptoms, the sample size was too small to make any definitive conclusions. Further research, as outlined above, should explore the relationships between appraisals and other bipolar vulnerability measures longitudinally. In addition, in line with research done by Alloy and colleagues in the US involving whether students with a more over-sensitive BAS are more prone to bipolar mood episodes in the future (e.g., Alloy et al. 2008), it would be interesting to explore whether cognitive risk as measured by the HAPPI predicts future mood episodes in students. It would have been interesting to have measured life events within the diary, particularly relevant, goal-related events, in our exploration of whether heightened cognitive risk conferred vulnerability to heightened symptoms and mood.

A further limitation relevant to both studies is that we did not conduct diagnostic interviews but based risk solely on cognitive style. Therefore, we do not know the diagnostic status of the participants, nor can we confirm that no participants were currently experiencing an episode of depression or hypomania (although we did screen using BDI and BMRS in Study 1).

The current research explored cognitive risk of developing mood swings and BD in a non-clinical sample. The cognitive model (Mansell et al. 2007) proposed that interpreting changes to internal states as signifying extreme personal meaning, and having access to contradictory interpretations of the same internal state, would maintain and exacerbate mood fluctuations on a continuum, justifying the use of an analogue sample. The present results provide tentative evidence for the integrative cognitive model, as the multiple, extreme and personalised appraisals measured by the HAPPI appear to confer risk of analogue bipolar symptoms and negative mood, both cross-sectionally and prospectively in the short-term.

Acknowledgments This work was undertaken at the University of Manchester, UK, and forms part of the first author's PhD thesis. The research was funded by the School of Psychological Sciences and Faculty of Medical and Human Sciences, University of Manchester, UK. Warren Mansell is supported by the ESRC Emotion Regulation of Others and Self (EROS) Research Grant. Thank you to Heinz Walter Krohne.

Conflict of interest None.

References

Alatiq, Y., Williams, J. M. G., & Goodwin, G. M. (2010). Dysfunctional beliefs in bipolar disorder: Hypomanic vs. depressive attitudes. *Journal of Affective Disorders, 122*(3), 294–300.

Alloy, L. B., Abramson, L. Y., Walshaw, P. D., Cogswell, A., Grandin, L., Hughes, M. E., et al. (2008). Behavioral approach system and behavioral inhibition system sensitivities and bipolar spectrum disorders: Prospective prediction of bipolar mood episodes. *Bipolar Disorders, 10*(2), 310–322.

Alloy, L. B., Abramson, L. Y., Walshaw, P. D., Cogswell, A., Smith, J. M., Neeren, A. N., et al. (2006). Behavioural approach system (BAS) sensitivity and bipolar spectrum disorders: A retrospective and concurrent behavioural high-risk design. *Motivation and Emotion, 30*(2), 143–155.

Alloy, L. B., Bender, R. E., Whitehouse, W. G., Wagner, C., Liu, R. T., Grant, D., et al. (2012). High Behavioral Approach System (BAS) sensitivity, reward responsiveness, and goal-striving predict first onset of bipolar spectrum disorders: A prospective behavioral high-risk design. *Journal of Abnormal Psychology, 121*(2), 339–351.

American Psychiatric Association (APA). (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

Angst, J., & Gamma, A. (2002). A new bipolar spectrum concept: A brief review. *Bipolar Disorders, 4*, 11–14.

Bauer, M. S., Crits-Christoph, P., Ball, W. A., Dewees, E., McAllister, T., Alahi, P., et al. (1991). Independent assessment of manic and depressive symptoms by self-rating. Scale characteristics and implications for the study of mania. *Archives of General Psychiatry, 48*(9), 807–812.

Bauer, M. S., Vojta, C., Kinosian, B., Altshuler, L., & Glick, H. (2000). The Internal State Scale: Replication of its discriminating abilities in a multisite, public sector sample. *Bipolar Disorders, 2*(4), 340–346.

Bech, P., Rafaelsen, O. J., Kramp, P., & Bolwig, T. G. (1978). The mania rating scale: Scale construction and inter-observer agreement. *Neuropharmacology, 17*(6), 430–431.

Beck, A. T. (1996). *Manual for the Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation.

Benazzi, F. (2007). Is overactivity the core feature of hypomania in bipolar II disorder? *Psychopathology, 40*(1), 54–60.

Bentall, R. P., Myin-Germeys, I., Smith, A., Knowles, R., Jones, S. H., Smith, T., et al. (2011). Hypomania personality, stability of self-esteem and response styles to negative mood. *Clinical Psychology & Psychotherapy, 18*(5), 397–410.

Bolger, N., Davis, A., & Rafaeli, E. (2003). Diary methods: Capturing life as it is lived. *Annual Review of Psychology, 54*, 579–616.

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS Scales. *Journal of Personality and Social Psychology, 67*(2), 319–333.

Clark, D. M. (1986). A cognitive approach to panic. *Behaviour Research and Therapy, 24*(4), 461–470.

Clark, D. M., Salkovskis, P. M., Hackmann, A., Wells, A., Ludgate, J., & Gelder, M. (1999). Brief cognitive therapy for panic disorder: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 67*(4), 583–589.

Clark, D. M., Salkovskis, P. M., Ost, L., Breitholtz, E., Koehler, K. A., Westling, B. E., et al. (1997). Misinterpretation of body sensations in panic disorder. *Journal of Consulting and Clinical Psychology, 65*(2), 201–213.

Cox, B. J., Enns, M. W., & Larsen, D. K. (2001). The continuity of depression symptoms: Use of cluster analysis for profile identification in patient and student samples. *Journal of Affective Disorders, 65*, 67–73.

Depue, R. A., & Iacono, W. G. (1989). Neurobehavioral aspects of affective disorders. *Annual Review of Psychology, 40*(1), 457–492.

Dodd, A. L., Mansell, W., Bentall, R. P., & Tai, S. (2011a). Do extreme beliefs about internal states predict mood swings in an analogue sample? *Cognitive Therapy & Research, 35*(6), 497–504.

Dodd, A. L., Mansell, W., Morrison, A. P., & Tai, S. (2011b). Factor structure of the Hypomanic Attitudes and Positive Predictions Inventory and associations with analogue symptoms in a student sample. *Personality and Individual Differences, 50*(3), 349–354.

Dodd, A. L., Mansell, W., Morrison, A. P., & Tai, S. (2011c). Extreme appraisals of internal states and bipolar symptoms: The Hypomanic Attitudes and Positive Predictions Inventory. *Psychological Assessment, 23*(3), 635–645.

Dodd, A. L., Mansell, W., Sadhnani, V., Morrison, A. P., & Tai, S. (2010). Principal components analysis of the Hypomanic Attitudes and Positive Predictions Inventory & associations with measures of personality, cognitive style & analogue symptoms in a student sample. *Behavioural and Cognitive Psychotherapy, 38*, 15–33.

Eckblad, M., & Chapman, L. J. (1986). Development and validation of a scale for hypomanic personality. *Journal of Abnormal Psychology, 95*(3), 214–222.

Eisner, L. R., Johnson, S. L., & Carver, C. S. (2008). Cognitive responses to failure and success relate uniquely to bipolar depression versus mania. *Journal of Abnormal Psychology, 117*(1), 154–163.

Gerrards-Hesse, A., Spies, K., & Hesse, F. W. (1994). Experimental inductions of emotional states and their effectiveness: A review. *British Journal of Psychology, 85*, 55–78.

Harvey, A. G., Watkins, E., Mansell, W., & Shafran, R. (2004). *Cognitive behavioural processes across psychological disorders: A transdiagnostic approach to research and treatment*. New York: Oxford University Press.

Hirschfeld, R. M. A., Holzer, C., Calabrese, J. R., Weissman, M., Reed, M., Davies, M., et al. (2003a). Validity of the mood disorder questionnaire: A general population study. *American Journal of Psychiatry, 160*(1), 178–180.

Hirschfeld, R. M. A., Lewis, L., & Vornik, L. A. (2003b). Perceptions and impact of bipolar disorder: How far have we really come? Results of the National Depressive and Manic-depressive Association 2000 survey of individuals with bipolar disorder. *Journal of Clinical Psychiatry, 64*(2), 161–174.

Hirschfeld, R. M. A., Williams, J. B. W., Spitzer, R. L., Calabrese, J. R., Flynn, L., Keck, P. E, Jr, et al. (2000). Development and validation of a screening instrument for bipolar spectrum disorder: The Mood Disorder Questionnaire. *American Journal of Psychiatry, 157*(11), 1873–1875.

Hofmann, B. U., & Meyer, T. D. (2006). Mood fluctuations in people putatively at risk for bipolar disorders. *British Journal of Clinical Psychology, 45*(1), 105–110.

Holzwarth, K., & Meyer, T. D. (2006). The dysregulation of the "behavioural activation system": An independent dimension. *Personality and Individual Differences, 41*(2), 319–328.

Johnson, S. L., Fulford, D., & Carver, C. S. (2012). The Double-Edged Sword of Goal Engagement: Consequences of Goal Pursuit in Bipolar Disorder. *Clinical Psychology & Psychotherapy, 19*(4), 351–362.

Johnson, S. L., & Jones, S. H. (2009). Cognitive correlates of mania risk: Are responses to success, positive mood, and manic symptoms distinct or overlapping? *Journal of Clinical Psychology, 65*(9), 891–905.

Johnson, S. L., McKenzie, G., & McMurrich, S. (2008). Ruminative responses to positive and negative affect among students diagnosed with bipolar disorder and major depressive disorder. *Cognitive Therapy and Research, 32*(5), 702–713.

Johnson, S. L., Ruggero, C. L., & Carver, C. S. (2005). Cognitive, behavioural, and affective responses to reward: Links with hypomanic symptoms. *Journal of Social and Clinical Psychology, 24*(6), 894–906.

Johnson, S. L., Sandrow, D., Meyer, B., Winters, R., Miller, I., Solomon, D., et al. (2000). Increases in manic symptoms after life events involving goal attainment. *Journal of Abnormal Psychology, 109*(4), 721–727.

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

Jones, S. H., & Day, C. (2008). Self appraisal and behavioural activation in the prediction of hypomanic personality and

depressive symptoms. *Personality and Individual Differences, 45*(7), 643–648.

Jones, S. H., Mansell, W., & Waller, L. (2006). Appraisal of hypomania-relevant experiences: Development of a questionnaire to assess positive self-dispositional appraisals in bipolar and behavioural high risk samples. *Journal of Affective Disorders, 93*(1–3), 19–28.

Krohne, H. W., Pieper, M., Knoll, N., & Breimer, N. (2002). The cognitive regulation of emotions: The role of success versus failure experience and coping dispositions. *Cognition and Emotion, 16*, 217–243.

Kwapil, T. R., Miller, M. B., Zinser, M. C., Chapman, L. J., Chapman, J., & Eckblad, M. (2000). A longitudinal study of high scorers on the hypomanic personality scale. *Journal of Abnormal Psychology, 109*(2), 222–226.

Lam, D. H., Wright, K., & Smith, N. (2004). Dysfunctional assumptions in bipolar disorder. *Journal of Affective Disorders, 79*(1–3), 193–199.

Mansell, W. (2006). The Hypomanic Attitudes and Positive Predictions Inventory (HAPPI): A pilot study to select cognitions that are elevated in individuals with bipolar disorder compared to non-clinical controls. *Behavioural and Cognitive Psychotherapy, 34*, 467–476.

Mansell, W., Colom, F., & Scott, J. (2005). The nature and treatment of depression in bipolar disorder: A review and implications for future psychological investigation. *Clinical Psychology Review, 25*(8), 1076–1100.

Mansell, W., & Jones, S. H. (2006). The Brief-HAPPI: A questionnaire to assess cognitions that distinguish between individuals with a diagnosis of bipolar disorder and non-clinical controls. *Journal of Affective Disorders, 93*(1–3), 29–34.

Mansell, W., & Lam, D. (2006). "I Won't Do What You Tell Me!": Elevated mood and the assessment of advice-taking in euthymic bipolar I disorder. *Behaviour Therapy and Research, 44*(12), 1787–1801.

Mansell, W., Morrison, A. P., Reid, G., Lowens, I., & Tai, S. (2007). The interpretation of, and responses to, changes in internal states: An integrative cognitive model of mood swings and bipolar disorders. *Behavioural and Cognitive Psychotherapy, 35*(5), 515–539.

Mansell, W., Paszek, G., Seal, K., Pedley, R., Jones, S. A., Thomas, N., et al. (2011). Extreme appraisals of internal states in bipolar I disorder: A multiple control group study. *Cognitive Therapy and Research, 35*, 87–97.

Mansell, W., Rigby, Z., Tai, S., & Lowe, C. (2008). Do current beliefs predict hypomanic symptoms beyond personality style? Factor analysis of the Hypomanic Attitudes and Positive Predictions Inventory (HAPPI) and its association with hypomanic symptoms in a student population. *Journal of Clinical Psychology, 64*(4), 450–465.

Mansell, W., & Scott, J. (2006). Dysfunctional beliefs in individuals with bipolar disorder. In R. Bentall & S. E. Jones (Eds.), *The psychology of bipolar disorder: New developments and research strategies* (pp. 73–90). New York: Oxford University Press.

Martin, M. (1990). On the induction of mood. *Clinical Psychology Review, 10*(6), 669–697.

Meyer, T. D. (2002). The Hypomanic Personality Scale, the Big Five, and their relationship to depression and mania. *Personality and Individual Differences, 32*(4), 649–660.

Meyer, T. D., & Hautzinger, M. (2003). Screening for bipolar disorders using the Hypomanic Personality Scale. *Journal of Affective Disorders, 75*(2), 149–154.

Meyer, B., Johnson, S. L., & Carver, C. S. (1999). Exploring behavioral activation and inhibition sensitivities among college students at risk for bipolar spectrum symptomatology. *Journal of Psychopathology and Behavioral Assessment, 21*(4), 275–292.

Meyer, B., Johnson, S. L., & Winters, R. (2001). Responsiveness to threat and incentive in bipolar disorder: Relations of the BIS/BAS Scales with symptoms. *Journal of Psychopathology and Behavioral Assessment, 23*(3), 133–143.

Morrison, A. P. (2001). The interpretation of instrusions in psychosis: An integrative cognitive approach to hallucinations and delusions. *Behavioural and Cognitive Psychotherapy, 29*(3), 257–276.

Nusslock, R., Abramson, L. Y., Harmon-Jones, E., Alloy, L. B., & Hogan, M. E. (2007). The effect of a goal striving life event and the onset of bipolar episodes: Perspective from the behavioral approach system dysregulation theory. *Journal of Abnormal Psychology, 116*, 105–115.

Power, M. J., Katz, R., McGuffin, P., Duggan, C. F., Lam, D., & Beck, A. T. (1994). The Dysfunctional Attitude Scale (DAS): A comparison of forms A and B and proposals for a new subscaled version. *Journal of Research in Personality, 28*(3), 263–276.

Raven, J. C. (1998). *Advanced progressive matrices*. Oxford: OPP Ltd.

Scott, J., & Pope, M. (2003). Cognitive styles in individuals with bipolar disorders. *Psychological Medicine, 33*(06), 1081–1088.

Searson, R., Mansell, W., Lowens, I., & Tai, S. (2012). Think Effectively About Mood Swings (TEAMS): A case series of cognitive–behavioural therapy for bipolar disorders. *Journal of Behaviour Therapy & Experimental Psychiatry, 43*(2), 770–779.

Tabachnick, B. G., & Fidell, L. S. (2001). *Using multivariate statistics* (4th ed.). Boston: Allyn and Bacon.

Taylor, P., & Mansell, W. (2008). "I Get So Energetic and Dominating!" A study of hypomanic personality and conflicting self-perception during activated states in a co-operative task. *Personality and Individual Differences, 45*(6), 483–487.

Thewissen, V., Bentall, R. P., Lecomte, T., van Os, J., & Myin-Germseys, I. (2008). Fluctuations in self-esteem and paranoia in the context of daily life. *Journal of Abnormal Psychology, 117*(1), 143–153.

Trevisani, D. P., Johnson, S. L., & Carver, C. (2008). Positive mood induction and facial affect recognition among students at risk for mania. *Cognitive Therapy and Research, 32*(5), 639–650.

Urosevic, S., Abramson, L. Y., Harmon-Jones, E., & Alloy, L. B. (2008). Dysregulation of the behavioral approach system (BAS) in bipolar spectrum disorders: Review of theory and evidence. *Clinical Psychology Review, 28*(7), 1188–1205.

Van der Gucht, E., Morriss, R., Lancaster, G., Kinderman, P., & Bentall, R. P. (2009). Psychological processes in bipolar affective disorder: Negative cognitive style and reward processing. *British Journal of Psychiatry, 194*, 146–151.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*(6), 1063–1070.

Weissman, A., & Beck, A. T. (1978). *Development and validation of the Dysfunctional Attitudes Scale: A preliminary investigation*. Paper presented at the annual meeting of the association for advancement of behavior therapy, Chicago.

Westermann, R. (1996). Relative effectiveness and validity of mood induction procedures: A meta-analysis. *European Journal of Social Psychology, 26*(4), 557.

Wright, K., & Lam, D. (2004). Bipolar affective disorder: Current perspectives on psychological theory and treatment. In M. J. Power (Ed.), *Mood disorders: A handbook of science and practice* (pp. 235–246). Chichester: Wiley.