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Preliminary communication

Iowa gambling task performance in euthymic bipolar I disorder: A meta-analysis and empirical study

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ABSTRACT

Background: The Iowa Gambling Task (IGT) has been recommended as an index of reward sensitivity, which is elevated in bipolar disorder. We conducted a meta-analysis of IGT performance in euthymic bipolar I disorder compared with control participants. Findings indicated that people with bipolar disorder make more risky choices than control participants, though the effect is small ($g=0.35$). It is not clear which of the many processes involved in IGT performance are involved in producing the observed group difference.

Methods: Fifty-five euthymic people with bipolar disorder and 39 control participants completed the IGT. The Expectancy Valence Model was used to examine differences in IGT. We also examined whether variation in IGT performance within the bipolar group was related to current mood, illness course, impulsivity, or demographics.

Results: Bipolar and control groups did not differ on the total number of risky choices, rate of learning, or any of the parameters of the Expectancy Valence Model. IGT performance in bipolar disorder was not related to any of the examined individual differences.

Limitations: It is possible that there are group differences that are too small to detect at our sample size or that are not amenable to study via the Expectancy Valence Model.

Conclusions: We were unable to identify group differences on the IGT or correlates of IGT performance within bipolar disorder. Though the IGT may serve as a useful model for decision-making, its structure may make it unsuitable for behavioral assessment of reward sensitivity independent of punishment sensitivity.

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1. Introduction

Bipolar I disorder is a severe psychiatric condition defined by episodes of mania. The reward system has been a focus of psychological research on mania etiology (Johnson et al., 2012b). Over 20 years ago, Depue and Iacono (1989) argued that mania symptoms are consistent with an overactive pursuit of and response to reward and hypothesized that mania vulnerability involves elevated reward sensitivity—a tendency to show increased behavioral, affective, and cognitive responses to reward-relevant stimuli (Urosevic et al., 2008; Johnson et al., 2012b). Consistent with this, researchers have found that manic symptoms are more likely after life events involving goal attainment (Nusslock et al., 2007; Johnson et al., 2008). Self-reported sensitivity to reward, as measured by the Behavioral Activation System (BAS) scales (Carver and White, 1994), is elevated among people with bipolar disorder (Meyer et al., 2001) and is associated with a more severe course of mania (Meyer et al., 2001), the conversion of bipolar spectrum disorders to more severe forms of disorder (Alloy et al., 2012), and the onset of bipolar spectrum

disorders (Alloy et al., 2008). Recent work has attempted to identify differences between those with and without bipolar disorder in responses to reward-relevant laboratory tasks (see Johnson et al., 2012b for review).

Recently, the National Institute of Mental Health (NIMH) RDoC workshop on Positive Valence Systems recommended the Iowa Gambling Task (IGT; Bechara et al., 1994) as a measure of approach motivation (NIMH, 2011). In the IGT, participants must choose cards from four decks. With each card selected, the participant gains money; for some cards, the participant gains money but also has to pay a penalty that may be larger than the amount gained. Two of the decks are “risky,” meaning that they provide larger payoffs but are more likely to lead to large losses, and two of the decks are “safe,” with smaller payoffs but smaller risks. In the long run, participants who play the risky decks will lose money, but participants who play the safe decks will gain money. Successful participants learn to choose cards from the safe decks over the course of the task. There are many ways to interpret IGT performance, but one interpretation is that those who select cards from risky decks are willing to hazard losses in the pursuit of larger rewards—a profile that may stem from greater reward sensitivity.

Several researchers have used the IGT with bipolar samples. Findings suggest that people experiencing acute mania and

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depression select more cards from risky decks than do control participants (Rubinsztein et al., 2006; Adida et al., 2011). However, reward models posit that people with bipolar disorder display elevated reward sensitivity even during remission. Some studies of IGT behavior among euthymic people with bipolar I disorder have found elevated risk taking among euthymic bipolar I participants (Adida et al., 2011; Malloy-Diniz et al., 2011), and others have found no differences in IGT behavior (Yechiam et al., 2008; Jogia et al., 2011; Martino et al., 2011). Given the mixed results, our first goal was to conduct a meta-analysis of relevant IGT findings.

2. Study 1

We identified published studies indexed in PubMed or PsycINFO with the key words (IGT or “Iowa Gambling”) and (mania, bipolar, or manic). Studies were excluded from analysis if they did not include a euthymic bipolar I group (Ibanez et al., 2012) and a control group (Christodoulou et al., 2006; Jollant et al., 2007).

Meta-analysis was performed in the metafor package for R (Viechtbauer, 2010) using a random-effects model with restricted maximum likelihood estimation. Fig. 1 shows the effect size associated with each study. As shown, euthymic bipolar I participants ($N=215$) make more risky choices than control participants ($N=340$). The effect size of Hedges' $g=.35$ is small to medium by Cohen's standards. There was also marginal evidence for heterogeneity across studies (Cochran's $Q(4)=8.5$, $p=0.07$).

3. Study 1 discussion

Our meta-analysis raises two related questions. First, what is the source of the group difference? IGT performance rests on several cognitive processes, and analyses of overall IGT performance do not specify which processes underlie group differences. Second, the small effect size and relatively (though not significantly) heterogeneous pattern suggest that IGT performance varies among people with remitted bipolar disorder and that individual differences are important to consider. In study 2, we

examine cognitive processes underlying IGT performance in bipolar disorder using an explicit cognitive model, and we examine a range of individual differences as theoretically plausible explanations of variation in IGT performance.

4. Study 2

As noted, the IGT has been described as a measure of reward sensitivity (NIMH, 2011) and of real-life decision-making (Bechara et al., 1994), but IGT performance results from a complex of both “hot” and “cold” cognitive processes (Buelow and Suhr, 2009). Beyond reward sensitivity, potential explanations for poor performance include insensitivity to losses, difficulty learning or remembering the contingencies associated with each deck, random or inconsistent selections, or simple lack of investment in the task. Overall differences in IGT performance provide little insight into which cognitive processes differ between groups.

One way to gain insight is to build models of specific cognitive processes involved in IGT performance. Parameters representing individual differences in these cognitive processes can be estimated and compared across groups. One advantage of such cognitive models is that they integrate the complete sequences of gains, losses, and decisions experienced by each participant.

The Expectancy Valence (EV) Model (Busemeyer and Stout, 2002) is one popular model of IGT performance that has been used productively to study psychiatric and neurological conditions (Yechiam et al., 2005). The EV model suggests that IGT performance reflects three individual differences: relative attention to losses vs. gains, weight given to recent vs. prior trials, and consistency in selecting the same decks as the task continues. Reward models of bipolar disorder might predict that differences in IGT performance stem from lower attention given to losses vs. gains (Yechiam et al., 2008), as reward sensitivity might foster greater attention to gains.

In the one available study of the EV model in bipolar disorder (Yechiam et al., 2008), people with acute mania or depression displayed lower consistency as the task progressed than did control participants. There were no differences between the

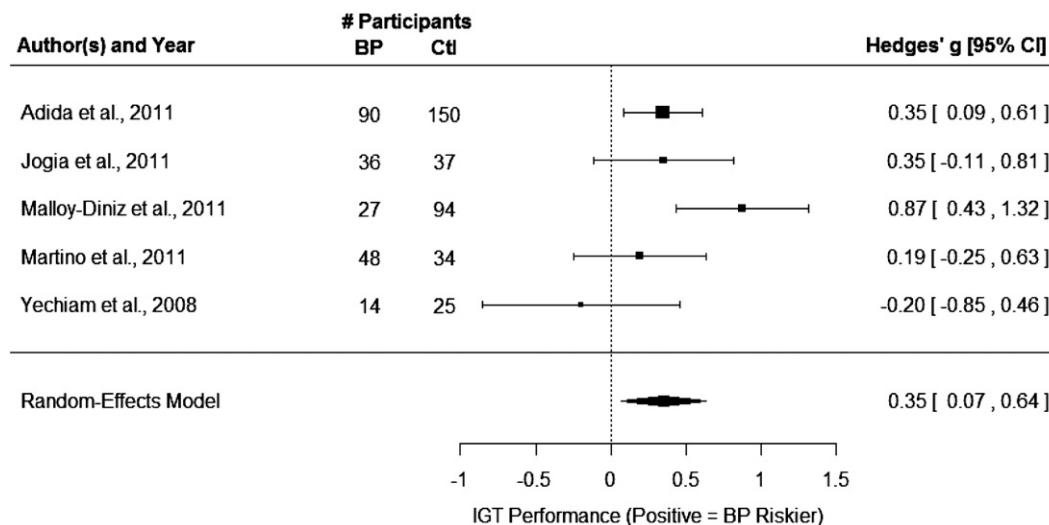


Fig. 1. A forest plot of previously published studies of IGT performance comparing euthymic bipolar I participants to controls is shown. Effect sizes are coded such that positive values indicate more risky choices by euthymic bipolar I participants. For most studies, the effect sizes are in terms of total number of safe choices or safe choices minus risky choices, but for Martino et al. (2011), the effect size is for total winnings from the task. Studies were identified by searching for the terms “Iowa Gambling” or “IGT” in combination with any of “bipolar disorder”, “mania”, “manic”, “hypomania”, “hypomanic”, “bipolar I”, “bipolar 1”, “bipolar II”, “bipolar 2”, “cyclothymia”, or “cyclothymic” in the title or abstract using PsycInfo and PubMed. Adida and colleagues (2011) includes the data from Clark et al. (2002), thus the 2002 study is not separately reported here. Similarly, the Malloy-Diniz et al. (2011) includes the data from Malloy-Diniz et al. (2009), so the 2009 study is omitted here. The effect size and sample size reported for Malloy-Diniz et al. (2011) is based on bipolar I participants with Young Mania Rating Scale (YMRS) scores less than 8 (Malloy-Diniz, personal communication), whereas the data reported in the original manuscript include participants with bipolar II disorder and/or YMRS scores up to 12.

euthymic bipolar I group and the control group in any EV model parameter estimates, but the study examined only 14 euthymic bipolar I participants and 25 control participants, meaning it was powered to detect only very large effects ($g > 0.96$ for power = 0.80 and $\alpha = .05$).

Beyond the EV model, many individual differences might relate to IGT performance within bipolar disorder, including symptom state, illness severity, or trait-like characteristics associated with the disorder. For example, positive affective state, which can intensify risky decision-making in nonclinical samples (Suhr and Tsanadis, 2007), might guide IGT performance among those with bipolar disorder (Buelow and Suhr, 2009). In bipolar disorder, even mild positive moods can affect cognitive performance (Stern and Berrenberg, 1979; Johnson et al., 2005; Roiser et al., 2009). It is important to consider subclinical mood states given the large effect sizes for IGT decrements related to mania (0.68, Adida et al., 2011) and hypomania (0.87; Malloy-Diniz et al., 2011 and personal communication) compared with controls.

Impulsivity is another construct that has received attention in both the bipolar disorder and IGT literatures. Elevated impulsivity has been related to bipolar disorder (Swann et al., 2003; Holmes et al., 2009) and poorer IGT performance (Franken et al., 2008; Upton et al., 2011). Risk-taking, which is related to impulsivity, is associated with bipolar II diagnosis (Hantouche et al., 2003). Impulsivity is also related to suicidality (Brezo et al., 2006), which has been linked to poorer IGT performance as well as bipolar illness severity (Malloy-Diniz et al., 2011; Martino et al., 2011).

4.1. Aims

One goal of this study was to assess whether EV model analyses would be more sensitive to group differences than traditional IGT analyses. A second goal was to consider factors that might explain variability in IGT performance within bipolar disorder, including impulsivity and clinical characteristics. We assessed IGT performance in euthymic participants with bipolar I disorder and in a well-matched control group.

5. Method

5.1. Participants

Participants were recruited through treatment centers, support groups, and community advertisements in the Miami and Palo Alto areas. Participants in the bipolar group ($N=55$) met criteria for bipolar I disorder as assessed by the Structured Clinical Interview for DSM-IV (SCID; First et al., 1997). Participants in the control group ($N=39$) did not meet current or lifetime criteria for any mood disorder, including bipolar I disorder, bipolar II disorder, cyclothymia, bipolar disorder NOS, major depressive disorder, minor depressive disorder, dysthymia, or depressive disorder NOS). All were between 18 and 65 years of age. Potential participants were excluded for substance abuse or dependence in the past year, primary psychotic disorder, general medical conditions of the central nervous system, history of serious head injury, or any developmental disability or language problem that could interfere with comprehension of study tasks. Potential participants who were prescribed traditional antipsychotics were excluded because traditional antipsychotics blunt reward sensitivity (Abler et al., 2007). Effort was made to recruit control participants with history of anxiety or substance-related disorder to enhance comparability of the control group.

Participants completed written informed consent procedures and received compensation for participation. Participants

completed other measures not described here (Johnson et al., 2012a). All procedures performed in this study were approved by the institutional review boards of the University of Miami and Stanford University.

5.2. Materials

5.2.1. SCID

The Structured Clinical Interview for DSM-IV (SCID; First et al., 1997) was used to assess lifetime and current psychiatric diagnoses. Staff who administered the SCID received extensive training from Dr. Johnson. Inter-rater reliability was 1.0 for the mania and depression modules as assessed by ratings of 10 randomly selected audio interviews. Course parameters such as number of episodes, number of hospitalizations, and age of onset were assessed for each diagnosis.

5.2.2. Somatotherapy index

We used the Somatotherapy Index (Bauer et al., 1997) to assess prescribed dosage and adherence for antidepressants, anticonvulsants, lithium, and atypical neuroleptics. We calculated an index of mood stabilizer adequacy by summing the reported dosages of lithium, valproate, carbamazepine, and atypical neuroleptics divided by their maximum recommended dosages.

5.2.3. Bech-Rafaelson Mania Scale (BRMS)

The BRMS (Bech et al., 1979) is an 11-item interview with standardized probes used to assess current manic symptom severity. Items cover mood, need for sleep, verbal and motor activity, flight of thoughts, and sexual interest. Responses are scored on a 5-point scale. Scores of 5 or lower indicate remission, scores from 16 through 27 indicate moderate mania, and scores of 28 or higher indicate severe mania. We obtained high inter-rater reliability (intraclass correlation = .84 based on 14 recordings) and acceptable internal consistency ($\alpha = .77$). The BRMS distinguishes current mania from interepisode status and is highly correlated with other measures of current mania (Bech, 2008).

5.2.4. Modified Hamilton Rating Scale for Depression (MHRSD)

To assess depressive symptoms, we used the MHRSD (Miller et al., 1985), a widely-used standardized interview. Scores range from 0 to 52, with scores on each item ranging from 0 to 2 or to 4. Scores below 7 indicate remission and scores over 17 indicate depressive relapse. We achieved excellent inter-rated reliability (intra-class correlation = .93 based on 14 recordings) and strong internal consistency ($\alpha = .82$). The MHRSD distinguishes depressive episodes from interepisode periods and is highly correlated with other measures of current depression.

5.2.5. Iowa Gambling Task (IGT)

Participants completed a computerized version of the IGT (Version 2.0, 2002, Antoine Bechara). At the start of the game, participants are told that \$2000 (in hypothetical money) is available and that they can gain or lose from that amount. On each trial, participants use a mouse to select a card from one of four decks and then see feedback on the amount won in the trial, amount lost in the trial (which may be \$0), and total cumulative earnings. On average, each selection from the “risky” decks yields a \$100 reward, and each selection from the “safe” decks yields a \$50 reward. Though the risky decks yield larger rewards, they also inflict larger penalties, averaging \$125 per trial compared with only \$25 from the safe decks. As such, selections from the safe decks give a \$25 net profit on average whereas selections from the risky decks lead to \$25 net losses on average. Participants completed 5 blocks of 20 trials (a total of 100 trials) with an

intertrial interval of 1500 ms. Participants were told to try to maximize their winnings but were not paid the amounts won in the game.

5.2.6. Positive Urgency Measure (PUM)

The PUM (Cyders et al., 2007) is a 14-item, self-report measure of the tendency to act impulsively in response to positive affective states. The PUM is related to but distinct from other impulsivity scales (Cyders et al., 2007). PUM scores have been observed to be more closely related to mania risk than are other impulsivity measures (Johnson et al., in press) and among people with bipolar disorder, PUM scores are related to poorer social outcomes (Victor et al., 2011).

5.3. Procedure

Potential participants who contacted study staff were interviewed by phone to determine potential eligibility. Those who appeared eligible were invited to the University of Miami or Stanford University, where they completed informed consent procedures and a more detailed diagnostic interview. Participants who were eligible based on diagnostic interviews completed symptom severity interviews. If symptom severity interviews did not indicate remission (MHRSD > 6 or BRMS > 5), participants were scheduled for monthly interviews to track symptom remission. Bipolar participants completed the IGT only after achieving symptom remission.

5.4. Analysis plan

We use three strategies to examine group differences in IGT performance. First, to facilitate comparison with previous studies, we compared the number of risky choices made by each group using a *t*-test. Next, we use a Generalized Estimating Equations approach to examine group differences across the five 20-trial blocks. Finally, we used the EV Model to compare the groups in terms of three cognitive processes postulated to guide IGT performance. We report results from the EV Model rather than the more recent Prospect Valence Model (Ahn et al., 2008) because it has been used in bipolar disorder (Yechiam et al., 2008) and several other psychiatric conditions (Yechiam et al., 2005). (Subsidiary analyses using the Prospect Valence Model yielded parallel results.)

5.4.1. Expectancy valence model

The EV Model (Busemeyer and Stout, 2002) postulates that variations in task performance are partly due to individual differences in three parameters and partly stochastic. Under the model, it is assumed that persons combine information about gains and losses from each trial to assign a value, or valence, to each deck. The valences associated with each trial are combined to assign an expectancy to each deck. The expectancies for each deck are then used to assign a probability that that deck will be chosen on the next trial. One parameter is associated with each of the three stages of this process.

As described in detail by Busemeyer and Stout (2002), after each trial *t*, the participant gains some amount of money and loses some amount of money (possibly 0). The gain and loss are combined way to produce a valence as follows:

$$v(t) = [(1-w) \times G(t) + w \times L(t)]$$

This is a weighted average of the gain and loss from the deck chosen at time *t*. The weight is determined by the parameter *w*, also called the attention to losses parameter, which takes values in [0,1]. If *w* is 0, losses are completely ignored, and if *w* is 1, gains are ignored. If *w* is 1/2, gains and losses are weighted equally.

Based on the valence from a given trial, the expected valence for the deck chosen (denoted D_i) on that trial is updated as follows:

$$E[D_i|t] = (1-a) \times E[D_i|t-1] + a \times v(t)$$

This is a weighted average of the expected valence for deck *i* on the previous trial and the valence experienced from deck *i* on the current trial. The weighting is determined by the recency parameter, *a*, which takes values in [0,1]. The higher the value of *a*, the more strongly recent trials are weighted when the expected valence is updated.

The parameters *w* and *a* specify how expected valences are updated. One possible strategy is to always choose the deck with the highest expected valence, but this approach does not allow for exploration. To allow for exploration, the EV Model assigns probabilities to decks as follows:

$$P[D_i|t+1] = \frac{e^{E[D_i|t]\theta(t)}}{\sum_{j=1}^4 e^{E[D_j|t]\theta(t)}}$$

Here, $\theta(t)$ determines how much influence the expectancies for each deck have on the probability that the deck will be selected. If $\theta(t)$ is 0, then the probability of choosing each deck on a given trial will be 1/4, regardless of the expected valences (maximum exploration). As $\theta(t)$ becomes large, the expected valence has more influence on the probability of choosing each deck. The EV model assumes that $\theta(t)$ changes over the course of the task according to:

$$\theta(t) = \left(\frac{t}{10}\right)^c$$

The parameter *c*, also called the consistency parameter, determines how $\theta(t)$ changes across the task. When *c* is positive, $\theta(t)$ increases as the task continues, which means that the expected valences become more influential on the probability of selecting each deck. When *c* is negative, $\theta(t)$ decreases as the task continues, causing the expected valences to have less influence on the probability that each deck is selected and thus leading to more random responding.

In sum, the EV model explains differences in IGT performance in terms of three individual differences—the extent to which participants attend to losses versus gains, the extent to which participants weight recent information, and the extent to which their responses become either more or less guided by their expectations about each deck as the task progresses.

We estimated the parameters of the EV model using maximum likelihood estimation based on each participant's individual data in R (see free code available from Anthony Bishara at <http://bisharaa.people.cofc.edu/modeling.htm>). Other approaches are available, including individual and hierarchical Bayesian estimation (Wetzels et al., 2010), but individual maximum likelihood estimation has been used most frequently in psychopathology research.

We assessed model fit by comparison with a baseline model assuming for a given participant the probability of choosing a given deck is the same on each trial. The extent to which the EV model is an improvement over the baseline model is quantified by the G^2 statistic, which is equal to twice the difference in log-likelihoods of the two models (Busemeyer and Stout, 2002). Larger values indicate better fit of the EV model compared with the baseline model.

6. Results

As shown in Table 1, bipolar and control groups did not differ on age, gender, years of education, or current manic or depressive symptoms. The bipolar group had higher rates of lifetime or

current anxiety disorder and of lifetime substance-related disorder. The bipolar group reported a relatively severe illness history.

6.1. Comparing the bipolar and control group on IGT performance

Collapsing across all 100 trials of the task, the number of safe choices did not differ between the bipolar ($M=55.3$, $sd=14.6$) and control groups ($M=57.3$, $sd=13.9$), $t(92)=0.7$, $p=.51$, $g=.14$). We used Generalized Estimating Equations (GEE) with unstructured within-subjects correlation and Huber standard errors to examine the change in the number of safe choices made across the five 20-trial blocks (see Fig. 2). The block effect was significant, $\chi^2(4)=29.3$, $p<.001$, indicating that participants learned to choose the safe decks more frequently over the course of the task. There was no main effect of bipolar I diagnosis, $\chi^2(1)=0.1$, $p=.77$, nor was there an interaction of block and diagnosis, $\chi^2(4)=3.2$, $p=.53$.

As shown in Table 2, the bipolar and control groups did not differ on estimates of any of the three EV model parameters—attention to losses, recency, or consistency. Because the parameter distributions were notably non-normal, we compared the sample means using permutation tests with 10,000 permutations in addition to t -tests. Across all participants, the mean(sd) G^2 statistic was 7.7(19.9), which is significantly greater than 0,

$t(93)=3.7$, $p<.001$, indicating that on average, the EV model fit participants' trial-level IGT data better than the baseline model. The bipolar and control groups did not differ on G^2 , $t(92)=0.3$, $p=.79$. It should be noted that for some participants, the EV model fit was not better than the baseline model fit: 12/39 control and 19/55 bipolar I participants had G^2 values less than zero. The proportion of participants for whom the EV model did not fit did not differ by diagnostic group, $\chi^2(1)=.03$, $p=.87$.

6.2. Correlates of IGT performance within the bipolar group

To consider whether IGT performance was related to individual differences, we examined whether demographic (age, gender, education), mood state (BRMS, MHRSD), illness severity indices (number of depressive episodes, number of manic episodes, number of depression hospitalizations, number of mania hospitalizations, number of suicide attempts), comorbidity (lifetime substance or alcohol use disorder, lifetime or current anxiety disorder), mood stabilizer use, or impulsivity (PUM), were correlated with the number of trials choosing from a risky deck, or with estimates of the three EV model parameters—attention to loss vs. reward, recency, and consistency. None of the 14 variables were significantly related to IGT

Table 1
Descriptive data.

Demographic and disease course variables	Bipolar I mean (SD) (N=55)	Control mean (SD) (N=39)	Group difference
Age	36.0 (11.9)	33.5 (12.8)	$t(92)=1.0$, $p=.33$
Gender (% female)	65	59	$\chi^2(1)=.02$, $p=.88$
Years education	14.3 (1.9)	14.1 (2.2)	$t(92)=.5$, $p=.63$
BRMS	3.2 (3.1)	2.6 (3.1)	$t(92)=.8$, $p=.42$
MHRSD	4.7 (5.6)	5.1 (6.1)	$t(92)=-.3$, $p=.73$
% with lifetime or current anxiety disorder	67	28	$\chi^2(1)=12.4$, $p<.001$
% with history of substance-related disorder	65	23	$\chi^2(1)=14.8$, $p<.001$
% taking mood stabilizer	53		
% taking antidepressant	40		
Previous MDEs	13.3 (12.4)		
Previous hospitalizations for MDE	1.3 (2.1)		
Age of MDE onset	18.3 (7.8)		
Previous manic episodes	9.6 (10.3)		
Previous hospitalizations for mania	1.5 (3.1)		
Age of mania onset	21.6 (9.1)		

Note. BRMS=Bech Rafaelsen Mania Scale, MHRSD=Modified Hamilton Rating Scale for Depression, MDE=Major Depressive

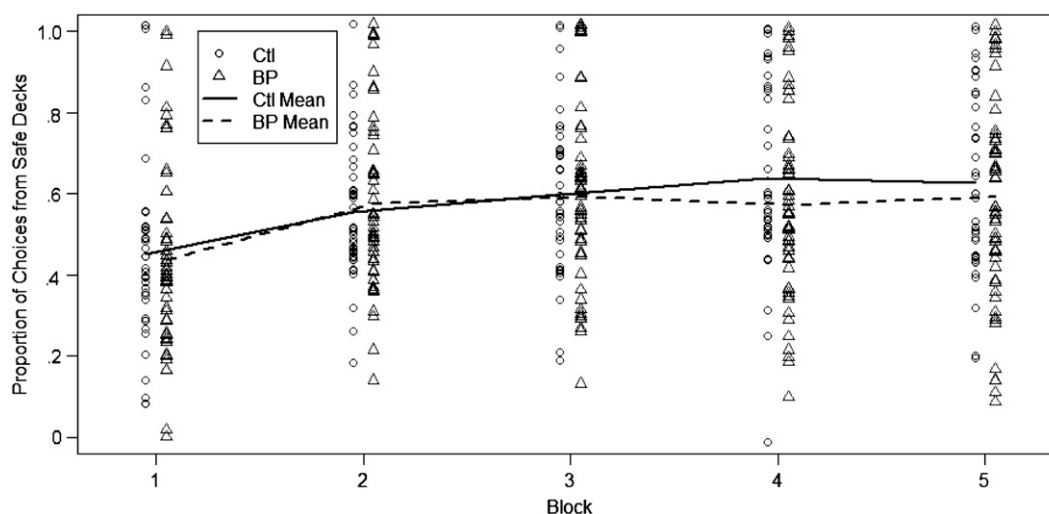


Fig. 2. The proportion of choices from safe decks, which is equal to one minus the proportion of choices from risky decks, is shown for all participants. The bipolar and control groups did not differ across the task or during any of the task's five 20-trial blocks shown on the x-axis. A small amount of random noise was added to each participant's data to avoid data points being plotted directly on top of each other.

performance (all $ps > 0.05$). The magnitude of correlations was also small, with all $|r|$ s less than 0.24.

6.3. Updating the meta-analysis

When this study's effect size estimate for the group difference in risky choices ($g=0.14$) is added to the meta-analysis reported above, results change only slightly. The overall effect size estimate changes from $g=.35$ to $g=.32$, with a 95% confidence interval of [0.08,0.56] (see Fig. 3). Cochran's test for heterogeneity remains short of significance, $Q(5)=9.6$, $p=0.09$.

7. Discussion

Study 1 was a meta-analysis of five studies of IGT performance among euthymic persons with bipolar I disorder compared with control participants. Findings of the meta-analysis suggested an effect size of Hedges' $g=.35$, which is relatively small compared with effect sizes associated with other cognitive tests in euthymic bipolar I

disorder (Robinson et al., 2006; Bora et al., 2009). There was also marginal evidence of heterogeneity in the effect sizes across studies.

Study 2 examined IGT performance in persons diagnosed with bipolar I disorder in remission and a well-matched control sample to consider two issues. First, we used a cognitive model to differentiate three processes that might contribute to IGT deficits. Second, we considered variables that might contribute to heterogeneity in IGT performance within bipolar disorder. In this sample, we found no evidence that persons with remitted bipolar I disorder differed from the control sample in their willingness to choose cards from risky decks, nor in the estimates of the EV Model parameters of attention to losses, attention to recent feedback, and consistency vs. exploration. The effect size for the group difference in overall IGT performance in this study ($g=.14$) is broadly consistent with findings in other remitted bipolar samples.

We also considered a range of variables that might help explain heterogeneity of IGT performance across and within bipolar samples, including demographic, personality, affective, and illness characteristics. We found no evidence that these variables were related to IGT performance in bipolar disorder.

Findings from this study did not illuminate processes that would help explain the modest group differences in IGT performance found in previous research. It is possible that bipolar disorder is distinguished by processes that are not indexed by the EV model. Indeed, for a third of participants, the EV model's fit was not superior to a model that did not incorporate learning, suggesting that some participants' approaches to the task included elements not well-captured by the model's parameters. These might include patterns of responding in which participants' responses are not dependent on feedback.

This study had other limitations. First, our sample size, though larger than most previous studies of IGT performance, was not large enough to detect small effects ($g > 0.6$ for power $> .80$ at $\alpha=.05$). Second, we did not investigate the effects of current mania or depression on IGT performance.

7.1. The IGT as a measure of reward sensitivity

As the IGT has been recommended as a measure of reward-related processes (NIMH, 2011), findings related to reward sensitivity in bipolar disorder deserve special consideration. This study yielded no evidence of an elevated sensitivity to reward (as compared to loss) among people with bipolar disorder, as assessed by the EV model. This is in apparent conflict with

Table 2
Expectancy valence model results.

	Bipolar I (N=55)	Control (N=39)
Attention to losses		
Mean	.35	.32
SD	.31	.25
Median	.30	.29
t-test	$t(92)=0.3$	
Permutation test	$p=.60$	
Recency		
Mean	.40	.38
SD	.42	.38
Median	.14	.20
t-test	$t(92)=0.5$	
Permutation test	$p=.74$	
Consistency		
Mean	.44	.44
SD	2.2	2.0
Median	.61	.81
t-test	$t(92)=0.0$	
Permutation test	$p=.99$	

Note. Permutation tests for sample mean using 10,000 permutations.

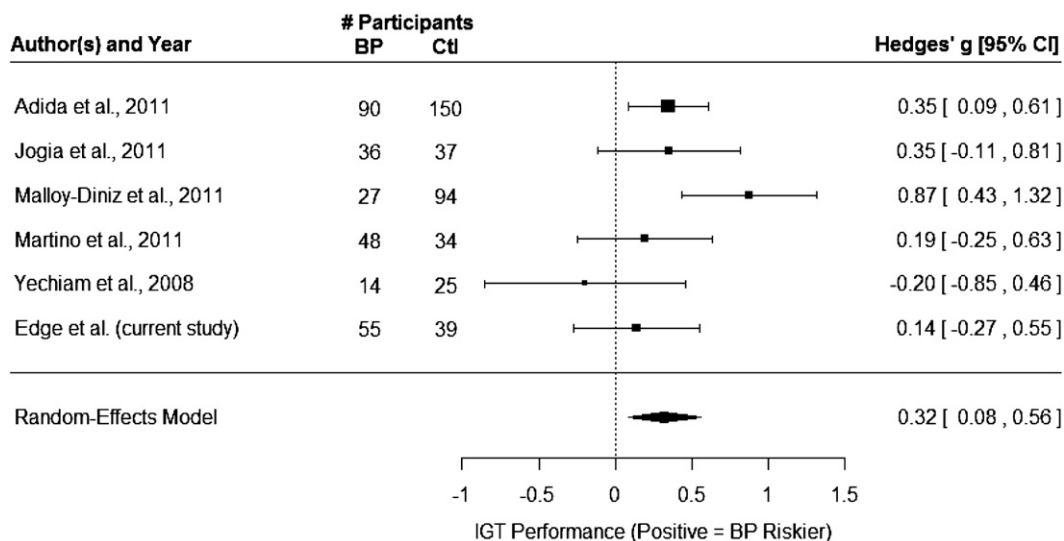


Fig. 3. A forest plot of the five studies meta-analyzed in Fig. 1 plus the current study is shown.

previous studies using self-report, behavioral, and psychophysiological measures of reward sensitivity (Johnson et al., 2012b).

In considering this issue, we note that the design of the IGT varies gains and losses together. In fact, losses vary more strongly than do gains. That is, a switch from a safe deck to a risky deck involves both an increased gain and (more markedly) increased losses. Given this structure, the IGT cannot measure sensitivity to rewards and sensitivity to losses separately. It measures only their strength relative to each other, as reflected in the first EV parameter, attention to losses vs. gains. The linking of reward sensitivity to loss sensitivity is problematic for exploration of reward sensitivity per se, as self-report data suggest that sensitivity to reward and punishment are independent dimensions (Carver and White, 1994).

The IGT's confounding of reward and loss sensitivity may be particularly problematic in studies of bipolar I disorder. Many people with bipolar disorder display elevated threat sensitivity, particularly in the context of depressive symptoms (Johnson et al., 2003), which would render the IGT of little use for detecting elevations in reward sensitivity in bipolar disorder. We recommend that researchers interested in reward sensitivity employ paradigms that measure reward sensitivity independently of punishment sensitivity.

Conflict of interest

All authors declare that they have no conflicts of interest.

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