EMPIRICAL ARTICLE

Differential Impairments Underlying Decision Making in Anorexia Nervosa and Bulimia Nervosa: A Cognitive Modeling Analysis

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ABSTRACT

Objective: This study examined the underlying processes of decision-making impairments in individuals with anorexia nervosa (AN) and bulimia nervosa (BN). We deconstructed their performance on the widely used decision task, the Iowa Gambling Task (IGT) into cognitive, motivational, and response processes using cognitive modeling analysis. We hypothesized that IGT performance would be characterized by impaired memory functions and heightened punishment sensitivity in AN, and by elevated sensitivity to reward as opposed to punishment in BN.

Method: We analyzed trial-by-trial data of IGT obtained from 224 individuals: 94 individuals with AN, 63 with BN, and 67 healthy comparison individuals (HC). The prospect valence learning model was used to assess cognitive, motivational, and response processes underlying IGT performance.

Results: Individuals with AN showed marginally impaired IGT performance compared to HC. Their performance was characterized by impairments in memory functions. Individuals with BN showed significantly impaired IGT performance compared to HC. They showed greater relative sensitivity to gains as opposed to losses than HC. Memory functions in AN were positively correlated with body mass index.

Discussion: This study identified differential impairments underlying IGT performance in AN and BN. Findings suggest that impaired decision making in AN might involve impaired memory functions. Impaired decision making in BN might involve altered reward and punishment sensitivity.

Keywords: anorexia nervosa; bulimia nervosa; decision making; cognitive modeling; reward processing; reward sensitivity; punishment sensitivity; memory deficits

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Introduction

Previous studies have shown that individuals with eating disorders perform worse in decision-making tasks such as the Iowa Gambling Task (IGT) than healthy controls.[1–3] The IGT has been widely used as a measure of decision making.[4] The traditional approach to analyzing IGT performance does not allow for quantitative comparisons of underlying processes and hence fails to identify any distinctive or comparable underlying impairment across disorders. Assessments that identify and assess underlying processes might advance understanding of potential differences or similarities in neurocognitive processes across disorders. Cognitive modeling can isolate and assess component processes involved in complex neurocognitive tasks, such as the IGT, and thus identify specific underlying impairments.[5] This study applied cognitive modeling to analyzing IGT performance in anorexia nervosa (AN) and bulimia nervosa (BN) and examined whether decision-making deficits in AN and BN were attributable to different underlying mechanisms. Understanding the potential differences or similarities in impairments underlying decision making between patients with AN and BN might have implications for specific treatment approaches for these two disorders. Some studies failed to find impaired decision-making task performance in individuals with eating disorders (e.g., Ref. 6). Cognitive modeling analysis, by deconstructing decision-making task performance into component processes, allows for the examination of

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subtle impairments in the underlying mechanisms, which are not reflected in the overall task performance.

Cognitive Modeling of Decision Making

Decision making is not a single process; rather, it involves multiple processes. However, the traditional approach to analyzing decision-making tasks presumes that overall performance of a decision-making task represents a unidimensional construct of decision making. For example, in the IGT, decision-making performance is traditionally indexed by the net number of cards chosen from the advantageous decks over 100 trials. This approach disregards the multiprocess nature of decision making and has limited utility in unraveling subtle differences or similarities that might exist across different clinical populations.

Cognitive modeling has been applied in various neurocognitive tasks and among different clinical populations to deconstruct task performance into more refined underlying processes.[7] Unlike the traditional approach to analyzing cognitive tasks, cognitive modeling deconstructs task performance into theorized underlying processes based on formal cognitive models. Formal cognitive models are informed by theories of cognitions and provide algorithms for theorized processes, which can then be evaluated with respect to actual data and can quantify theorized processes.[5,8] In other words, formal cognitive models can provide a theoretical basis for the analysis of the multiple processes involved in cognitive tasks. Formal cognitive models also provide quantitative precision in assessing the theorized underlying processes, which allows for quantitative comparisons on specific component processes across different populations.

The expectancy valence learning (EVL) model is a validated formal cognitive model of IGT, which has shown better fit with actual data than other models of IGT.[5] It has been used in several clinical populations and has identified specific impairments in each disorder.[5,9–11] In the EVL model, there are three theorized processes involved in IGT performance, namely, *motivational*, *learning*, and *response* processes. It is theorized that participants make a card selection in a given trial based on their expectation of valence (i.e., an affective feeling, positive or negative, associated with a given deck or an implicit association between a given deck and good/bad outcomes), which is formed through learning/memory of the experiences of gains and losses received following choosing a given deck in preceding trials. The formation of the expectation of valence is a function of one's sensitivity to gains and losses. For example, a person who has higher sensitivity to losses than gains will form an expectation of negative valence for a given deck that has given equal amounts of gains and losses. The individual difference in sensitivity to gains as opposed to loss is represented by the *motivational parameter* in the EVL model (see Methods section for details). The formation of the expectation of valence is also dependent on memory and learning functions. Individuals who have impaired memory and learning functions are less able to use information from preceding trials to guide their decisions. The individual difference in this process is represented by a *learning/memory parameter* that indexes how much experiences in past trials are discounted (memory decay). Finally, a person might desire to explore various options as opposed to follow the expectation of valence that has formed. The *response consistency parameter* represents the degree of consistency in making decisions with respect to the expectancies of valences (exploitation) as opposed to making random choices (exploration).

Previous studies have shown that, using the EVL model of decision making, IGT performance was found to be characterized by different underlying mechanisms across disorders, including motivational biases in drug users[12] and adolescents with early-onset schizophrenia,[11] impaired learning/memory functions in individuals with Huntington's disease,[5] and response inconsistency in autistic spectrum conditions[9] and bipolar disorders.[10] This analytic approach could also reveal potential differential impairments underlying the IGT in AN and BN.

Neuropsychological Characteristics in AN and BN

Individuals with AN and BN are characterized by different neuropsychological profiles.[13,14] It has been suggested that decision-making deficits in AN might be attributable to impaired implicit learning.[15,16] Individuals with AN have also reported heightened sensitivity to punishment[17,18] and shown increased activation in the striatum in response to the omission of taste rewards[19] and in the amygdala in response to threatening stimuli.[20] On the other hand, individuals with BN have consistently shown altered feedback sensitivity. Brain imaging studies with individuals with BN have shown attenuated activation in response to food rewards in the brain regions relating to reward processing, including regions in the striatum and the insular cortex.[21] Altered activations in the striatum in response to monetary rewards have also been observed in individuals with recovered BN

compared to healthy controls.[22] It is suggested that initial heightened reward sensitivity leads to attenuation of the reward response over time, which feeds forward into additional reward-seeking behavior such as binge-eating.[23]

Both AN and BN have shown decision-making deficits as reflected in the IGT in previous studies.[1,2] Studies on the performance of IGT over trials (net cards chosen from advantageous decks by blocks) did not reveal any significant differences between AN and BN; patients with AN and BN both showed no significant improvement over trials while healthy comparison (HC) participants did.[2] In a study on skin conductance responses to gains and losses after each trial, it was found that individuals with AN showed attenuated responses to both gains and losses compared to individuals with BN or HC.[2] However, it has been suggested that attenuated skin conductance responses might be caused by underweight.[1] It is unclear whether IGT performance of AN and BN are characterized by common or different underlying impairments or both.

The Present Study

This study applied cognitive modeling to analyze the performance of individuals with AN and BN in comparison to HC on the widely used decision-making task, the IGT. Reasoning from evidence suggesting differential neuropsychological profiles of AN and BN, we hypothesized that decision-making deficits in AN and BN would stem from differential underlying processes. Specifically, we hypothesized that decision making in AN would be characterized by impaired learning/memory functions and heightened sensitivity to punishment, whereas decision making in BN would be characterized by heightened sensitivity to reward as opposed to punishment.

Method

Participants

Data of 226 participants were obtained at Université Montpellier (67 healthy control; 45 AN; 42 BN),[a] Johns Hopkins University (36 AN; 10 BN), and Altrecht Eating Disorders Rintveld (13 AN; 11 BN). Informed consent was obtained from each participant. Two participants in the AN group from the subsample at Johns Hopkins University who completed only 73 and 83 trials of 100 trials of IGT were excluded from this study. In our analysis, there were a total of 224 participants: 67 participants in the healthy control (HC) group, 94 participants with a diagnosis of AN, and 63 participants with a diagnosis of BN. The majority of the sample was female (97%); there were six male participants in the AN group and one in the BN group. Ethical approvals were obtained from the Institutional Review Boards of the institutions conducting the studies.

All participants in the HC group were recruited at Université Montpellier. They were interviewed by a psychiatrist using the Mini International Neuropsychiatric Interview[24] and determined to have no lifetime history of eating disorder or current axis I psychiatric disorders, and their BMIs were between 18 and 25kg/m^2. Participants in the AN and BN groups were recruited through inpatient and outpatient units at Université Montpellier; inpatient unit at Johns Hopkins University; and online forum at Altrecht Eating Disorders Rintveld. Participants at Université Montpellier and Johns Hopkins University were interviewed by a psychiatrist assessing criteria for AN and BN as defined in the *Diagnostic and Statistical Manual of Mental Disorders, 4*th *Edition* (DSM-IV; American Psychological Association, 1994). Participants at Université Montpellier who met criteria for a current depressive disorder were excluded. Participants at Altrecht Eating Disorders Rintveld were assessed with the self-report Eating Disorders Diagnostic Scale.[25]

IGT Procedure

All participants completed a computerized version of the IGT.[4] Four decks of cards were presented on a computer screen. Each participant had to choose a card from one of the decks in each trial for 100 trials. Deck A and deck B were the disadvantageous decks which gave a larger gain in each trial but a larger expected loss over 100 trials than decks C and D. Participants were not told which decks were advantageous. They were instructed to make card selections that would maximize their gains. No actual monetary payments were made; however, hypothetical winning has shown the same patterns of activation in the brain as actual payments.[26]

The IGT procedure varied in two ways across three sites. In the IGT used with the sample at Université Montpellier, the amount of gains in each trial varied ($75–125 for decks A and B; $25–75 for decks C and D), whereas the gains in the IGT trials administered to the sample at Johns Hopkins University and Altrecht Eating Disorders Rintveld were fixed ($100 for decks A and B;

[a]IGT performance of all the HC, 37 AN, and 32 BN was published by Guillaume et al. (8). However, trial-by-trial IGT data of this sample have not been published elsewhere. Trial-by-trial data of some participants included in Guillaume et al. were not available. Hence, these participants were not included in this study. They, however, did not differ on BMI, age, or overall IGT performance from participants included in this study. Trial-by-trial data of 8 AN and 10 BN from an ongoing study (with the same criteria of inclusion) that were available at the time of the analysis were included in the present study as well.

$50 for decks C and D). This variation of the schedule of gains was not expected to influence the cognitive modeling results because outcomes of gains and losses of every single trial were taken into account in the estimations of cognitive model parameters. Second, although the IGT procedure was administered with computers in all three sites, the IGT procedure at Johns Hopkins University was administered inside an fMRI scanner and participants had to respond to a trial within 5 seconds. We believe that all participants had adequate time to respond because all participants in our subsamples who were given unlimited time to respond responded within 5 seconds in each trial.

Knowledge of IGT was assessed in participants recruited at Université Montpellier. Participants were assigned score 0 if they indicated no conscious knowledge specifying a preference for one of the two advantageous decks; 1 if they indicated conscious knowledge specifying a preference for one of the two advantageous decks.

Depressive Symptoms, Impulsivity, and Body Mass Index

Depressive symptoms were assessed in participants recruited at Université Montpellier and John Hopkins University by the Beck Depression Inventory.[27] Depressive symptoms were not assessed in participants recruited at Altrecht Eating Disorders Rintveld. *Impulsivity* was assessed among participants at Université Montpellier using the Barratt Impulsivity Scale (BIS; 28). The BIS consists of 30 items assessing six subdimensions of impulsivity including attentional impulsivity (e.g., I don't pay attention), cognitive instability (e.g., I have racing thoughts), motor impulsivity (e.g., I act on the spur of the moment), perseverance (e.g., I change jobs), self-control (e.g., I plan tasks carefully), and cognitive complexity (e.g., I get easily bored when solving thought problems). The BIS scale showed adequate internal consistency (α = .76). "Body mass index" (BMI) was computed by objectively measured height and weight at Université Montpellier and John Hopkins University, and by reported height and weight at Altrecht Eating Disorders Rintveld.[29]

Cognitive Modeling Analysis—Prospect Valence LearningModel

In this study, we applied the prospect valence learning (PVL) model,[30] a revised version of the EVL Model, to analyze data of trial-by-trial IGT card selections. The PVL model, compared to the EVL model, was shown to provide better fit for IGT data and give more reliable parameter estimates.[30] The PVL model differs from the EVL model in that the motivational process is indexed by two parameters—*loss aversion* and *feedback sensitivity* rather that one parameter of loss aversion. The mathematical details of this model are included in Appendix A. Descriptions of the ranges and the meanings of each parameter are as follows.

Feedback sensitivity determines the nonlinear relation between the magnitudes of gains or losses and the magnitudes of valences. It ranges from 0 to 1. A higher value represents higher sensitivity to the magnitude of outcomes. A value of 1 indicates that the intensity of the experience of gains/losses is in direct proportion to the magnitude of the gains/losses. A value of 0 indicates that the person experiences feedback of all magnitudes equally. Loss aversion determines the relative influence of losses as opposed to gains on the formation of valence in loss trials. It ranges from 0 to 5, with higher values representing higher relative sensitivity to losses as opposed to gains (i.e., more loss-aversive). A value of 1 indicates equal sensitivity to gains and losses. Values smaller than 1 indicates greater sensitivity to gains than losses; values greater than 1 indicate greater sensitivity to losses than gains. The *learning/memory parameter* determines how much the past expectancy is discounted in the formation of expectancy of valence of the current trial. It ranges from 0 to 1. A higher value represents better learning and memory. The *response consistency parameter* represents the degree to which the respondent tends to explore different options as opposed to choose the card that is associated with the optimal expectancy based on previous evidence. It ranges from 0 to 5.[31] A higher value of *c* indicates that higher consistency in making decisions based on expectancies of valences while a lower value indicates random, erratic decision-making style.

To summarize, the PVL model deconstructs IGT performance into learning/memory, feedback sensitivity, loss aversion, and response consistency parameters. These four model parameters were estimated for each individual using Hierarchical Bayesian models (see Ahn et al., 2011 and Appendix B). We evaluated group differences on model parameters by examining the 95% highest density interval (HDI), which is an interval that spans 95% of the posterior distribution of differences.[32] If the 95% HDI of group differences excludes zero, it would indicate that the groups are credibly different[b]. We computed Cohen's *d* (mean difference/pooled standard deviation) for each significant group difference to indicate its effect size.

The IGT schedules of gains and losses were slightly different across sites. However, cognitive modeling analysis took into account the gains and losses of every single trial, and hence the variation in the schedules of gains and losses was not expected to bias estimates of model parameters. Hence, we combined data from all three sites for the modeling analysis. We also evaluated group differences in each subsample separately using F tests, to evaluate the consistency of results across sites.

TABLE 1. Means and standard deviations of sample characteristics and IGT overall performances

	HC ($N = 67$)	AN ($N = 94$)	BN ($N = 63$)	Sig. Group Diff. from Post Hoc Analysis (p Values)
Age	25.45 (6.70)	25.58 (8.45)	26.91 (10.82)	
BMI	20.58 (1.78; $N = 64$)	15.47 (1.89; $N = 89$)	22.80 (4.55; $N = 62$)	AN < HC < BN (p's < .001)
BDI[a]	1.37 (2.21)	16.89 (13.21; $N = 76$)	11.35 (9.97; $N = 49$)	HC < BN, AN ($p < .001$); BN < AN ($p = .007$)
IGT net score	8.15 (29.91)	−.86 (25.25)	−5.14 (34.66)	BN < HC ($p = .033$)
IGT block1	-3.07 (6.01)	−2.78 (6.43)	−4.03 (7.18)	
IGT block2	1.67 (6.75)	−.85 (6.26)	−.89 (7.63)	
IGT block3	3.22 (9.22)	−.45 (7.82)	.30 (8.45)	HC > AN ($p = .021$)
IGT block4	3.46 (9.54)	1.20 (9.09)	−.67 (10.44)	HC > BN ($p = .046$)
IGT block5	2.87 (9.27)	2.01 (9.27)	.14 (11.26)	
Knowledge[b]	.55 (.50)	.43 (.50)	.40 (.50)	
BIS[b]	54.17 (6.07)	53.29 (6.52)	56.08 (7.56)	

Notes: BMI, body mass index; BDI, Beck Depression Inventory; IGT net score, the number of cards chosen from the disadvantageous decks subtracted from the number of cards chosen from the advantageous decks; Knowledge, knowledge of IGT.
[a]Data were available only in the Université Montpellier and Johns Hopkins University subsamples.
[b]Data were available only in the Université Montpellier subsample.

Group comparisons on overall IGT performance (net number of advantageous cards chosen), IGT performance by blocks, and clinical characteristics were conducted using F tests with post hoc comparison tests with Bonferroni corrections. Because previous studies suggest that depressive symptoms, BMI, knowledge of IGT, and impulsivity might explain group differences in IGT performance, we conducted group comparisons with these variables statistically controlled for in the subsamples with the information available. We also examined correlations among model parameters, depressive symptoms, BMI, knowledge of IGT, and impulsivity.

Results

Table 1 presents sample characteristics and IGT performance for each group. BMI was significantly different across groups, $F_{2,212} = 128.99$, $p < .001$. AN had significantly lower BMI than BN and HC, while BN had significantly higher BMI than HC. In the Université Montpellier and Johns Hopkins University subsamples in which depressive symptoms were assessed, BDI scores were significantly different across groups, $F_{2,189} = 45.15$, $p < .001$. The patient groups had significantly higher levels of depressive symptoms than HC; the level of depressive symptoms of AN was significantly higher than that of BN. The IGT performance net scores were also significantly different across groups, $F_{2,221} = 3.50$, $p = .032$. Performance in BN was significantly worse than HC ($p = .033$). Performance in AN was marginally impaired compared to HC ($p = .058$). With regards to performance by blocks, differences across groups were significant in Blocks 2 and 3, and marginally significant in block 4 [Block 2: $F_{2,221} = 3.26$, $p = .040$; Block3: $F_{2,221} = 3.90$, $p = .022$; Block4: $F_{2,221} = 3.01$, $p = .051$]. Knowledge of IGT and impulsivity did not differ across groups.

Model Parameters

Table 2 presents group means of model parameters and 95% HDI for mean differences for all subsamples. As shown in **Table 2**, the learning/memory parameter was credibly lower or relatively impaired in individuals with AN compared to HC. Feedback sensitivity, loss aversion, and "response consistency" were not significantly different between AN and HC.

The motivational parameters were significantly different in individuals with BN compared to HC and AN. Feedback sensitivity was credibly higher in BN compared to HC and AN, and loss aversion was credibly lower in BN compared to HC and AN. "Learning and response consistency" were not different between BN and HC or between BN and AN.

Formation of Valences in Relation to Gains and Losses

To illustrate the significant differences in feedback sensitivity and loss aversion between BN and HC, **Figures 1**A and **1**B depict the associations between gain/loss magnitudes and valences, as calculated from Eq. (1) in Appendix A using the estimated parameters of feedback sensitivity and loss aversion. As shown in **Figure 1**A, individuals with BN showed stronger sensitivity to magnitudes of gains compared to HC. Likewise, individuals with BN showed stronger sensitivity to magnitudes of loss compared to HC as shown in **Figure 1**B. However, they showed relatively stronger sensitivity to gains than losses. For the same amount of gains and losses, the valence was greater for gains than for losses in BN, while the valence was smaller for gains than for losses in HC.

Group Comparisons in Subsamples

Because there were slight variations in IGT procedures and sample characteristics across sites, we

TABLE 2. Group means of model parameters and 95% high-density interval (HDI) for mean differences

Model Parameter	HC ($N = 67$)	AN ($N = 94$)	BN ($N = 63$)	95% HDI for Mean Differences	Effect Size (Cohen's d)
Learning/memory	0.68 (0.21)	0.56 (0.20)	0.61 (0.24)	AN < HC (mean diff. = .12, 95% HDI = .021–.22)	0.59
Feedback sensitivity	0.17 (0.13)	0.18 (0.12)	0.37 (0.15)	BN > HC (mean diff. = .20, 95% HDI = .06–0.36) BN > AN (mean diff. = .19, 95% HDI = .06–0.35)	1.42 1.40
Loss aversion	1.38 (0.99)	1.28 (0.90)	0.90 (0.87)	BN < HC (mean diff. = .48, 95% HDI = −.01–1.01) BN < AN (mean diff. = .38, 95% HDI = .038–0.90)	0.52 0.43
Choice consistency	0.44 (0.31)	0.48 (0.34)	0.49 (0.35)		

TABLE 3. Group means of model parameters by site

	HC	AN	BN	F-Tests and Post Hoc Comparisons (p Values)
Université Montpellier				
Learning/memory	.68	.54	.63	$F_{2,151} = 5.10$ ($p = .007$); HC >AN ($p = .005$)
Feedback sensitivity	.17	.18	.34	$F_{2,151} = 21.00$ ($p < .001$); BN > HC ($p < .001$)
Loss aversion	1.38	1.21	.92	$F_{2,151} = 3.05$ ($p = .050$); HC > BN ($p = .044$)
Choice consistency	.44	.58	.48	$F_{2,151} = 2.18$ ($p = .116$)
Johns Hopkins University				
Learning/memory		.56	.67	$F_{1,44} = 4.026$ ($p = .051$)
Feedback sensitivity		.18	.45	$F_{1,44} = 66.38$ ($p < 001$)
Loss aversion		1.48	.99	$F_{1,44} = 2.70$ ($p = .108$)
Choice consistency		.31	.43	$F_{1,44} = 1.59$ ($p = .214$)
Altrecht Eating Disorders Rintveld				
Learning/memory		.64	.51	$F_{1,22} = 2.01$ ($p = .171$)
Feedback sensitivity		.17	.42	$F_{1,22} = 29.95$ ($p < .001$)
Loss aversion		.93	.73	$F_{1,22} = .30$ ($p = .590$)
Choice consistency		.59	.57	$F_{1,22} = .02$ ($p = .892$)

conducted group comparisons of model parameters for each subsample to evaluate the consistency of results across sites. **Table 3** presents the results. Several consistent patterns of group differences were found across sites and between the combined sample and subsamples, including (1) feedback sensitivity in BN was greater than that in AN in all subsamples and greater than HC in the subsample of Université Montpellier; (2) loss aversion in BN in all subsamples was smaller than that in HC; and (3) learning in AN in all subsamples were smaller than that in HC in the subsample of Université Montpellier. Although HC participants were only recruited at Université Montpellier, their model parameter estimates were comparable to those of the healthy comparison participants in another study in which the PVL model was used.[33]

There was also some inconsistency. Loss aversion in AN in the combined sample was not significantly greater than that in HC. However, loss aversion in the Université Montpellier AN subsample was significantly lower than that in HC. Furthermore, loss aversion the Johns Hopkins University AN subsample was greater than that in HC subsample, while loss aversion in the Altrecht Eating Disorders Rintveld AN subsample was smaller than the HC.

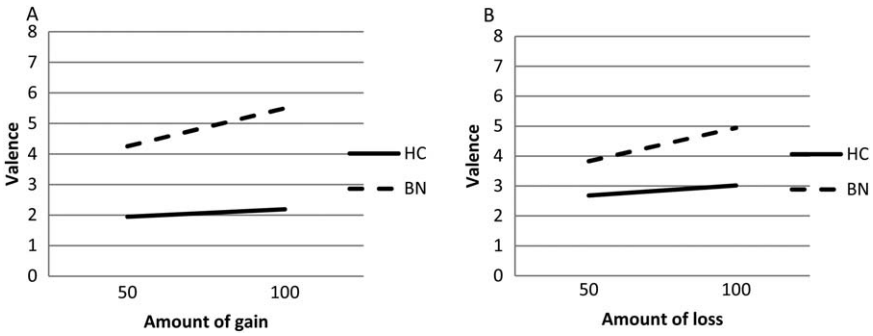
Group Comparisons Controlling for Covariates

We conducted the same group comparisons analysis controlling for depressive symptoms in the subsamples of Université Montpellier and Johns Hopkins University. We found the same patterns of results and group differences remained significant. We also conducted the same group comparisons analysis controlling for knowledge of IGT and impulsivity in the subsample of Université Montpellier and found that group differences remained significant.

Correlations Between Model Parameters and Clinical Characteristics

Among individuals with AN, BMI was positively correlated with the learning/memory parameter

FIGURE 1. (A) The association between the magnitudes of gain and valence; (B) The association between the magnitudes of loss and valence. (A and B) show the associations between gain/loss magnitudes and valences, as calculated from the estimated parameters of feedback sensitivity and loss aversion. As shown in (A), individuals with BN showed stronger sensitivity to magnitudes of gains compared to HC. Likewise, individuals with BN showed stronger sensitivity to magnitudes of loss compared to HC as shown in (B). However, they showed relatively stronger sensitivity to gains than losses. For the same amount of gains and losses, the valence was greater for gains than for losses in BN, while the valence was smaller for gains than for losses in HC.



($r = .28$, $p = .009$). Among individuals with AN in the subsamples of Université Montpellier and Johns Hopkins University, depressive symptoms were not significantly correlated any model parameters. Among individuals with AN in the subsample of Université Montpellier, impulsivity was not significantly correlated with any model parameters. However, the subdimension of impulsivity, attentional impulsivity, was significantly and negatively correlated with learning in AN ($r = -.36$, $p = .022$).

We found that BMI and model parameters were not correlated among individuals with BN. Among individuals with BN in the subsamples of Université Montpellier and Johns Hopkins University, depressive symptoms were not significantly correlated with any other model parameters. Among individuals with BN in the subsample of Université Montpellier, impulsivity was not significantly correlated with any model parameters. However, the subdimension of impulsivity, cognitive instability, was significantly correlated with response consistency and loss aversion in BN (response consistency: $r = .34$, $p = .044$; loss aversion: $r = -.41$, $p = .012$).

Discussion

Using cognitive modeling analysis, we deconstructed IGT performance into learning, motivational, and response processes and identified impairments that characterized decision making in AN and BN. Consistent with our hypothesis, decision making in AN was characterized by impaired learning/memory functions. However, inconsistent with our hypothesis, loss aversion in AN was not significantly different from HC. As hypothesized, decision making in BN was characterized by elevated sensitivity to reward as opposed to punishment, indicated by a combination of elevated feedback sensitivity and attenuated loss aversion.

Decision Making in AN

As hypothesized, decision making in AN was characterized by impaired learning/memory. This finding indicates that individuals with AN made decisions based on the experiences of more recent trials and discounted experiences of relatively distant trials. This finding is consistent with previous studies showing that memory functions are impaired in AN.[13] It has been recently suggested that IGT impairments of AN might be attributable to impaired implicit learning (i.e., learning that occurs without conscious awareness) more than impaired explicit learning.[15,16] In the present study, the learning/memory parameter derived from the model did not distinguish between implicit and explicit memory functions. Future studies might examine implicit learning with instructions that manipulate participants' use of explicit memory. Additionally, the present study showed that BMI was positively correlated with the learning/memory parameter among individuals with AN. This finding indicates that memory/learning deficits in AN might be partly associated with malnutrition.

Loss aversion was not significantly different between AN and HC. In the analysis of subsamples, the patterns of group differences in loss aversion were inconsistent. Loss aversion in the Johns Hopkins University AN subsample was greater than that in the Université Montpellier HC subsample, while loss aversion in the other two AN subsamples were smaller than that in the HC subsample. These findings did not support our hypothesis that

sensitivity to punishment would be elevated in AN. We conducted additional analysis to examine if the different proportions of the AN restrictive and binge-purge subtypes in the subsamples would explain the inconsistent pattern of results across subsamples. We found that loss aversion in the restrictive subtype was greater than that in the binge-purge subtype in both subsamples; however, the different combinations of subtypes did not explain the much higher loss aversion in the Johns Hopkins University subsample than that in the Université Montpellier subsample. These findings are inconsistent with previous findings showing elevated punishment sensitivity in AN.[18,20] It is possible that loss aversion in the present study, defined as the degree to which the experience of losses in a given trial affects decisions of following trials, might be a different construct from *punishment sensitivity*, measured as neurological and subjective reactivity to losses. It is also possible that variables not measured in this study, such as information on severity, duration, and onset of illness, might explain the inconsistent results regarding loss aversion across subsamples.

Decision Making in BN

As expected, IGT performance in individuals with BN was characterized by elevated sensitivity to reward as opposed to punishment (as depicted in Figs. **1**A and **1**B). The same results were found in the combined sample as well as across subsamples. These findings support the hypothesis that individuals with BN are more sensitive to reward than punishment. These findings are also consistent with previous studies showing that individuals with BN reported elevated sensitivity to reward[17] and showed attenuated brain activations in the reward circuitry in response to food rewards, which is believed to drive increased reward seeking behavior.[21]

Comparisons of Model Parameters Controlling for Depressive Symptoms, Impulsivity, Knowledge of IGT

Previous studies have suggested that depressive symptoms might contribute to IGT performance deficits in eating disorders.[1,6] We compared model parameters across groups controlling for depressive symptoms in the subsamples of Université Montpellier and Johns Hopkins University, and found that group differences remained significantly different in the same pattern. These findings indicated that the differences we found between groups in model parameters (in the two subsamples, which consist of 90% of the participants of the whole sample) were not fully accounted for by different levels of depressive symptoms. We did not find a significant correlation between the conventional index of IGT performance (net number of advantageous cards chosen) and depressive symptoms, nor did we find any significant correlations between depressive symptoms and model parameters. Likewise, we found the same patterns of results regarding group comparisons of model parameters after controlling for impulsivity.

Correlations Between Model Parameters and Clinical Characteristics

It has been suggested that impulsivity might be correlated with IGT performance, particularly in BN; however, impulsivity was not found to be correlated with overall IGT performance in previous studies.[2] By deconstructing IGT performance into more refined underlying processes, we found that attentional impulsivity was significantly and negatively correlated with learning in AN. This finding suggests that learning impairments in AN might be associated with the inability to inhibit orientating to distracting stimuli and to stay focused on task in hand. We also found that cognitive instability was significantly and negatively correlated with loss aversion in BN. This finding suggests that sensitivity to punishment in BN might be associated with inability to inhibit thoughts irrelevant to the task on hand.

Limitations

A limitation of this study is that the majority of the data were obtained at Université Montpellier in which IGT performance of eating disorders patients was not significantly impaired as compared to controls.[6] The relatively unimpaired performance in eating disorders patients at Université Montpellier was different from findings in most previous studies, in which IGT performances in eating disorders patients were impaired.[1–3] One might be concerned that participants in this subsample were somewhat different from other samples. However, the only noticeable difference was that this sample excluded those who were clinically depressed. Nonetheless, group comparisons controlling for depressive symptoms remained significant. Because not all participants were assessed for depressive symptoms, the invariant results after controlling for depressive symptom were limited to the subsamples of Université Montpellier and Johns Hopkins University, which, however,

contributed 90% of the participants of this study. Furthermore, the model parameter estimates of the HC group at Université Montpellier are comparable with those in another study in which the PVL model was used.[33]

Another major limitation in this study was that data were pooled from three different sites. The IGT procedure was administered in slightly different ways across sites. This variation in procedure might have affected IGT performance. However, cognitive modeling analysis takes into account of the variation in the schedules of gains and losses in estimating model parameters. Based on the nature of our analysis and the parallels in findings across subsamples, we think that the variation in IGT procedure did not have any major influence on the findings of this study. Additionally, participants at Johns Hopkins University were administered the IGT inside an fMRI scanner and were given a 5-s time limit to respond. Although we believe that the participants from Johns Hopkins University had adequate time to respond, the time limit might still have influenced IGT performance and restricted results comparability across subsamples. Nonetheless, the comparisons of model parameters conducted separately for each subsample showed consistent results across subsamples, including the elevated sensitivity reward as opposed to punishment in BN and impaired learning/memory in AN.

Furthermore, the present study cannot resolve the important question of whether impaired memory functions, altered motivational processes, and decision-making deficits are the causes of eating disorder pathology or the concomitants of other mechanisms. Answers to this question might come from future studies investigating developmental changes in these neuropsychological processes in relation to the development of eating pathology. Such studies could provide stronger evidence on how these cognitive and motivational characteristics come to be associated with anorexic and bulimic symptoms.

Finally, although the PVL model has been shown to be useful in analyzing IGT in a range of clinical populations and to have better model fit than several alternative models,[30,31] it is not the only cognitive model of IGT. Future studies might compare parameters generated by different decision-making models of IGT and examine the consistency of results across different models and studies. Additionally, the IGT used in this study did not involve actual monetary payments, which might have reduced motivation to complete the task carefully. However, hypothetical winning have shown the same patterns of activation in the brain as actual payments.[26]

Conclusions

This study demonstrates the utility of cognitive modeling analysis in identifying impairments in component processes underlying decision making in AN and BN. This approach allows for more fine-grained understanding of IGT performance deficits than the traditional summary index. This study has shown that impaired IGT performance were associated with memory deficits in AN and elevated sensitivity to reward as opposed to punishment in BN. Findings of this study suggest that future research might examine the role of impaired memory and motivational biases in the etiology and maintenance of anorexic and bulimic symptoms respectively. Clinicians working with eating disorders patients might consider how memory deficits and motivational biases play a role in patients' decision making, and how they might relate to resistance to change and maintenance of symptoms.

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Appendix A

The prospect valence learning (PVL) model deconstructs the Iowa Gambling Task (IGT) performance into four parameters: Feedback sensitivity, loss aversion, learning, and response consistency. It is theorized in the PVL model that participants make a card selection in a given trial based on their expectation of valence (i.e., an affective feeling associated with a given deck or an implicit association between a given deck and good/bad outcomes), which is formed through learning/memory of the experiences of gains and losses received following choosing a given deck in preceding trials. As indicated in Eq. (A1), the formation of valence at time t (denoted as $u(t)$) is a function of the magnitude of gains and losses (denoted as $x(t)$), loss aversion (denoted as λ), and feedback sensitivity.

$$u(t)\begin{cases} x(t)^a & if\ (x(t) \geq 0 \\ -\lambda|x(t)|^a & if\ (x(t) < 0 \end{cases} \quad (1)$$

Feedback sensitivity determines the non-linear relation between the magnitudes of gains or losses and the magnitudes of valences, as indicated in $x(t)^{\alpha}$ The non-linear relation is to account for the frequency effect—people often experience stronger reaction to losing $1 four times than losing $4 once (32). A value of 1 means that the intensity of the experience of gains/losses is in direct proportion to the magnitude of the gains/losses (i.e., no frequency effect, meaning that the person is sensitive to the magnitudes of the gains/losses). A value of 0 means that the person experiences feedback of all magnitudes equally (i.e., the frequency effect is large, meaning that losing $1 four times would be four times more unpleasant than losing $4 once). *Loss aversion* determines the relative influence of losses as opposed to gains on the formation of valence in loss trials, as indicated in $-\lambda|x(t)|^{\alpha}$ in Eq. (A1). It ranges from 0 to 5, with higher values representing higher relative sensitivity to losses as opposed to gains (i.e., more loss-aversive).

The *learning parameter*, denoted as A in Eq. (A2), determines how much the past expectancy of a given deck j ($E(t-1)_j$) is discounted in the formation of expectancy of valence of the current trial $E(t)_j$ using the decay-reinforcement learning rule.

$$E(t)_j = A \cdot E(t-1)_j + \delta_j(t) \cdot u(t) \quad (2)$$

where $\delta_j(t)$ is a dummy variable and is coded as 1 if deck j is chosen in trial t or 0 if deck j is not chosen. If deck j is not chosen in trial t, the expectancy of valence for deck j $E(t)_j$ will be a discount of the

expectancy of the previous trial $(A \cdot E(t-1)_j)$). If deck j is chosen for trial t, the expectancy of valence for deck j $E(t)_j$ will be a discount of the expectancy of the previous trial $(A \cdot E(t-1)_j)$) as well as the influence of $u(t)$, the experience of the gain and loss at trial t.

The probability of choosing each deck j is represented in Eq. (A3) based on the softmax choice rule (34)—people may not always choose the option that is associated with the best valence; they may prefer exploring other options (exploration) as opposed to adhering to the option that is best based on previous experiences (exploitation).

$$\Pr[D(t+1)=j] = \frac{e^{\theta(t)\cdot E_j(t)}}{\sum_{k=1}^{4} e^{\theta(t)\cdot E_k(t)}} \qquad (3)$$

where $\theta(t)$ in Eq. (A3) determines the degree of exploitation and is set to $3^c - 1$. c is referred as the *response consistency parameter*, which ranges from 0 to 5 (35). A higher value of c indicates that higher consistency in making decisions based on expectancies of valences while a lower value indicates random, erratic decision-making style.

Appendix B

Hierarchical Bayesian Estimation

We used hierarchical Bayesian analysis (HBA) for estimating model parameters (1). HBA is an advanced branch of Bayesian statistics employing the basic principles of Bayesian statistical inference (2,3). In the Bayesian framework, parameters θ are represented in probability distributions and have prior probabilities $\Pr(\theta)$, before any (new) evidence is considered. With new evidence (or data) D, the prior probabilities of θ are updated into posterior probabilities, $\Pr(\theta|D)$ by the Bayes' rule. HBA gives more reliable estimates of individual and group parameters by allowing for individual differences, while information across individuals was extracted.

In our formulation, the parameters of individual participants are generated from parent distributions by using independent beta distributions for each parameter:

$$A'_i \sim Beta(\mu'_A, \kappa'_A)$$
$$\alpha'_i \sim Beta(\mu'_\alpha, \kappa'_\alpha)$$
$$\lambda'_i \sim Beta(\mu'_\lambda, \kappa'_\lambda)$$
$$c'_i \sim Beta(\mu'_c, \kappa'_c)$$

Beta distributions are re-parameterized with μ_x and κ_x for each parameter x^c. μ_x is the mean of the beta distribution and κ_x represents our confidence on the distribution. κ_x determines whether the distribution is narrowly (large values of κ_x) or broadly (small values of κ_x) loaded over μ_x. α and A were limited to values between 0 and 1 ($A_i = A'_i$, $\alpha_i = \alpha'_i$), and λ and c were limited to values between 0 and 5 ($\lambda_i = 5 \cdot \lambda'_i$, $c_i = 5 \cdot c'_i$). For the prior distributions for the parameters, the uniform distributions were used for μ_x, and Gamma (1, 1) for κ_x, which are weakly informative priors.

OpenBUGS (4) was used for Markov chain Monte Carlo (MCMC) sampling and for posterior inference. A total of 100,000 samples were drawn after 100,000 burn-in samples with three chains. For each parameter, the Gelman–Rubin test (3) was run to confirm the convergence of the chains (a.k.a. Rhat). Rhat values, which show the convergence of parameters, were 1.00 for almost all parameters, and at most 1.02, which suggested MCMC chains converged to the target posterior distributions.

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