Archival Report

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Neurocomputational Mechanisms Underlying Differential Reinforcement Learning From Wins and Losses in Obesity With and Without Binge Eating

Maria Waltmann, Nadine Herzog, Andrea M.F. Reiter, Arno Villringer, Annette Horstmann, and Lorenz Deserno

ABSTRACT

BACKGROUND: Binge-eating disorder (BED) is thought of as a disorder of cognitive control, but evidence regarding its neurocognitive mechanisms is inconclusive. Key limitations of previous research include a lack of consistent separation between effects of BED and obesity and a disregard for self-report evidence suggesting that neurocognitive alterations may emerge primarily in loss- or harm-avoidance contexts.

METHODS: To address these gaps, in this longitudinal study we investigated behavioral flexibility and its underlying neurocomputational processes in reward-seeking and loss-avoidance contexts. Obese participants with BED, obese participants without BED, and healthy normal-weight participants (n = 96) performed a probabilistic reversal learning task during functional imaging, with different blocks focused on obtaining wins or avoiding losses. They were reinvited for a 6-month follow-up assessment.

RESULTS: Analyses informed by computational models of reinforcement learning showed that unlike obese participants with BED, obese participants without BED performed worse in the win than in the loss condition. Computationally, this was explained by differential learning sensitivities in the win versus loss conditions in the groups. In the brain, this was echoed in differential neural learning signals in the ventromedial prefrontal cortex per condition. The differences were subtle but scaled with BED symptoms, such that more severe BED symptoms were associated with increasing bias toward improved learning from wins versus losses. Across conditions, obese participants without BED switched more between choice options than healthy normal-weight participants. This was reflected in diminished representation of choice certainty in the ventromedial prefrontal cortex.

CONCLUSIONS: Our study highlights the importance of distinguishing between obesity with and without BED to identify unique neurocomputational alterations underlying different styles of maladaptive eating behavior.

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Binge-eating disorder (BED) is a common psychiatric condition (1,2) characterized by repetitive, subjectively uncontrollable overeating. It causes significant distress and is linked to a number of serious comorbidities such as depression, anxiety, and obesity (2–6). BED is recognized as an important public health issue (2), but the neurocognitive drivers of binge eating—as distinct from the excessive food intake without loss of control that characterizes obesity—remain poorly understood.

BED can be thought of as a disorder of cognitive-behavioral control (7–10). Consistent with this view, self-report evidence suggests that impulsivity and compulsivity are elevated in patients (11,12). However, the experimental evidence is less clear. Thus, research investigating delay discounting, risky decision making, or set-shifting abilities, for example, in BED has yielded mixed results [reviewed in (7,8,10)]. Methodological limitations may account for this inconsistency (8). Many experimental tasks lack adequate reliability (13,14), thereby reducing their power to detect differences between individuals with and without BED. Furthermore, many studies have used either a normal-weight or an obese control group [(15,16), but see (17)], but both are necessary to capture differential effects of excess weight and BED. Most studies have been cross-sectional, precluding investigations of within-participant changes associated with symptoms. Finally, previous research may not have sufficiently engaged with literature showing enhanced negative urgency in BED (12,18,19), which suggests that patients may show more impulsive behavior in harm-avoidance contexts.

To address these limitations, in this study we examined behavioral flexibility, an important aspect of cognitive-behavioral control, and its neural correlates in obese individuals with BED (BED group), obese individuals without BED (OB group), and normal-weight individuals (NW group) using a longitudinal design. We used a reversal learning task that has

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previously been used to investigate BED in conjunction with functional magnetic resonance imaging (fMRI) (20) and is known to produce reliable metrics (21). To capture potentially different behavior in reward-seeking versus loss-avoidance contexts, we introduced separate win and loss conditions. To capture within-participant changes in binge-eating symptoms and behavioral flexibility, we reinvited participants for a 6-month follow-up. To obtain more mechanistic insights, we used computational modeling using reinforcement learning models to inform our behavioral and MRI analyses.

Consistent with previous work, we hypothesized that both BED and OB groups would perform worse than the NW group due to enhanced switching between options (20,22,23). We expected this to be computationally accounted for by greater choice stochasticity (noise in the decision-making process) (20,23,24) and neurally reflected as reduced coding of learning signals—(counterfactual) prediction errors (PEs) and relative expected values—in the medial prefrontal cortex, as reported previously (20,25). Given the results of earlier studies (22,26), we further speculated that motivational context might differentially affect BED versus OB participants. Specifically, considering the role of negative urgency in disordered eating (12,18,19), we hypothesized that BED participants would perform worse than OB in the loss-avoidance condition.

METHODS AND MATERIALS

Participants and Procedure

For this substudy of a larger project (25), we initially enrolled 129 participants between 16 and 49 years (43 each of NW, OB, and BED participants) matched for age, gender, and body mass index in the case of OB and BED groups (for inclusion and exclusion criteria, see the Supplement). The presence of full-blown or subclinical BED was ascertained using the Eating Disorder Examination Interview (27) (details in the Supplement). Thirteen participants had contraindications for MRI scanning and completed the experiment outside the scanner. We excluded these participants and their matches from the analysis reported here (final n = 96). However, we report the analysis on the full sample in the Supplement and note discrepancies.

As part of the study protocol (https://osf.io/fyn6q), participants performed a probabilistic reversal learning task during fMRI (25). A minimum of 6 months after their first visit (T1), participants were reinvited for a follow-up session (T2) during which they repeated the task without MRI measurement. The interval was chosen to allow for change in binge-eating symptoms; however, due to restrictions during the COVID-19 pandemic, many participants were reassessed after a longer period of time (maximum 28 months, median = 7.85 months). Participants provided written informed consent and were financially compensated for their time (parental consent and Amazon voucher for minors). The Leipzig University ethics committee granted ethical approval (385/17-ek). For sample characteristics, see Table 1.

Task

We employed a modified probabilistic reversal learning task to assess reinforcement learning and behavioral flexibility (20,25,28–30). Our version (25) had 2 blocks of 140 trials in which participants made repeated choices between 2 cards. The cards had different probabilities of yielding a win (+10 cents) versus a neutral (±0 cents) outcome (80% and 20%) in the win block and of yielding a loss (−10 cents) versus a neutral outcome (±0 cents) in the loss block (order counterbalanced) (Figure 1A). Five times in each block, the outcome contingencies reversed, and participants had to relearn them. Neutral outcomes represent negative feedback (no win) in the win condition and positive feedback (no loss) in the loss condition. This allowed us to differentiate asymmetrical learning from valenced feedback (positive vs. negative) from asymmetrical learning for reward seeking versus loss avoidance. For additional details on the task and procedure, see the Supplement.

Analysis

Task Performance. We used trial-by-trial logistic mixed-effects models with maximal random effects (31) using the fitglme function in MATLAB (version R2023a; The MathWorks, Inc.) to estimate accuracy (probability of choosing the better card), choice switching (probability of choosing another card than in the previous trial), and perseveration (probability of choosing the same card after it has been punished twice). As predictors, we included group (NW, OB, BED, with OB as reference category), condition (win vs. loss), and previous feedback (positive vs. negative) for choice switching. Using the OB group as the reference allowed us to test the 2 comparisons central to our design: the difference between the BED and OB groups, reflecting effects of loss-of-control eating separate from effects of excess weight, and the difference between the NW and OB groups, reflecting effects of excess weight separate from loss-of-control eating. We also differentiated between prereversal trials, i.e., the trials leading up to each reversal (115 trials in total per block), and postreversal trials, i.e., the 5 trials that directly followed each reversal (25 trials per block) (for details and descriptive statistics, see the Supplement).

Computational Modeling. To assess processes underlying behavior, we fit 15 computational models (full descriptions in the Supplement). According to the winning model based on integrated Bayesian Information Criterion (Figure S1), agents learn the expected value of each card by using trial-by-trial PEs (i.e., the difference between the expected value and the actual outcome) to update the value of both the chosen option (equation 1) and the unchosen option (equation 2). The latter update requires making an inference about the outcome of the counterfactual choice (Figure 1B).

$$Q_{chosen,trial+1} = Q_{chosen,trial} + \alpha\left(\rho * reward - Q_{chosen,trial}\right) \quad (1)$$

where

$$\alpha = \alpha_{+} \text{ and } \rho = \rho_{+} \quad \forall \text{ reward} > 0$$

$$\alpha = \alpha_{-} \text{ and } \rho = \rho_{-} \quad \forall \text{ reward} < 0$$

Table 1. Demographics and Sample Characterization

	NW Group, *n* = 32	OB Group, *n* = 32	BED Group, *n* = 32	*p*
Age, Years	29.28 (±6.13)	30.26 (±6.09)	29.82 (±6.90)	.83
BMI	22.30 (±2.04)	35.54 (±3.57)	35.47 (±4.67)	.94
Follow-Up Interval, Years	0.80 (±0.50)	0.90 (±0.38)	0.76 (±0.29)	.55
Attrition, % Dropout After T1	21.88%	37.50%	34.38%	.37
Gender, Female	75.00%	75.00%	75.00%	>.99
Full-Time Education, Years	17.77 (±3.92)	16.58 (±5.11)	17.76 (±3.48)	.44
TMT-A (38)	19.69 (±4.43)	20.52 (±5.19)	19.81 (±4.95)	.77
TMT-B	41.86 (±10.64)	43.10 (±15.32)	37.24 (±8.41)	.12
Digit Span Forward (39)	6.69 (±1.26)	6.25 (±0.95)	6.45 (±1.09)	.29
Digit Span Backward (39)	5.34 (±1.36)	4.81 (±1.26)	5.26 (±1.09)	.19
Digit Symbol Substitution Task (39)	83.41 (±11.57)	81.16 (±15.19)	78.03 (±12.24)	.26
Verbal IQ, Wortschatztest (40)	109.78 (±8.88)	102.84 (±9.64)	105.31 (±5.83)	<.001
EDE-Q Binge Episodes, Last 28 Days (41)	0.32 (±1.14)	0.48 (±1.86)	6.84 (±5.04)	<.001
EDE-Q Total (41)	0.79 (±0.95)	1.67 (±1.31)	2.48 (±0.78)	<.001
EDE-Q Restraint (41)	0.74 (±0.86)	1.41 (±1.22)	1.58 (±1.01)	<.001
EDE-Q Eating Concern (41)	0.22 (±0.25)	0.62 (±0.71)	1.85 (±0.90)	<.001
EDE-Q Weight Concern (41)	1.87 (±2.24)	4.12 (±3.17)	6.06 (±1.74)	<.001
EDE-Q Shape Concern (41)	0.34 (±1.13)	0.53 (±1.36)	0.44 (±0.92)	.81
BIS-15 (42)	29.71 (±7.53)	30.03 (±6.19)	35.26 (±6.92)	<.001
UPPS Urgency (43)	24.94 (±5.38)	26.52 (±5.32)	34.32 (±5.28)	<.001
UPPS Premeditation (−) (43)	22.06 (±4.68)	21.45 (±4.43)	23.45 (±5.42)	.26
UPPS Perseverance (−) (43)	19.19 (±5.79)	18.29 (±3.36)	22.03 (±4.35)	.01
UPPS Sensation Seeking (43)	31.65 (±7.32)	31.81 (±7.25)	30.94 (±6.79)	.88
WBIS (44)	22.27 (±12.32)	39.34 (±14.60)	50.52 (±12.88)	<.001
YFAS (45)	0.16 (±0.45)	0.75 (±1.57)	3.94 (±2.57)	<.001
FCQ (46)	10.13 (±3.53)	11.69 (±3.84)	16.11 (±3.59)	<.001
BDI (47)	4.31 (±5.51)	7.84 (±5.66)	15.79 (±8.99)	<.001
STAI-Trait (48)	37.23 (±11.18)	38.97 (±9.76)	50.61 (±10.43)	<.001

Values are presented as mean (±SD) or %. *p* Values reflect 1-way analyses of variance except for BMI, which reflects a *t* test between OB and BED participants. The UPPS Premeditation (−) and Perseverance (−) subscales reflect lack of that behavior.

BDI, Beck Depression Inventory; BED, obese participants with binge-eating disorder; BIS-15, Barratt Impulsiveness Scale–Short Version; BMI, body mass index; EDE-Q, Eating Disorder Examination Questionnaire; FCQ, Food Craving Questionnaire; NW, normal-weight participants; OB, obese participants without binge-eating disorder; STAI, State-Trait Anxiety Inventory; T, time; TMT, Trail Making Test; UPPS, Urgency, Premeditation, Perseverance, Sensation Seeking; WBIS, Weight Bias Internalization Scale; YFAS, Yale Food Addiction Scale, modified version.

$$Q_{unchosen,trial+1} = Q_{chosen,trial} + \kappa\alpha\left(-(\rho * reward) - Q_{unchosen,trial}\right) \quad (2)$$

where

$$\alpha = \alpha_{-} \; and \; \rho = \rho_{+} \quad \forall \; reward > 0$$

$$\alpha = \alpha_{+} \; and \; \rho = \rho_{-} \quad \forall \; reward < 0$$

Action selection was performed by a softmax rule:

$$p(a_i) = \frac{exp(Q_{a_i})}{\sum_{j=1}^{K} exp(Q_{a_j})}$$

The model had separate learning rates (α) for positive and negative feedback and a weight (κ) on the learning rate for updates of the unchosen option. The reinforcement sensitivity parameter (ρ) determines the maximum difference between expected values and thus poses a lower bound to choice stochasticity. The model allowed for different sensitivity to positive and negative feedback, resulting in asymmetrical stay-switch behavior (e.g., with higher positive ρ, the tendency to stay after positive feedback would be stronger than the tendency to switch after negative feedback). The model showed overall good fit and recoverability (see the Supplement). We compared fitted parameters from the winning model between groups using linear mixed-effects models (using fitlme in MATLAB).

Effects of Binge-Eating Frequency. To investigate the effects of binge-eating frequency (BEF) on task performance, we repeated all generalized linear mixed-effects models in the BED group only, with average BEF across sessions and change in BEF as predictors. Change was included as the difference from the average in each session (i.e., if BEF was 6 at T1 and 4 at T2, the average would be 5, and change would be +1 at T1 and −1 at T2). This allowed for the separation of within- and between-participant effects related to symptom

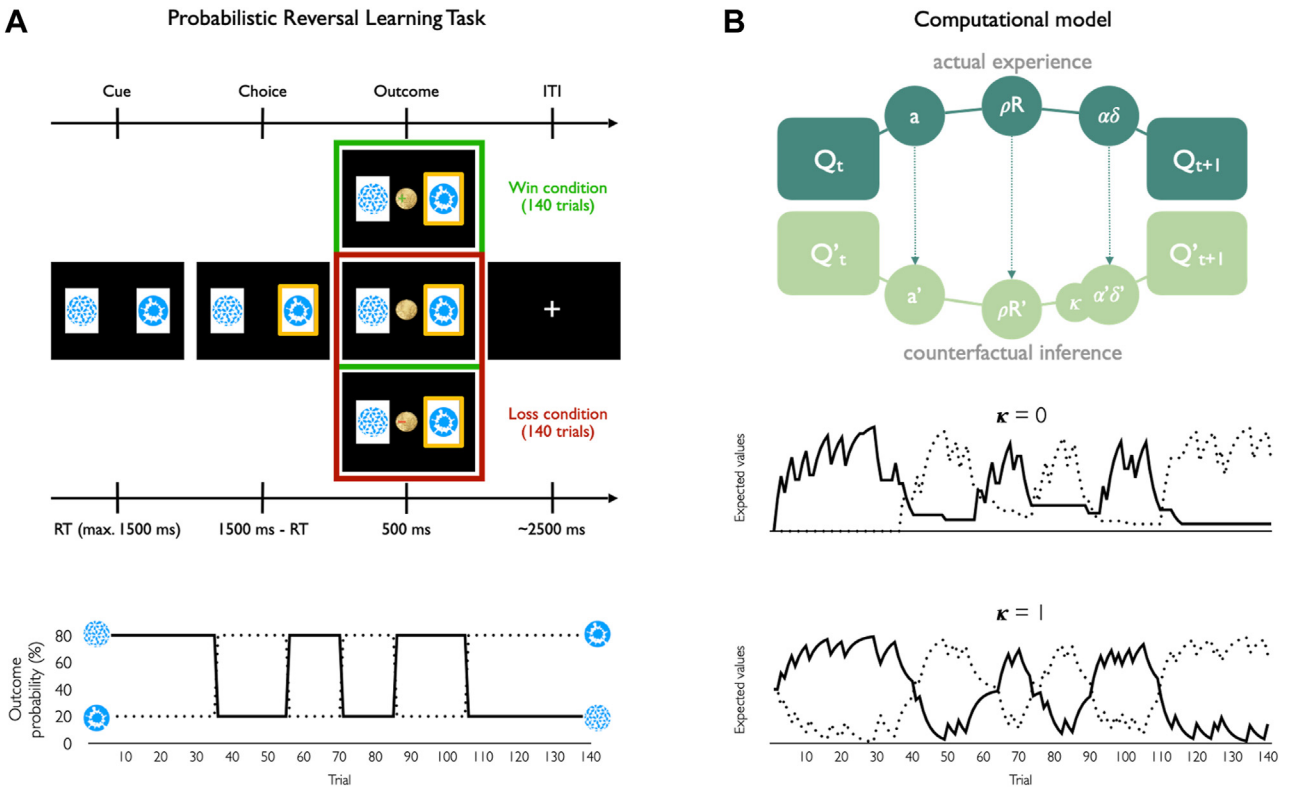

Figure 1. **(A)** Schematic of the probabilistic reversal learning task. Participants made 140 binary choices between 2 abstract stimuli (cards) with different probabilities of rewards, neutral outcomes, or losses. The goal was to gain as much money as possible and lose as little money as possible, depending on the condition (win or loss). In the win condition, a positive outcome meant gaining 10 cents, while a neutral response (±0 cents) represented a negative outcome. In the loss condition, a neutral response (±0 cents) was a positive outcome, while a negative outcome meant losing 10 cents. In each trial, the stimuli were presented for a maximum of 1500 ms or until the participant responds. A frame then appeared around the chosen card and remained visible for the 1500 ms minus the response time. Feedback was indicated through pictures of coins: a 10 cent coin for wins, a 0 cent coin for neutral outcomes, and a −10 cent coin for losses. Trials ended with a variable intertrial interval (ITI) (mean = 2500 ms) during which participants saw a fixation cross. The lower panel shows the reward contingencies. In the initial 35 trials, the stimuli had win/loss probabilities of 20% and 80%, respectively. The contingencies reversed 5 times throughout the task (after the 35th, 55th, 70th, 85th, and 105th trials), requiring participants to adapt their behavior to maximize gains and avoid losses. The order of conditions was randomized. **(B)** Upper panel: schematic of the winning computational model. Agents learn the expected value (Q) of each card based on their actions (a) and the rewards (R) that they receive at each trial. More specifically, agents use prediction errors (δ), the difference between expected values and actual outcomes, to update the values of both the chosen and the unchosen options. Notably, the latter update depends on making an inference about the outcome of the counterfactual choice. The learning rate (α) determines how much recent feedback is prioritized over older feedback, the reinforcement sensitivity (ρ) determines choice stochasticity, and the double-update weight (κ) scales the learning rate for counterfactual updates. The model had separate learning rates and reinforcement sensitivities for positive and negative feedback. Action selection was performed by a simple softmax rule. Middle panel: development of expected values when $\kappa = 0$, i.e., when no counterfactual inference took place. Lower panel: development of expected values when $\kappa = 1$, i.e., when inferred counterfactual feedback was incorporated in the same way as actual feedback. RT, reaction time.

severity. Crucially, the regression coefficient for within-participant changes reflected change in symptoms associated with a change in BEF. We removed 1 participant with an implausibly high BEF (>3 SDs from the mean, i.e., 80 binge-eating episodes/month).

Post Hoc Tests, Sensitivity, and Exploratory Analyses. For all models, we used simple effects analyses to examine interactions. We ascertained that the results were not driven by group differences in depression or anxiety in sensitivity analyses. We further explored effects of body mass index in OB and BED groups and of the Urgency, Premeditation, Perseverance, Sensation Seeking (UPPS) Impulsive Behavior Scale subscales urgency and lack of perseverance across groups. The results are reported in the Supplement.

Functional Magnetic Resonance Imaging

For scanning sequences and preprocessing steps, see the Supplement. We applied event-related analyses using the general linear model implemented in SPM12, with feedback onsets, cue onsets, missing trials, and the 6 movement parameters as regressors. We added parametric modulators informed by computational modeling as described previously (25). Thus, we added single- and double-update (DU) PEs as modulators of feedback onsets and choice probability (relative expected value of the chosen option) as modulator of cue onsets (for details, see the Supplement). Data from the win and loss blocks were analyzed in a single model, with each block modeled as a separate session. The regressors were convolved with the canonical hemodynamic response function.

For second-level analyses, we estimated random-effects analyses of variance (ANOVAs) on the contrast images of the parametric modulators. The ANOVAs included a within-participant condition factor (win vs. loss) and a between-participant group factor. Therefore, we estimated models predicting choice probability coding from group and condition and predicting PE coding from single update versus DU, group, and condition. For group comparisons, we focused on the ventromedial prefrontal cortex (vmPFC) based on previous work (20). For small-volume correction, we used a region of interest defined as a 4-mm sphere (encompassing 33 voxels) around the peak vmPFC voxel associated with valuation ([2 46 −8]) identified in a meta-analysis (32). Results were considered significant at a familywise error (FWE) small volume–corrected $p < .05$, where FWE correction was applied to the peak level.

RESULTS

Task Performance

Context-Independent Effects. There were no straightforward performance differences between groups. However, the BED and NW groups had larger differences in accuracy between pre- and postreversal trials than the OB group (BED − OB × trial type: beta = −0.25, $t_{44717} = -2.4$, $p = .02$; NW − OB × trial type: beta = −0.25, $t_{44717} = -2.49$, $p = .01$) (Figure 2A). Simple effects analyses suggested that the difference between the BED group and the OB group was mainly driven by worse performance of the BED group after reversals (BED − OB prereversal: beta = 0.29, $t_{44717} = 1.53$, $p = .13$; BED − OB postreversal: beta = −0.21, $t_{44717} = -2.49$, $p = .01$). The difference between the OB group and the NW group appeared to be driven by both relatively worse performance before reversals (NW − OB: beta = 0.34, $t_{44717} = -1.84$, $p = .07$ prereversal) and better performance after reversals (NW − OB: beta = −0.17, $t_{44717} = -2.05$, $p = .04$ postreversal) in the OB group. BED and NW groups did not differ.

The difference in accuracy between pre- and postreversal trials tracks successful learning during prereversal trials, suggesting that the OB group learned less efficiently than the NW group, with the BED group falling in between. Our analysis of choice switching corroborated this: The BED and OB groups did not differ. However, the OB group had a smaller difference in choice switching before and after reversals than the NW group (NW − OB × trial type: beta = 0.13, $t_{43943} = 2.26$, $p = .02$) (Figure 2B). Simple effects analyses suggested that this was driven by enhanced switching before (NW − OB: beta = −0.41, $t_{43943} = -1.96$, $p = .05$) but not after (NW − OB: beta = −0.15,

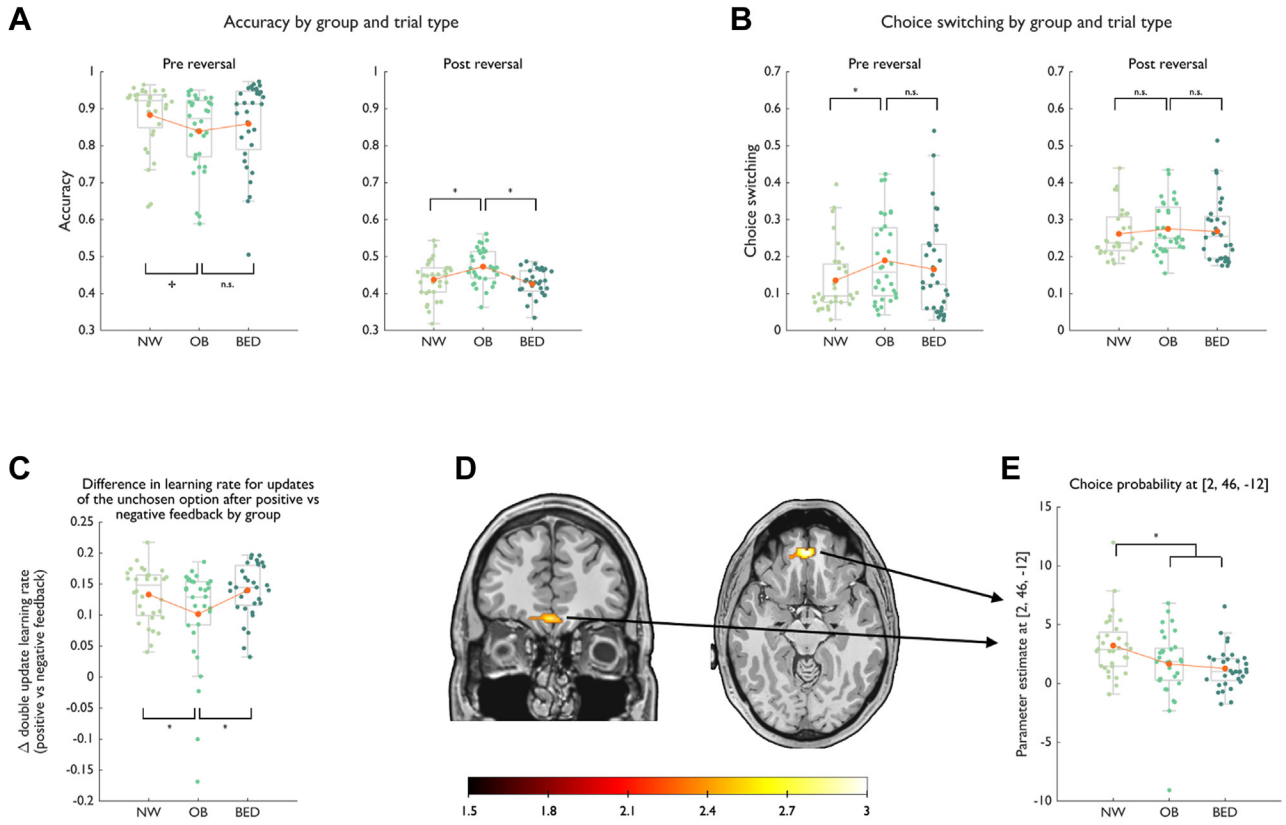

Figure 2. **(A)** Accuracy in pre- and postreversal trials by group. **(B)** Choice switching in pre- and postreversal trials by group. **(C)** Difference in double-update (counterfactual) learning rate for positive and negative feedback by group. **(D)** Ventromedial prefrontal cortex cluster reflecting the normal-weight (NW) group > (obese without binge-eating disorder [OB] and obese with binge-eating disorder [BED] groups) contrast on blood oxygen level–dependent response to choice probability ($t = 2.83$, familywise error rate-corrected p at [2, 46, −12] = .01). **(E)** Individual parameter estimates at [2, 46, −12] by group. Individual dots represent predicted values from (generalized) linear mixed-effects models, gray boxplots reflect their distribution, yellow dots and lines indicate group means. *$p < .05$, +$p < .1$. n.s., not significant.

$t_{43943} = -0.79$, $p = .43$) reversals in the OB group. BED and NW groups did not differ. Excessive switching between options, especially before reversals, reflects inefficient learning (correlation between prereversal choice switching and accuracy: $r = -0.91$, $p < .001$).

There were no context-independent differences between BED, OB, and NW groups in terms of perseveration.

Context-Dependent Effects. Motivational context had a different impact on accuracy in the BED than in the OB group (BED − OB × condition: beta = 0.12, $t_{44717} = 2.42$, $p = .02$) (Figure 3A). Thus, the BED group had similar accuracy in the win and loss conditions (condition in BED: beta = 0.02, $t_{44717} = 0.5$, $p = .62$), while the OB group performed worse in the win than in the loss condition (condition in OB: beta = −0.10, $t_{44717} = 2.94$, $p = .003$). BED and NW groups did not differ.

There were no context-dependent differences between the BED, OB, or NW groups in terms of choice switching. However, the group differences in accuracy were mirrored in perseveration (BED − OB × condition: beta = −0.28, $t_{9008} = -2.53$, $p = .01$) (Figure 3C). Simple effects analyses showed that the BED group perseverated to a similar extent in the win and loss conditions (condition in BED: beta = −0.07, $t_{9008} = -0.88$, $p = .38$), while the OB group perseverated more in the win than in the loss condition (condition in OB: beta = 0.21, $t_{9008} = 2.66$, $p = .008$). BED and NW groups did not differ.

Like excessive switching, perseveration signals ineffective learning (correlation between perseveration and accuracy: $r = -0.19$, $p = .03$). Therefore, the results suggest that in contrast to the BED group, the OB group may learn more effectively in the loss condition due to reduced perseveration.

Sensitivity Analyses. When participants without MRI measurement were included in the analysis ($n = 129$), the context-independent effects were no longer significant. However, the context-dependent group differences between BED and OB participants in accuracy and perseveration remained (accuracy: BED − OB × condition: beta = 0.09, $t_{61370} = 2.11$, $p = .04$; perseveration: BED − OB × condition: beta = −0.19, $t_{12309} = -1.98$, $p = .05$) (see the Supplement for details).

Effects of BEF

Next, we investigated how average BEF and longitudinal change in BEF were associated with task performance. Consistent with the group-level findings, results showed a context-dependent effect of average BEF on accuracy (average BEF × condition: beta = 0.14, $t_{13014} = 1.99$, $p = .05$). As Figure 3B shows, the difference in accuracy between conditions (win > loss) increased with increasing average BEF. There was no effect of longitudinal change in BEF on accuracy.

There were no effects of average BEF on choice switching. However, there was an interaction between longitudinal change in BEF and condition (beta = −0.36, $t_{12807} = -2.09$, $p = .04$) (Figure 3D). Thus, participants switched less between options in the win condition when they reported higher BEF relative to baseline (change in BEF in win condition: beta = −1.26, $t_{12807} = -3.38$, $p < .001$; change in BEF in loss condition: beta = −0.53, $t_{12807} = -1.14$, $p = .25$).

Together, the findings suggest that worse BED symptoms may be associated with a bias toward improved learning from wins versus losses, possibly due to diminishing choice switching in the win condition as symptoms worsen.

In addition, there was a complex 3-way interaction between change in BEF, previous feedback, and trial type on choice switching (for details, see the Supplement). There were no significant effects of average BEF or change in BEF on perseveration.

Computational Modeling

Context-Independent Effects. Contrary to expectations, there were no significant group differences in reinforcement sensitivities (ρ). However, BED participants had more asymmetrical learning rates (α) and DU learning rates (α*κ) for positive and negative feedback than OB participants (α: BED − OB × feedback: beta = 0.04, $t_{372} = 2.29$, $p = .02$; α*κ: BED − OB × feedback: beta = 0.02, $t_{372} = 2.53$, $p = .01$) (Figure 2C). Simple effects analyses showed a trend for slower double-update learning after negative but not positive feedback in BED participants (BED − OB in negative feedback: beta = −0.05, $t_{372} = -1.93$, $p = .06$; BED − OB in positive feedback: beta = −0.01, $t_{372} = -0.52$, $p = .60$). NW participants also had more asymmetrical DU learning rates (α*κ) for positive and negative feedback than OB participants (NW − OB × feedback: beta = 0.02, $t_{372} = 2.07$, $p = .04$; Figure 2C). This seemed to be due to slower DU learning after positive feedback and faster DU learning after negative feedback in OB participants, although the group effect was not significant for either positive or negative feedback.

Asymmetrical learning rates for positive and negative feedback make agents more resistant to uninformative (stochastic) negative feedback. Thus, reduced asymmetry between learning rates leads to less sharply distinguished expected values and therefore to choice switching (correlation between DU learning rate asymmetry and choice switching: $r = -0.36$, $p < .001$). Thus, the findings suggest that reduced asymmetry of DU learning rates for positive and negative feedback may account for our finding of enhanced choice switching in OB participants.

Context-Dependent Effects. Across groups, participants had higher reinforcement sensitivities (ρ), i.e., behaved less noisily, in the loss condition (condition: beta = −0.03, $t_{380} = -2.21$, $p = .03$). However, there were no group differences.

BED and OB groups differed significantly in terms of their learning rates (α) in the win and loss conditions (BED − OB × condition: beta = 0.02, $t_{372} = 2.09$, $p = .04$). BED, but not OB, participants had lower learning rates in the loss than in the win condition (condition in BED: beta = 0.1, $t_{372} = 2.41$, $p = .02$; condition in OB: beta = −0.004, $t_{372} = -0.54$, $p = .58$). This pattern did not quite match our observations at the behavioral level. Therefore, we reasoned that a lower learning rate in the loss condition in the BED group might compensate for higher reinforcement sensitivity in the loss condition across groups. The result would be higher learning sensitivity in the loss condition in the OB compared with the BED group, possibly accounting for improved performance in this condition.

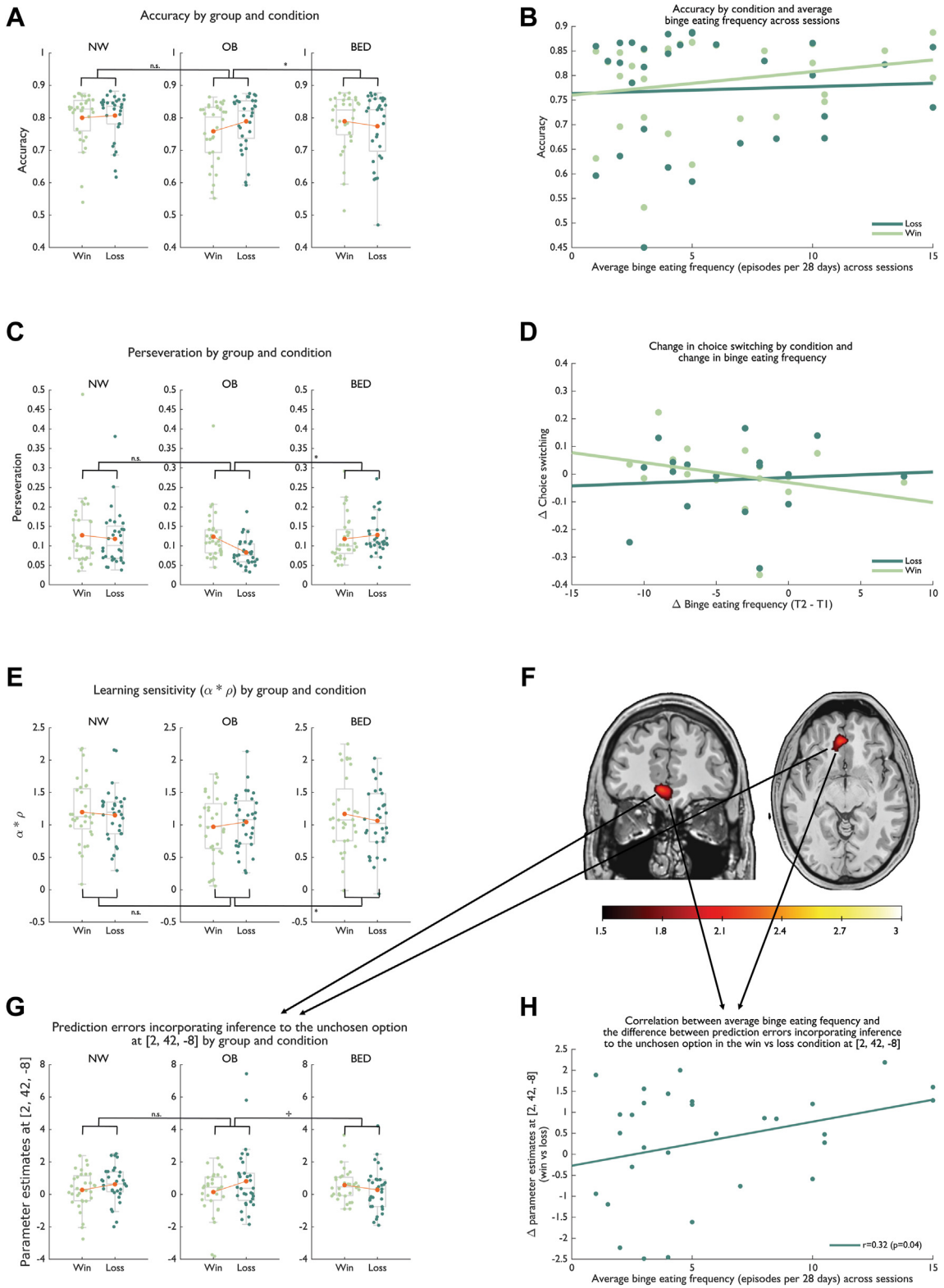

Figure 3. **(A)** Accuracy by condition and group. **(B)** Accuracy by condition and average binge-eating frequency (episodes per 28 days) within the obese with binge-eating disorder (BED) group. **(C)** Perseveration by condition and group. **(D)** Change in choice switching by condition and change in binge-eating frequency across sessions. **(E)** Learning sensitivity, the product of learning rate and reinforcement sensitivity, by condition and group. **(F)** Ventromedial prefrontal

Therefore, we calculated the product of learning rate and reinforcement sensitivity ($\rho^*\alpha$, the learning sensitivity). BED participants had higher learning sensitivity for wins than losses, while OB participants had higher learning sensitivity for losses than wins (BED − OB × condition: beta = 0.09, t_{372} = 2.06, p = .04) (Figure 3E). Critically, learning sensitivity ($\rho^*\alpha$) was highly correlated with accuracy (r = 0.82, $p < .001$). Thus, differences in learning sensitivities in the win and loss conditions may account for context-dependent differences in accuracy between the BED and OB groups.

There were no context-dependent differences in double-update learning rate between BED, OB, and NW groups. There were no associations between BEF and model parameters.

Sensitivity Analyses. The results did not change when we excluded 7 participants fit at chance level. When participants without MRI measurement were included during fitting (n = 129), the difference between BED and OB participants in learning sensitivity for wins and losses and learning rates for positive and negative feedback were still significant (for details, see the Supplement). The other group differences were no longer significant.

fMRI Results

Next, we explored how our findings of group differences at the behavioral and computational level were reflected in the coding of model-derived learning signals in the brain (for group-level results, see the Supplement).

Context-Independent Effects. The BED and OB groups showed less activation associated with choice probability in the vmPFC than the NW group (t = 2.83, FWE small volume–corrected p at [2, 46, −12] = .014) (Figure 2D, E). Because choice probability can be understood as reflecting confidence in the upcoming choice, this is consistent with our findings at the behavioral and computational level: OB showed enhanced switching, which we have argued may be a consequence of less sharply distinguished expected values due to less asymmetrical learning from positive and negative feedback. There were no other context-independent group differences.

Context-Dependent Effects. There was a marginal interaction between DU and single update, condition, and group with respect to PE coding in the vmPFC (t = 2.16, FWE small volume–corrected at [2, 42, −8] p = .063) (Figure 3F, G). Activation in response to DU PEs was stronger in the win condition in BED participants and stronger in the loss condition in OB participants. That is, the neural learning signal incorporating counterfactual inference was more pronounced in the win than in the loss condition in BED participants, and the opposite was true in OB participants. Importantly, this signal was also correlated with average BEF in BED participants, such that more binge-eating episodes were associated with larger differences in DU signal between the win and loss conditions (r = 0.32, p = .04) (Figure 3H). This echoes our findings of higher learning sensitivity in reward-seeking versus loss-avoidance contexts in BED participants and more asymmetrical learning from wins versus losses in participants with more frequent binge-eating episodes. There were no other context-dependent group differences.

DISCUSSION

In this study, we used a probabilistic reversal learning task, computational modeling, and fMRI in a longitudinal design to investigate shared and distinct neurocognitive mechanisms of altered decision making in the BED and OB groups compared with the NW group.

We demonstrated subtle differences between the BED and OB groups with regard to learning in different motivational contexts. Thus, unlike BED participants, OB participants performed better when learning for loss avoidance (loss condition) than reward seeking (win condition), putatively owing to less perseveration. This is broadly consistent with our hypotheses regarding BED but somewhat at odds with previous reports of difficulty with learning from losses in obesity (33,34). However, the samples used in Kostner *et al.* (33) and Coppin *et al.* (34) were not screened for BED and may have been confounded, which is something that the current study was designed to avoid. For our data, computational modeling suggested that the condition-specific performance difference may reflect relatively enhanced learning sensitivity (product of reinforcement sensitivity and learning rate) in the loss condition in the OB group and relatively reduced learning sensitivity in the loss condition in the BED group. Consistent with this, a neural learning signal incorporating counterfactual inference in the vmPFC was comparatively stronger in the loss condition in the OB group and comparatively weaker in the loss condition in the BED group. This effect was only marginally significant. However, reduced coding of counterfactual PEs in the vmPFC has previously been shown to characterize BED (20), and the signal was correlated with BEF, with greater differences between conditions (win > loss) being associated with higher frequencies. This mirrored the association between BEF and the difference in performance between the win and loss conditions, where higher frequencies were also associated with greater differences between conditions. In addition, and consistent with this, worsening symptoms were associated with less choice switching in the win but not in the loss condition. In sum, the results suggest that the BED and OB groups may be characterized by different neurocognitive learning biases, with better learning from wins than losses in the BED group and better learning from losses than wins in the OB group.

Independent of motivational context, OB participants showed more choice switching, particularly before reversals, leading to

cortex cluster reflecting the single vs. double update by condition by group (obese without binge-eating disorder [OB] vs. BED) contrast on blood oxygen level–dependent response to prediction errors (t = 2.16, familywise error rate-corrected p at [2, 42, −8] = .06). **(G)** Individual parameter estimates at [2, 42, −8] by group. **(H)** Correlation between individual parameter estimates at [2, 42, −8] and average binge-eating frequency across sessions in the BED group. Individual dots represent predicted values from (generalized) linear mixed-effects models, gray boxplots reflect their distribution, yellow dots and lines indicate group means. *$p < .05$, +$p < .1$. n.s., not significant; NW, normal-weight individuals; T, time.

worse performance in OB compared with NW participants before and better performance after reversals compared to BED and NW participants. Computationally, switching was accounted for by less asymmetrical counterfactual learning rates for positive and negative feedback in OB participants compared with BED and NW participants. A lack of asymmetry makes agents more sensitive to uninformative feedback and thus leads to less sharply distinguished expected values and enhanced choice switching. At the neural level, obese participants (with and without BED) showed reduced coding of choice probability—a reflection of the difference between expected values—in the vmPFC. The effects were small but resonate with previous reports of enhanced switching in OB participants (23). The absence of discernible differences between BED and OB participants in prereversal accuracy, switching behavior, and fMRI observations suggest a general effect of obesity irrespective of BED. However, given group differences in postreversal accuracy and model parameters that diverge from this trend, we hesitate to draw definitive conclusions.

Together, our results indicate that obesity in the context of BED may vary qualitatively from obesity without loss-of-control eating. There may be a bias toward worse learning from losses than wins in BED participants and vice versa in OB participants. This fits with the clinical picture of BED, where patients repeat actions that they know will make them feel bad. It is also consistent with demonstrably enhanced negative urgency in this group (18,19,35), which may disturb learning and decision making for loss avoidance. There is evidence that inhibitory control and risk taking in BED are affected by negative mood (36,37). It would be interesting to test whether this extends to the reinforcement learning realm by experimentally manipulating stress or mood before task performance in the lab or via more ecological, smartphone-based approaches.

Our results are intriguing but not without limitations. The effects we showed are subtle, complex, and not always easily interpreted. Importantly, while we saw specific differences between BED and OB participants, the BED and NW groups did not differ significantly from one another. This raises the question of whether the BED − OB differences are driven by BED-specific alterations or a normalization of obesity-associated alterations. While our analysis of the effects of binge-eating severity and the absence of context-specific differences between OB and NW groups suggest the former, more research in large clinical multicenter samples will be necessary to replicate and clarify group differences and properly disentangle the dimensional effects of body mass index and binge-eating severity. Our data yielded important leads, but the groups were too small to produce sufficiently dependable dimensional estimates, let alone to investigate potential nonlinear effects, which may be especially interesting with regard to obesity without BED (38). Likewise, our evidence is confined to the monetary domain; however, food rewards may be processed differently.

Conclusions

Our data suggest that reinforcement learning in obesity with and without BED may be subject to qualitatively different neurocomputational learning biases. Thus, individuals with BED may have a bias toward worse learning from losses than wins, and obese individuals without BED may have a bias toward worse learning from wins than losses. Furthermore, obesity without BED was associated with reinforcement learning difficulties due to enhanced choice switching. Our findings highlight the importance of distinguishing between obesity with and without BED.

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LD and AH designed the study and acquired funding; NH and MW acquired the data; and MW and LD analyzed the data and wrote the original draft of the manuscript. All the authors reviewed and edited the manuscript.

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ARTICLE INFORMATION

From the Department of Child and Adolescent Psychiatry, Psychosomatics and Psychotherapy, Centre of Mental Health, University of Würzburg, Würzburg, Germany (MW, AMFR, LD); Max Planck Institute for Human Cognitive and Brain Sciences, Leipzig, Germany (MW, NH, AV, AH, LD); CRC-940 Volition and Cognitive Control, Faculty of Psychology, Technical University of Dresden, Dresden, Germany (AMFR); Department of Psychology, Julius-Maximilians-University of Würzburg, Würzburg, Germany (AMFR); MindBrainBody Institute, Berlin School of Mind and Brain, Charité-Universitätsmedizin Berlin and Humboldt, Universität zu Berlin, Berlin, Germany (AV); Department of Psychology and Logopedics, Faculty of Medicine, University of Helsinki, Helsinki, Finland (AH); and Department of Psychiatry and Psychotherapy, Technical University of Dresden, Dresden, Germany (LD).

Address correspondence to Maria Waltmann, Ph.D., at waltmann_m@ukw.de.

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