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Re-evaluating the predictive value of reinforcement motivation on outcome in anorexia nervosa: The role of affective symptoms

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Abstract

Background. Reinforcement motivation, particularly heightened punishment sensitivity and its imbalance relative to reward sensitivity ("motivational bias"), can predict outcomes in anorexia nervosa (AN). However, previous studies did not account for affective symptoms, which are intertwined with reinforcement processes and illness trajectory. This study examined whether affective symptoms explain outcomes beyond reinforcement motivation.

Methods. Adolescent girls and young women with AN (n = 192; M_{age} = 16.33 years, SD = 2.61) completed self-report measures of punishment/reward sensitivity, depressive symptoms, and trait anxiety at treatment admission and at 1-year follow-up. Outcomes included ED symptoms, body mass index, and multidimensional recovery (Morgan–Russell outcome assessment schedule). Cross-sectional comparisons included long-term recovered individuals (n = 154) and healthy controls (n = 440); longitudinal associations were tested using nested linear regression.

Results. Patients with AN and recovered individuals showed elevated punishment sensitivity (η^2_p = 0.19) and stronger motivational bias (punishment>reward; η^2_p = 0.17). At baseline, reinforcement motivation and affective symptoms showed limited predictive utility. Longitudinally, shifts in motivational bias were associated with ED symptoms and multidimensional outcomes ($|\beta| > 0.248$, $p_{FDR} < .002$), whereas changes in affective symptoms were the most robust and consistently associated predictors ($|\beta| > 0.173$, $p_{FDR} < .017$).

Conclusions. Depressive symptom improvement emerged as the key predictor of outcome, showing stronger associations with follow-up ED symptoms and psychosocial functioning than reinforcement motivation. While changes in reinforcement motivation accompanied ED outcomes, affective symptom change was more predictive across outcomes. These findings emphasize the importance of prioritizing affective processes within developmentally tailored AN treatment frameworks.

Introduction

Anorexia nervosa (AN) is a psychiatric illness characterized by an intense fear of weight gain, extreme food restriction, and low body weight (American Psychiatric Association, 2022). It commonly emerges during adolescence and disproportionately affects female individuals (Smink, Van Hoeken, & Hoek, 2012). AN is associated with severe medical complications (Mehler & Brown, 2015), psychosocial impairment (Ágh et al., 2016), comorbid affective psychopathology (Marzola, Fassino, Amianto, & Abbate-Daga, 2017; Pollice, Kaye, Greeno, & Weltzin, 1997), and has one of the highest mortality rates among psychiatric disorders (Krug et al., 2025). Even with evidence-based treatments, outcomes in AN are often unsatisfactory and relapse is common (Brockmeyer, Friederich, & Schmidt, 2018; Bruns, Meier, & Jansen, 2025). Understanding the risk and maintenance factors underlying AN is essential for developing targeted interventions and improving outcomes (Schmidt & Treasure, 2006; Stice, 2002).

Among risk and maintenance factors in AN, reinforcement motivation, especially sensitivity to reward and punishment, has received considerable attention (Harrison, O'Brien, Lopez, & Treasure, 2010; Haynos et al., 2020). Gray's Reinforcement Sensitivity Theory (2000) describes two neurobiological systems guiding behavior: the behavioral activation system (BAS), promoting reward approach, and the behavioral inhibition system (BIS), mediating avoidance of

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potential punishment or conflict. Converging behavioral and neuroimaging evidence suggests that in AN, approach responses to rewards, particularly food and social stimuli, are often diminished, while avoidance of potential punishment (e.g. weight gain or aversive emotional states) is typically heightened. Both are shaped by contextual factors such as controllability and effort (Brown, Nuñez, & Wierenga, 2024; Haynos et al., 2020; Wierenga et al., 2014). This imbalance may reduce the motivational pull of rewarding stimuli while amplifying the drive to avoid threats, thereby maintaining restrictive eating. Importantly, AN emerges during a neurodevelopmental period of ongoing frontostriatal maturation (Hagan, Lloyd, & Gorrell, 2026), suggesting that normative developmental processes may interact to shape illness trajectory.

Studies employing self-report measures derived from Reinforcement Sensitivity Theory, including the BIS/BAS scales (Carver & White, 1994) and the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ; Torrubia, Ávila, Moltó, & Caseras, 2001), consistently report elevated punishment sensitivity in individuals with AN, and often blunted reward sensitivity, compared with healthy controls (HC; Claes et al., 2021; Glashouwer et al., 2014; Harrison et al., 2010, 2011; Jappe et al., 2011; Matton, Goossens, Vervaet, & Braet, 2015; Monteleone et al., 2014; Shott et al., 2012; Uniacke et al., 2024; Via et al., 2014). Elevated punishment sensitivity is also observed after weight restoration and in long-term recovered individuals (Harrison et al., 2016; Harrison et al., 2011; King et al., 2022), suggesting it may represent a trait-like vulnerability. Together with evidence from diverse cognitive tasks (Bernardoni et al., 2021; Bischoff-Grethe et al., 2013; Christian, Hudson, Bell, & Goldschmidt, 2025; Frank, Collier, Shott, & O'Reilly, 2016; Jonker, Bennik, & de Jong, 2018; Matton et al., 2017; Uniacke et al., 2024; Wierenga et al., 2022; Wierenga, Brown, & Reilly, 2025), this raises questions about the prognostic relevance of reinforcement motivation for treatment outcomes.

Recent studies investigated whether reinforcement motivation can predict clinical outcomes in AN. Jonker, Glashouwer, and de Jong (2022) found that adolescents with higher self-reported punishment sensitivity at the beginning of treatment showed less improvement in eating disorder (ED) symptoms at a one-year follow-up, and those who showed a longitudinal decrease in punishment sensitivity had greater ED symptom improvement. Complementing these findings in a large sample of adolescents and adults with AN, Abber, Murray, Brown, and Wierenga (2024) reported that patients showed a stronger "motivational bias" toward punishment sensitivity (relative to reward sensitivity) compared to HC. Moreover, reductions in motivational bias during partial hospitalization, more so than changes in reward sensitivity, were associated with ED symptom improvement at discharge (after ~100 days). Critically, however, these studies did not account for co-occurring affective symptoms including depression and anxiety. This is a notable limitation given the high prevalence of affective symptoms in AN, their established associations with punishment- and reward sensitivity generally (Katz, Matanky, Aviram, & Yovel, 2020) and with ED symptoms specifically (Marzola et al., 2017; 2020; Sala, Egbert, Lavender, & Goldschmidt, 2018), and the well-documented finding that comorbid affective symptoms are associated with worse long-term outcomes (Solmi et al., 2024; Steinhausen et al., 2021). Beyond serving as a broad severity marker, specific subcomponents of affect may act as mechanisms of change. For instance, developmental research highlights that in adolescents, domains of anhedonia such as deficits in anticipatory pleasure and motivational engagement can impair the capacity for behavioral change (Ma, Sahni, & McCabe, 2026). In AN, such affective disturbances may perpetuate disordered eating behaviors by reducing the incentive value of rewards outside the illness domain.

Building on Jonker et al. (2022) and Abber et al. (2024), the present study tested two related hypotheses: (1) whether reinforcement motivation (as assessed with the BIS/BAS scales) predicts one-year clinical outcomes in adolescent girls and young women with AN who underwent inpatient treatment while controlling for depression and trait anxiety, and (2) whether co-occurring affective symptoms provide predictive value for clinical outcomes beyond, or more strongly than, reinforcement motivation alone. In addition to examining longitudinal changes in ED symptoms (assessed with the Eating Disorder Inventory 2 (EDI-2; Paul & Thiel, 2005) and age-adjusted BMI, we also assessed multidimensional Morgan-Russell outcome assessment schedule (MROAS; Morgan & Hayward, 1988) scores to better capture broader recovery outcomes as recently emphasized (Phillipou et al., 2025). Addressing these questions is critical for refining mechanistic models of AN and for identifying more precise intervention targets.

Methods

Participants

Data from 869 female adolescents and young women (12–29 years old) were analyzed. The primary longitudinal sample included 192 acutely underweight patients with AN (M_{age} = 16.33 years, SD = 2.61; 89.6% restrictive subtype), diagnosed according to DSM-V (American Psychiatric Association, 2022), who were initially assessed within 96 h after admission to behaviorally-oriented nutritional rehabilitation programs at the University Hospital Carl Gustav Carus Dresden (baseline; AN-T1) and again at a one-year follow-up (AN-T2; M_{days} = 393.2, SD = 22.4 days following initial assessment). Mean treatment duration was 115.3 days (SD = 38.1). While 60.4% of the longitudinal sample could be considered fully weight restored at follow-up (BMI-SDS ≥ −1), 39.6% had not yet achieved or maintained this status.

For cross-sectional comparisons with the AN sample at baseline, data from 154 long-term recovered individuals with a history of AN (recAN; M_{age} = 22.47 years, SD = 3.41; 64.9% restrictive subtype during illness) and 440 HC (M_{age} = 19.08 years, SD = 4.53) were included. A subset of recAN participants (n = 18) also participated during acute illness at baseline (AN-T1) and at the one-year follow-up (AN-T2). Study procedures were approved by the Ethics Commission of the TU Dresden, and written informed consent was obtained from all participants (and their legal guardians if underage). Further participant details and preregistration information are provided in the Supplementary Material.

Diagnostic procedures

Current and past ED symptoms were assessed in all participants by trained research assistants with the Structured Interview for Anorexia and Bulimia Nervosa (SIAB-EX; Fichter & Quadflieg, 2001). At baseline, patients with AN had a BMI <17.5 kg/m^2 (or below the 10th age percentile if younger than 18 years) and had no sustained recent weight gain (>2.5 kg within the past 4 weeks or 2 kg within the past 2 weeks). RecAN participants (1) maintained a BMI >18.5 (or above the 10th age percentile if younger than 18 years), (2) menstruated, and (3) reported not engaging in any disordered eating behaviors as assessed with the SIAB-EX for at least 6 months prior to participation (duration of recovery: M_{years} = 4.39, SD = 3.1). HC participants had a BMI within 18.5–28 kg/m^2 (or between the

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10th and 94th age percentile if younger than 18 years), were eumenorrheic, and had no lifetime BMI <17.5 kg/m^2 (or below the 10th percentile if younger than 18 years) or history of psychiatric illness as assessed with the Mini International Neuropsychiatric Interview (Sheehan et al., 1998, 2010). See Supplementary Material for details on participant exclusion criteria, including medication use and medical or psychiatric conditions.

Eating disorder and associated symptom assessment

"Core" cognitive-behavioral ED-related symptoms were assessed by averaging the Drive for Thinness, Body Dissatisfaction, and Bulimia subscales of the EDI-2 (Paul & Thiel, 2005), depressive symptoms with the Beck Depression Inventory (BDI-II; Hautzinger, Keller, Beck, & Kühner, 2009), and trait anxiety symptoms with the corresponding subscale of the State and Trait Anxiety Inventory (STAI; Laux, Glanzmann, & Spielberger, 1981) as detailed in the Supplementary Material. Missing responses on these questionnaires prevented calculating summary scores for a small number of participants (<6% per questionnaire). No imputation was performed; analyses were conducted using complete cases only, consistent with the degrees of freedom reported in the relevant data tables. BMI standard deviation scores (BMI-SDS) were computed as an age- and sex-corrected weight-to-height ratio index (Hemmelmann et al., 2010; Kromeyer-Hauschild et al., 2001).

Reinforcement motivation assessment

Punishment and reward sensitivity were assessed with the German version of the BIS/BAS questionnaire (Strobel, Beauducel, Debener, & Brocke, 2001) including 24 items (four dummy items) gauging dispositional tendencies of avoidance and approach motivation according to Gray's Reinforcement Sensitivity Theory (Gray, McNaughton, & MacNaughton, 2000) on a scale from 1 ("very false for me") to 4 ("very true for me"). Seven items measure avoidance motivation (e.g. "Criticism or scolding hurts me quite a bit") on a punishment sensitivity subscale (BIS) and 13 items measure approach motivation on three reward-related subscales including reward responsiveness (five items, e.g. "When good things happen to me, it affects me strongly"), reward drive (four items, e.g. "I go out of my way to get things I want") and fun seeking (four items, e.g. "I crave excitement and new sensations"), which together build a total reward sensitivity score (BAS; Supplementary Material). Following a recent study (Abber et al. 2024), we also calculated motivational bias scores quantifying the relative balance of punishment- and reward sensitivity. To this end, we first scaled BIS to BAS scores (given that the range of possible scores differs between the measures) and then subtracted the scaled punishment sensitivity scores from the reward sensitivity scores as in prior studies (Sutton & Davidson, 1997; Tomer et al., 2014). Consequently, positive motivational bias scores indicate higher BAS relative to BIS scores, reflecting greater reward sensitivity and approach motivation, while negative scores denote higher BIS relative to BAS scores, reflecting greater punishment sensitivity and avoidance motivation.

Clinical outcome

In addition to assessing ED-related cognitive-behavioral symptoms (EDI-2 core subscales) and physical characteristics (BMI-SDS) as outcome measures, as in recent related studies (Abber et al., 2024; Jonker et al., 2022; Jonker, Glashouwer, & de Jong, 2025), we also employed the MROAS (Morgan & Hayward, 1988), a well-established and validated semi-structured interview (Dobrescu et al., 2020) evaluating factors including nutritional status, menstrual function, mental state, and socioeconomic status as a multidimensional outcome measure. Given our relatively young sample and the questionable suitability/implicit heteronormativity of items assessing sexual identity/relationships for minors (Nagamitsu et al., 2019), we excluded the sexual adjustment subscale and calculated an adjusted mean global MROAS score (range 0–12; higher scores indicate better overall outcome) based on the remaining subscales as in previous studies (Arold et al., 2023; Boehm et al., 2021; Lange et al., 2019). MROAS data were missing for one participant in the longitudinal sample.

Statistical analysis

Analyses were conducted in SPSS 30.0 (IBM Corp., 2024) and R 4.3.3 (R Core Team, 2024). We first tested for change in clinical and psychological variables of interest between baseline and follow-up within the longitudinal AN sample at the focus of this study and for cross-sectional differences between the baseline AN, recAN, and HC groups. Importantly, we verified the representativeness of the main longitudinal AN sample by comparing baseline characteristics between participants who completed assessments at both time points and those for whom only baseline data were available (Supplementary Table S2). Next, given established associations between reinforcement sensitivity, affective and eating disorder symptoms (Haynos et al., 2020; Katz et al., 2020), we explored interrelationships among key variables in the longitudinal AN sample by computing Pearson's zero-order correlations at baseline (T1), follow-up (T2), and for change (Δ) scores, calculated as T1–T2. These measures included BIS, BAS, and motivational bias scores as primary predictors; EDI-2, BMI-SDS, and MROAS scores as outcome variables; and BDI-II and STAI-T scores as psychologically relevant factors. The resulting correlation matrices were inspected to explore whether motivational orientation was associated with symptoms/outcomes as in prior research (Abber et al., 2024; Katz et al., 2020; Marzola et al., 2017).

Our main objective was to assess the predictive value of reinforcement motivation on clinical outcomes in the longitudinal AN sample while accounting for concurrent affective symptoms. To this end, we first applied the same basic regression approach used in prior work (Abber et al., 2024; Jonker et al., 2022), examining (1) whether baseline BIS and motivational bias scores predicted one-year follow-up ED symptoms (T2 EDI-2), BMI-SDS (T2 BMI-SDS) and MROAS scores; and (2) whether longitudinal changes in BIS and motivational bias over the same one-year period were associated with these outcomes. Critically, however, our main analyses extended this framework by employing and comparing several increasingly complex nested linear regression models to account for the established relationship between depressive and anxiety symptoms in ED psychopathology and treatment response (Bulik, 2002; Godart, Flament, Perdereau, & Jeammet, 2002; Mattar et al., 2011; Pleplé et al., 2021; Pollice et al., 1997). In detail, models were fitted by entering either baseline or longitudinal change in BIS or motivational bias as predictors, together with age and the corresponding baseline values of the outcome variable (EDI-2 and BMI-SDS; Step 1). For all outcomes, Step 1 was followed by the addition of baseline or longitudinal change in BDI-II scores in Step 2 and STAI-T scores in Step 3. These nested models were compared to determine whether affective symptoms significantly improved model fit beyond reinforcement-related predictors. An overview of all compared regression models is provided in Supplementary Table S3. For MROAS scores, which were measured only at

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follow-up, models included only the BIS or motivation bias predictors (Step 1) in addition to age. We compared nested models using *F*-tests, with AIC and BIC indices used to evaluate model parsimony and explanatory power (Supplementary Methods). Only models in which the addition of affective predictors improved model fit were retained for subsequent analyses and presentation of the extended models. Further, they were expanded to include two-way interactions between depressiveness and motivational predictors in order to explore possible non-additive effects of these key predictors. Corrections for multiple testing (Benjamini & Hochberg, 1995) were applied to each group of nested models during model comparison, and again to predictors within the final models. To account for multicollinearity, we examined variance inflation factors (VIF) for all regression models and further report the increment to the outcome prediction (ΔR^2) for each model predictor. To further examine the longitudinal predictive value of baseline motivational and affective characteristics for outcomes, we additionally fitted cross-lagged panel models (Supplementary Material).

Results

Table 1 summarizes the descriptive statistics for the longitudinal AN sample and the cross-sectional recAN and HC groups, along with inferential comparisons across key variables. As expected, patients with AN exhibited elevated depressive symptoms and trait anxiety relative to HC, which improved at follow-up but remained above HC levels, a pattern also observed in recAN. Also in line with previous findings (Glashouwer et al., 2014; Harrison et al., 2010; 2011; Haynos et al., 2020; Jappe et al., 2011), patients with AN showed lower reward sensitivity (BAS) and elevated punishment sensitivity (BIS) relative to HC, consistent with a motivational bias marked by heightened sensitivity to punishment relative to reward (Abber et al., 2024). None of the reinforcement motivation measures changed over time in the longitudinal sample and differences relative to HC persisted at follow-up. Also of note, while punishment sensitivity (BIS) remained elevated in recAN and motivational bias (reflecting higher punishment relative to reward sensitivity) was also more pronounced relative to HC (who showed higher reward relative to punishment sensitivity), no group difference in reward sensitivity (BAS) was detected.

Baseline comparisons between participants with AN who did and did not complete follow-up showed no significant differences (Supplementary Table S2), indicating that attrition did not bias the longitudinal sample.

Correlations between reinforcement sensitivity, symptoms, and outcomes

Consistent with previous research (Abber et al., 2024; Haynos et al., 2020; Katz et al., 2020), reinforcement sensitivity showed consistently significant associations with both eating disorder and affective symptoms ($rs \geq .27$, all $ps < .001$), while BMI remained largely uncorrelated with psychological measures (except a positive link to MROAS scores) across baseline and follow-up assessments. Detailed correlation matrices for both time points and change scores are shown in Supplementary Tables S4–S6).

Testing alignment with the literature

Analogous to Jonker et al. (2022) and Abber et al. (2024), basic regression analyses first examined the predictive value of motivational parameters (BIS, motivational bias) on outcomes (T2 EDI-2, T2 BMI-SDS, MROAS), without including affective symptom measures. We calculated change in motivational bias (reflecting longitudinal change in the relative balance between punishment- and reward sensitivity) as baseline minus follow-up (T1–T2), such that positive values indicate a shift toward relatively greater punishment relative to reward sensitivity over time. Results revealed that neither baseline BIS nor baseline motivational bias scores predicted any outcome measure (Table 2). This was also the case when change in BIS was used as a predictor (Models 1C.1, 2C.1, 3C.1; Table 3). Motivational bias change was positively associated with T2 EDI-2 scores and negatively associated with follow-up MROAS (but not T2 BMI-SDS), indicating that a greater shift in motivational bias toward punishment sensitivity was associated with higher ED symptoms and less favorable multidimensional recovery outcomes (Models 1D.1, 3D.1; Table 3). Also of note, higher age at baseline negatively predicted follow-up BMI-SDS (Models 2A.1, 2B.1, 2C.1, 2D.1) and, in one case, MROAS (Model 3D.1). In all models, VIFs remained acceptably low (≤1.2).

Main regression analyses controlling for affective symptoms

Baseline depressive symptoms show stronger associations with follow-up ED symptoms than reinforcement motivation

In our main analyses examining baseline predictors, depressive symptoms emerged as a significant predictor of follow-up ED symptoms (Table 4): A significant interaction between baseline depressive symptoms and BIS emerged in Model 1A.2, indicating that higher depressiveness predicted less T2 ED symptoms among participants with lower punishment sensitivity. Baseline depressive symptoms also directly predicted follow-up ED symptoms in one model (1A.2), but their inclusion did not improve overall model fit according to model comparison for any other outcome measure (Supplementary Table S7; Supplementary Methods). Likewise, when reversing the order of the nested model predictors by including only affective symptoms in the baseline model, subsequently adding baseline motivational measures did not improve model fit, while parsimony was reduced (Supplementary Table S8). Consistent with these findings, baseline BIS and motivational bias did not independently predict follow-up outcomes in the main regression analyses when accounting for affective symptoms. Across all final baseline models, VIFs remained acceptably low (≤1.6). This pattern of results was generally corroborated by cross-lagged panel models that simultaneously comprised all outcome measures, affective and motivational predictors: T1 depressiveness predicted T2 ED symptoms, while T1 punishment sensitivity or motivational bias did not (Supplementary Results, Supplementary Table S1, Supplementary Figures S1 and S2).

Longitudinal changes in affective symptoms outperform changes in reinforcement motivation in predicting outcomes

In contrast to the models using only baseline predictors, analyses incorporating longitudinal changes in BIS and motivational bias showed that adding longitudinal changes in affective symptoms as covariates consistently improved model performance and explained additional variance in treatment outcomes (Table 5; Supplementary Table S9). Conversely, adding change in reinforcement motivation after accounting for affective symptoms improved model fit in only one of six models (Supplementary Table S10). Notably, greater reduction in BDI-II scores was generally associated with lower ED symptoms, higher BMI-SDS and higher MROAS scores at follow-up (Table 5). It is noteworthy, however, that while

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Table 1. Descriptive statistics and group comparisons summary of statistical measures

Measure	AN-T1 Mean ± SD	AN-T2 Mean ± SD	recAN Mean ± SD	HC Mean ± SD	AN-T1 vs AN-T2	AN-T1 vs HC	AN-T2 vs HC	AN-T2 vs recAN	recAN vs HC
BMI (kg/m^2)	14.62 ± 1.34	18.71 ± 2.16	21.25 ± 2.03	21.25 ± 2.30	$t(191) = 24.96$, d = 1.80***	F = 1240.84, η^2_p = 0.66***	F = 142.56, η^2_p = 0.19***	F = 89.11, η^2_p = 0.21***	F = 6.70, η^2_p = 0.01*
BMI-SDS	−3.28 ± 1.08	−1.11 ± 1.31	−0.37 ± 0.68	−0.06 ± 0.67	$t(191) = 22.14$, d = 1.60***	F = 2043.84, η^2_p = 0.77***	F = 203.85, η^2_p = 0.25***	F = 91.67, η^2_p = 0.21***	F = 11.61, η^2_p = 0.02***
EDI–2 core	26.96 ± 6.75	24.79 ± 7.79	18.63 ± 6.71	14.27 ± 4.37	$t(186) = 3.19$, d = 0.23***	F = 727.76, η^2_p = 0.54***	F = 477.75, η^2_p = 0.43***	F = 46.76, η^2_p = 0.12***	F = 75.35, η^2_p = 0.11***
BDI-II	27.02 ± 11.59	19.01 ± 13.35	8.56 ± 8.91	4.13 ± 4.61	$t(182) = 6.76$, d = 0.50***	F = 1104.97, η^2_p = 0.64***	F = 419.77, η^2_p = 0.40***	F = 38.60, η^2_p = 0.10***	F = 79.35, η^2_p = 0.12***
STAI-trait	54.26 ± 11.53	50.69 ± 12.76	40.19 ± 11.43	33.91 ± 8.63	$t(183) = 3.35$, d = 0.29***	F = 482.70, η^2_p = 0.45***	F = 342.51, η^2_p = 0.36***	F = 41.85, η^2_p = 0.11***	F = 60.81, η^2_p = 0.10***
BIS total	23.19 ± 3.19	23.21 ± 3.64	22.53 ± 3.34	19.96 ± 3.21	$t(191) = 0.12$, d = 0.01 n.s.	F = 144.43, η^2_p = 0.19***	F = 135.62, η^2_p = 0.18***	F = 5.44, η^2_p = 0.02*	F = 56.73, η^2_p = 0.09***
BAS total	38.80 ± 4.77	39.05 ± 4.99	39.85 ± 4.85	40.28 ± 4.21	$t(191) = 0.77$, d = 0.06 n.s.	F = 11.72, η^2_p = 0.02***	F = 9.04, η^2_p = 0.01**	F = 6.61, η^2_p = 0.02*	F = 1.73, η^2_p = 0.003 n.s.
BIS/BAS motivational bias	−4.26 ± 8.13	−4.06 ± 8.86	−2.00 ± 8.57	3.22 ± 7.41	$t(191) = 0.38$, d = 0.03 n.s.	F = 127.33, η^2_p = 0.17***	F = 117.74, η^2_p = 0.16***	F = 10.24, η^2_p = 0.03***	F = 43.55, η^2_p = 0.07***
MROAS	n/a	7.22 ± 2.40	n/a	n/a	n/a	n/a	n/a	n/a	n/a

Note: AN-T1, anorexia nervosa at baseline; AN-T2, anorexia nervosa at follow-up; recAN, recovered anorexia nervosa; HC, healthy control; BDI-II, Beck Depression Inventory-II; BMI, body mass index; BMI-SDS, body mass index standard deviation score; EDI-2, Eating Disorder Inventory-2; STAI, State–Trait Anxiety Inventory; BIS, behavioral inhibition system; BAS, behavioral activation system; MROAS, Morgan–Russell outcome assessment schedule. Descriptive statistics for longitudinal AN sample at the focus of this report and the cross-sectional recAN and HC groups, along with inferential statistics comparing longitudinal differences in the AN group (fifth column) and cross-sectional between-group differences across variables of interest. Longitudinal comparisons in the AN group were conducted with paired *t*-tests (Cohen's d) and cross-sectional between-group comparisons using univariate ANCOVAs controlling for age (partial eta squared). Significance levels are indicated as follows: *$p < .05$, **$p < .01$, ***$p < .001$.

Table 2. Basic regression analyses predicting outcome at follow-up with baseline punishment sensitivity/baseline motivational bias

Model		R^2	ΔR^2	ΔR^2 95% CI	VIF	B	SE	β	t	p	p_{FDR}
1A.1	Outcome: T2 EDI–2 core										
	Full model: $F(3, 174) = 2.30$, $p = 0.079$	.04									
	Intercept					14.763	5.207		2.835	.005	.020
	Baseline EDI–2 core		0.02	[.00; .07]	1.1	0.182	0.090	0.158	2.010	.046	.092
	Baseline age		0.00	[.00; .01]	1.0	0.058	0.223	0.020	0.257	.797	.797
	Baseline BIS		0.00	[.00; .02]	1.1	0.173	0.198	0.070	0.874	.383	.511
1B.1	Outcome: T2 EDI–2 core										
	Full model: $F(3, 174) = 2.35$, $p = 0.073$	.04									
	Intercept					18.667	4.287		4.355	<.001	<.001
	Baseline EDI–2 core		0.02	[.00; .06]	1.1	0.176	0.091	0.153	1.928	.055	.111
	Baseline age		0.00	[.00; .01]	1.0	0.054	0.223	0.019	0.244	.808	.808
	Baseline motivational bias		0.01	[.00; .03]	1.2	−0.075	0.078	−0.078	−0.966	.336	.447
2A.1	Outcome: T2 BMI-SDS										
	Full model: $F(3, 177) = 24.09$, $p < .001$	.29									
	Intercept					2.861	0.764		3.747	<.001	<.001
	Baseline BMI-SDS		**0.06**	**[.00; .12]**	**1.1**	**0.318**	**0.081**	**0.259**	**3.918**	**<.001**	**<.001**
	Baseline age		**0.15**	**[.06; .24]**	**1.1**	**−0.206**	**0.034**	**−0.410**	**−6.090**	**<.001**	**<.001**
	Baseline BIS		0.00	[.00; .01]	1.0	0.017	0.027	0.041	0.634	.527	.527
2B.1	Outcome: T2 BMI-SDS										
	Full model: $F(3, 177) = 24.04$, $p < .001$	.29									
	Intercept					3.229	0.540		5.982	<.001	<.001
	Baseline BMI-SDS		**0.06**	**[.00; .12]**	**1.1**	**0.316**	**0.082**	**0.257**	**3.865**	**<.001**	**<.001**
	Baseline age		**0.15**	**[.06; .24]**	**1.1**	**−0.205**	**0.034**	**−0.410**	**−6.058**	**<.001**	**<.001**
	Baseline motivational bias		0.00	[.00; .01]	1.0	−0.006	0.011	−0.036	−0.556	.579	.579
3A.1	Outcome: MROAS										
	Full model: $F(2, 177) = 1.76$, $p = 0.176$	.02									
	Intercept					8.880	1.579		5.625	<.001	<.001
	Baseline age		0.02	[.00; .06]	1.0	−0.128	0.068	−0.142	−1.873	.063	.094
	Baseline BIS		0.00	[.00; .01]	1.0	0.018	0.058	0.023	0.303	.763	.763
3B.1	Outcome: MROAS										
	Full model: $F(2, 177) = 1.94$, $p = 0.147$	.02									
	Intercept					9.305	1.117		8.332	<.001	<.001
	Baseline age		0.02	[.00; .06]	1.0	−0.133	0.068	−0.148	−1.947	.053	.080
	Baseline motivational bias		0.00	[.00; .02]	1.0	−0.015	0.022	−0.051	−0.668	.505	.505

Note: BMI-SDS, body mass index standard deviation score; EDI-2, Eating Disorder Inventory-2; BIS, behavioral inhibition system; MROAS, Morgan–Russell outcome assessment schedule. Results of the regression analyses to test the predictive value of baseline motivational predictors (BIS, motivational bias) for several outcome measures, in alignment with Jonker et al. (2022) and Abber et al. (2024). Significant model predictors are marked in bold. A Δ denotes change scores across timepoints (baseline–follow-up). For an overview of model nomenclature, refer to Supplementary Table S3. For each model/predictor, the table columns report the following: R^2 = full model R^2; ΔR^2 = difference between the R^2 of the full model and the R^2 of the refit model without the corresponding term, this is equal to the squared semi-partial correlation coefficient and denotes the variance uniquely attributable to each predictor; ΔR^2 95% CI = 95% confidence interval for ΔR^2; VIF = variance inflation factor; B = unstandardized beta; SE = standard error; β = standardized beta; t = predictor t-value; p = uncorrected p-value; p_{FDR} = p-value, adjusted for false discovery rate via the Benjamini–Hochberg method.

change in motivational bias remained significantly linked to T2 EDI-2 even after controlling for ΔBDI-II, it was no longer clearly related to MROAS scores. Across all models, VIFs were acceptable (≤2.4).

Examination of ΔR^2 (the improvement in outcome prediction that is uniquely associated with each predictor in the regression model; Table 5) showed that changes in affective indices accounted for more unique variance in the outcome measures than punishment sensitivity/motivational bias. Further, particularly ΔBDI-II scores exhibited notably stronger standardized regression coefficients (β), suggesting that changes in affective symptoms contribute

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Table 3. Basic regression analyses predicting outcome at follow-up with change in punishment sensitivity/motivational bias between baseline and follow-up

Model		R^2	ΔR^2	ΔR^2 95% CI	VIF	B	SE	β	t	p	p_{FDR}
1C.1	Outcome: T2 EDI–2 core										
	Full model: $F(3, 169) = 3.06$, $p = 0.029$	.05									
	Intercept					17.157	4.146		4.138	<.001	<.001
	Baseline EDI–2 core		**0.04**	**[.00; .09]**	**1.0**	**0.227**	**0.087**	**0.197**	**2.616**	**.010**	**.019**
	Baseline age		0.00	[.00; .01]	1.0	0.083	0.220	0.028	0.376	.707	.707
	ΔBIS		0.01	[.00; .05]	1.0	−0.288	0.186	−0.116	−1.548	.123	.165
1D.1	Outcome: T2 EDI–2 core										
	Full model: $F(3, 169) = 11.29$, $p < .001$	.17									
	Intercept					14.755	3.916		3.768	<.001	<.001
	Baseline EDI–2 core		0.06	[.00; .12]	1.0	0.285	0.082	0.247	3.461	.001	.001
	Baseline age		0.00	[.00; .02]	1.0	0.143	0.206	0.049	0.691	.491	.491
	Δ motivational bias		**0.13**	**[.04; .22]**	**1.0**	**0.295**	**0.058**	**0.364**	**5.114**	**<.001**	**<.001**
2C.1	Outcome: T2 BMI-SDS										
	Full model: $F(3, 172) = 23.39$, $p < .001$	.29									
	Intercept					3.209	0.547		5.865	<.001	<.001
	Baseline BMI-SDS		**0.06**	**[.00; .12]**	**1.1**	**0.322**	**0.083**	**0.261**	**3.869**	**<.001**	**<.001**
	Baseline age		**0.15**	**[.06; .24]**	**1.1**	**−0.202**	**0.034**	**−0.402**	**−5.996**	**<.001**	**<.001**
	ΔBIS		0.00	[.00; .02]	1.0	0.021	0.027	0.051	0.793	.429	.429
2D.1	Outcome: T2 BMI-SDS										
	Full model: $F(3, 172) = 24.91$, $p < .001$	.30									
	Intercept					3.259	0.543		6.006	<.001	<.001
	Baseline BMI-SDS		**0.06**	**[.00; .12]**	**1.1**	**0.307**	**0.082**	**0.249**	**3.745**	**<.001**	**<.001**
	Baseline age		**0.16**	**[.07; .25]**	**1.1**	**−0.208**	**0.033**	**−0.415**	**−6.246**	**<.001**	**<.001**
	Δ motivational bias		0.02	[.00; .05]	1.0	−0.018	0.009	−0.126	−1.966	.051	.051
3C.1	Outcome: MROAS										
	Full model: $F(2, 172) = 2.53$, $p = 0.082$	.03									
	Intercept					9.057	1.119		8.091	<.001	<.001
	Baseline age		0.02	[.00; .06]	1.0	−0.117	0.067	−0.130	−1.733	.085	.127
	ΔBIS		0.01	[.00; .04]	1.0	0.077	0.056	0.103	1.375	.171	.171
3D.1	Outcome: MROAS										
	Full model: $F(2, 172) = 7.40$, $p < .001$	.08									
	Intercept					9.309	1.091		8.536	<.001	<.001
	Baseline age		**0.02**	**[.00; .06]**	**1.0**	**−0.134**	**0.066**	**−0.149**	**−2.033**	**.044**	**.044**
	Δ motivational bias		**0.06**	**[.00; .13]**	**1.0**	**−0.062**	**0.018**	**−0.248**	**−3.383**	**.001**	**.001**

Note: BMI-SDS, body mass index standard deviation score; EDI-2, Eating Disorder Inventory-2; BIS, behavioral inhibition system; MROAS, Morgan–Russell outcome assessment schedule. Results of the regression analyses to test the predictive value of longitudinal change in motivational predictors (ΔBIS, Δ motivational bias) for several outcome measures, in alignment with Jonker et al. (2022) and Abber et al. (2024). Significant model predictors are marked in bold. A Δ denotes change scores across timepoints (baseline–follow-up). For an overview of model nomenclature, refer to Supplementary Table S3. For each model/predictor, the table columns report the following: R^2 = full model R^2; ΔR^2 = difference between the R^2 of the full model and the R^2 of the refit model without the corresponding term, this is equal to the squared semi-partial correlation coefficient and denotes the variance uniquely attributable to each predictor; ΔR^2 95% CI = 95% confidence interval for ΔR^2; VIF = variance inflation factor; B = unstandardized beta; SE = standard error; β = standardized beta; t = predictor t-value; p = uncorrected p-value; p_{FDR} = p-value, adjusted for false discovery rate via the Benjamini–Hochberg method.

more strongly to the outcome than changes in reinforcement motivation. Together, these results indicate that affective symptom improvement plays a central role in overall recovery, whereas changes in reinforcement motivation appear to contribute more narrowly, primarily to follow-up ED symptoms.

Discussion

Recent studies have highlighted the prognostic relevance of reinforcement motivation, specifically punishment sensitivity and its relation to reward sensitivity (referred to as "motivational bias"), for clinical outcomes in individuals with AN (Abber et al., 2024;

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Table 4. Main regression analyses predicting ED symptoms at follow-up with baseline punishment sensitivity/motivational bias and affective symptoms, while allowing for interactions between depressive and motivational predictors

Model		R^2	ΔR^2	ΔR^2 95% CI	VIF	B	SE	β	t	p	p_{FDR}
1A.2 incl. IA	Outcome: T2 EDI–2 core										
	Full model: $F(5, 172) = 4.05$, $p = .002$	.11									
	Intercept					14.931	4.375		3.413	.001	.005
	Baseline EDI–2 core		**.05**	**[.00; .11]**	**1.4**	**0.315**	**0.100**	**0.273**	**3.160**	**.002**	**.006**
	Baseline age		.00	[.00; .00]	1.1	0.031	0.218	0.011	0.143	.886	.886
	Baseline BDI-II		**.02**	**[.00; .07]**	**1.6**	**−0.130**	**0.060**	**−0.194**	**−2.155**	**.033**	**.049**
	Baseline BIS		.02	[.00; .06]	1.3	0.388	0.206	0.156	1.888	.061	.073
	BDI-II × BIS		**.05**	**[.00; .10]**	**1.1**	**0.044**	**0.015**	**0.219**	**2.960**	**.004**	**.007**
1B.2 incl. IA	Outcome: T2 EDI–2 core										
	Full model: $F(5, 172) = 3.08$, $p = .011$	.08									
	Intercept					15.249	4.512		3.380	.001	.005
	Baseline EDI–2 core		**.05**	**[.05; .11]**	**1.4**	**0.298**	**0.101**	**0.258**	**2.942**	**.004**	**.011**
	Baseline age		.00	[.00; .01]	1.1	0.053	0.222	0.018	0.238	.812	.812
	Baseline BDI-II		.02	[.00; .07]	1.6	−0.134	0.062	−0.199	−2.151	.033	.066
	Baseline motivational bias		.02	[.00; .05]	1.4	−0.146	0.082	−0.151	−1.774	.078	.093
	BDI-II × motivational bias		.02	[.00; .06]	1.1	−0.011	0.006	−0.143	−1.902	.059	.088

Note: IA, interactions; BMI-SDS, body mass index standard deviation score; EDI-2, Eating Disorder Inventory-2; BIS, behavioral inhibition system; BDI-II, Beck Depression Inventory; MROAS, Morgan–Russell outcome assessment schedule. Results of the regression analyses to test the predictive value of baseline motivational predictors (BIS, motivational bias) for follow-up ED symptoms, extending previous work by including baseline affective symptoms and allowing for interactions between depressiveness and motivational characteristics. This table is confined to those models in which adding depressiveness and/or trait anxiety improved model fit (see Methods section and the Supplementary Material for our model selection procedure). Significant model predictors are marked in bold. BDI-II, BIS and motivational bias were mean-centered to reduce multicollinearity. A Δ denotes change scores across timepoints (baseline–follow-up). For an overview of model nomenclature, refer to Supplementary Table S3. For each model/predictor, the table columns report the following: R^2 = full model R^2; ΔR^2 = difference between the R^2 of the full model and the R^2 of the refit model without the corresponding term, this is equal to the squared semi-partial correlation coefficient and denotes the variance uniquely attributable to each predictor; ΔR^2 95% CI = 95% confidence interval for ΔR^2; VIF = variance inflation factor; B = unstandardized beta; SE = standard error; β = standardized beta; t = predictor t-value; p = uncorrected p-value; p_{FDR} = p-value, adjusted for false discovery rate via the Benjamini–Hochberg method.

Jonker et al., 2022, 2025). Building on this work, we tested whether (1) reinforcement motivation can predict outcomes in individuals with AN, even after controlling for co-occurring affective symptoms, and (2) affective symptoms predict outcomes better than reinforcement motivation alone. Using a longitudinal design in a comparatively large sample of underweight adolescent girls and young women with AN, complemented by cross-sectional comparisons with long-term recovered individuals (recAN) and healthy controls (HC), we found that change in motivational bias following treatment was associated with ED symptom severity one year after beginning treatment, extending prior findings (Abber et al., 2024). However, at baseline, while neither punishment sensitivity nor motivational bias were reliably associated with any outcome, higher depressive symptoms directly predicted greater reductions in ED symptoms at follow-up (and in exploratory interaction with punishment sensitivity), which was further corroborated by the cross-lagged panel analyses. Most importantly, longitudinal shifts in depressive symptoms emerged as the most robust predictor of improvements across all outcome domains, outpacing the predictive relevance of reinforcement-related variables alone. These results, while observational, underscore the central role of affective symptom improvement in AN recovery, as supported by different lines of evidence (Franko et al., 2018; Haynos et al., 2024; Oldershaw, Startup, & Lavender, 2019).

Consistent with prior work, individuals with AN (or a history thereof) exhibited elevated punishment sensitivity (Glashouwer et al., 2014; Harrison et al., 2010; 2016; Harrison, Treasure, & Smillie, 2011; Haynos et al., 2020; Jappe et al., 2011; Jonker et al., 2022) and a stronger motivational bias toward punishment relative to reward sensitivity in comparison to HC (Abber et al., 2024). Generally aligning with both studies that inspired ours (Abber et al., 2024; Jonker et al., 2022), longitudinal shifts in motivational bias (toward higher punishment relative to reward sensitivity) were associated with higher ED symptoms and less favorable global outcomes (MROAS scores). Crucially, however, regression models incorporating changes in affective symptoms showed superior fit compared with models including only reinforcement motivation measures. Reductions in depressive symptoms emerged as the strongest and most consistent predictors of improvements across all outcome domains. Together, while confirming a modest relationship between reinforcement motivation and later ED symptoms in AN (Abber et al., 2024; Jonker et al., 2022, 2025), our findings imply that affective improvements play a central role in overall recovery, highlighting emotional processes as an important therapeutic target (Ejdemyr et al., 2025;Haynos et al., 2024; Tchanturia, Doris, Mountford, & Fleming, 2015; Wildes & Marcus, 2011).

Notably, in contrast to the observed associations between outcomes and longitudinal motivational and affective changes, neither baseline punishment sensitivity, baseline motivational bias, nor changes in punishment sensitivity directly predicted outcomes in our sample. This pattern diverges from Jonker et al. (2022), who observed in a comparatively smaller adolescent cohort ($n = 62$) that higher baseline punishment sensitivity, assessed using both the BIS and the sensitivity to punishment subscale of the SPSRQ, was

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Table 5. Main regression analyses predicting outcome at follow-up with change in punishment sensitivity and motivational bias between baseline and follow-up, while allowing for interactions between depressive and motivational predictors

Model		R^2	ΔR^2	ΔR^2 95% CI	VIF	B	SE	β	t	p	p_{FDR}
1C.2 incl. IA	Outcome: T2 EDI–2 core										
	Full model: $F(5, 167) = 8.27$, $p <$ .001	.20									
	Intercept					16.543	3.868		4.277	<.001	<.001
	Baseline EDI–2 core		**.07**	**[.00; .14]**	**1.1**	**0.309**	**0.082**	**0.268**	**3.755**	**<.001**	**<.001**
	Baseline age		.00	[.00; .00]	1.0	−0.005	0.204	−0.002	−0.023	.982	.982
	ΔBDI-II		**.14**	**[.05; .24]**	**1.2**	**−0.247**	**0.045**	**−0.410**	**−5.443**	**<.001**	**<.001**
	ΔBIS		.00	[.00; .01]	1.1	0.063	0.184	0.025	0.342	.733	.879
	ΔBDI-II × ΔBIS		.00	[.00; .02]	1.0	−0.009	0.012	−0.056	−0.800	.425	.638
1D.2 incl. IA	Outcome: T2 EDI–2 core										
	Full model: $F(5, 167) = 11.96$, $p <$ .001	.26									
	Intercept					13.895	3.735		3.720	<.001	<.001
	Baseline EDI–2 core		**.09**	**[.01; .16]**	**1.1**	**0.351**	**0.079**	**0.305**	**4.436**	**<.001**	**<.001**
	Baseline age		.00	[.00; .01]	1.0	0.065	0.196	0.022	0.333	.740	.740
	ΔBDI-II		**.09**	**[.02; .17]**	**1.2**	**−0.201**	**0.043**	**−0.334**	**−4.636**	**<.001**	**<.001**
	Δ motivational bias		**.07**	**[.00; .13]**	**1.2**	**0.227**	**0.058**	**0.280**	**3.898**	**<.001**	**<.001**
	ΔBDI-II × Δ motivational bias		.01	[.00; .03]	1.1	−0.005	0.004	−0.085	−1.242	.216	.259
2C.2 incl. IA	Outcome: T2 BMI-SDS										
	Full model: $F(5, 170) = 16.58$, $p <$ .001	.33									
	Intercept					3.080	0.539		5.713	<.001	<.001
	Baseline BMI-SDS		.06	**[.00; .11]**	**1.1**	**0.311**	**0.082**	**0.252**	**3.802**	**<.001**	**<.001**
	Baseline age		**.14**	**[.06; .23]**	**1.1**	**−0.197**	**0.033**	**−0.394**	**−5.990**	**<.001**	**<.001**
	ΔBDI-II		**.03**	**[.00; .08]**	**1.1**	**0.020**	**0.007**	**0.198**	**2.966**	**.003**	**.005**
	ΔBIS		.00	[.00; .01]	1.2	−0.009	0.028	−0.022	−0.325	.746	.746
	ΔBDI-II × ΔBIS		.00	[.00; .02]	1.0	0.002	0.002	0.055	0.860	.391	.469
2D.2 incl. IA	Outcome: T2 BMI-SDS										
	Full model: $F(5, 170) = 16.67$, $p <$ .001	.33									
	Intercept					3.179		0.539	5.895	<.001	<.001
	Baseline BMI-SDS		**.06**	**[.00; .12]**	**1.1**	**0.314**	**0.081**	**0.255**	**3.884**	**<.001**	**<.001**
	Baseline age		**.15**	**[.06; .23]**	**1.1**	**−0.201**	**0.033**	**−0.401**	**−6.086**	**<.001**	**<.001**
	ΔBDI-II		**.03**	**[.00; .06]**	**1.1**	**0.018**	**0.007**	**0.173**	**2.573**	**.011**	**.016**
	Δ motivational bias		.00	[.00; .02]	1.2	−0.010	−0.071	0.009	−1.045	.297	.357
	ΔBDI-II × Δ motivational bias		.00	[.00; .00]	1.1	<.0.001	0.001	0.013	0.197	.844	.844
3C.3 incl. IA	Outcome: MROAS										
	Full model: $F(5, 169) = 6.96$, $p <$ .001	.17									
	Intercept					8.561	1.053		8.129	<.001	<.001
	Baseline age		.01	[.00; .04]	1.0	−0.088	0.063	−0.098	−1.394	.165	.248
	ΔBIS		.00	[.00; .02]	1.3	−0.057	0.059	−0.077	−0.975	.331	.397
	ΔBDI-II		.02	[.00; .06]	2.1	0.039	0.211	0.019	2.073	.040	.079
	ΔSTAI-T		.02	[.00; .06]	2.4	0.569	0.259	0.238	2.200	.029	.079
	ΔBIS × ΔBDI-II		.00	[.00; .01]	1.0	0.002	0.004	0.034	0.484	.629	.629
3D.2 incl. IA	Outcome: MROAS										
	Full model: $F(5, 169) = 8.41$, $p <$ .001	.17									
	Intercept					8.957	1.052		8.510	<.001	<.001

(*Continued*)

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Table 5. (*Continued*)

Model		R^2	ΔR^2	ΔR^2 95% CI	VIF	B	SE	β	t	p	p_{FDR}
	Baseline age		.02	[.00; .05]	1.0	−0.111	0.063	−0.124	−1.761	.080	.100
	Δ motivational bias		.02	[.00; .05]	1.2	−0.036	0.019	−0.143	−1.901	.059	.098
	ΔBDI-II		**.08**	**[.01; .16]**	**1.1**	**0.057**	**0.014**	**0.309**	**4.123**	**<.001**	**<.001**
	ΔBDI-II × Δ motivational bias		.00	[.00; .00]	1.1	<.001	0.001	−0.016	−0.228	.820	.820

Note: IA, interactions; BMI-SDS, body mass index standard deviation score; EDI-2, Eating Disorder Inventory-2; BIS, behavioral inhibition system; BDI-II, Beck Depression Inventory; MROAS, Morgan–Russell outcome assessment schedule; STAI-T, State–Trait Anxiety Inventory–Trait. Results of the regression analyses to test the predictive value of longitudinal change in motivational predictors (BIS, motivational bias) for several outcome measures, extending previous work by including affective symptoms and allowing for interactions between depressiveness and motivational characteristics. This table is confined to those models in which adding depressiveness and/or trait anxiety improved model fit (see Methods section and the Supplementary Material for our model selection procedure). Significant model predictors are marked in bold. BDI-II, BIS and motivational bias were mean-centered to reduce multicollinearity. A Δ denotes change scores across timepoints (baseline–follow-up). For an overview of model nomenclature, refer to Supplementary Table S3. For each model/predictor, the table columns report the following: R^2 = full model R^2; ΔR^2 = difference between the R^2 of the full model and the R^2 of the refit model without the corresponding term, this is equal to the squared semi-partial correlation coefficient and denotes the variance uniquely attributable to each predictor; ΔR^2 95% CI = 95% confidence interval for ΔR^2; VIF = variance inflation factor; B = unstandardized beta; SE = standard error; β = standardized beta; t = predictor t-value; p = uncorrected p-value; p_{FDR} = p-value, adjusted for false discovery rate via the Benjamini–Hochberg method.

associated with poorer ED symptom improvement, as measured by both the semi-structured Eating Disorders Examination (EDE; Cooper & Fairburn, 1987) and the EDE-Questionnaire (EDE-Q; Fairburn & Beglin, 1994) at a 1-year follow-up. Conversely, Abber et al. (2024) found in a larger mixed adolescent/adult cohort (n = 262) that baseline motivational bias scores, also assessed via BIS and SPSRQ, did not predict outcomes (EDE-Q symptoms or BMI) over a ~ 100-day follow-up. In our predominately adolescent sample, baseline BIS and motivational bias scores were not significant predictors of any one-year outcome, regardless of whether affective symptoms were included as covariates (which was the case in 2 out of 6 models), with the exception of one single interaction between BIS and BDI-II in Model 1A.2. Specifically, the interaction indicated that higher baseline depressiveness predicted lower follow-up ED symptom severity among low punishment sensitive individuals than among those with high punishment sensitivity. One possible interpretation is that depressive symptom severity may index a higher potential for improvement in some patients, particularly when avoidance motivation is less pronounced. However, given that this effect was not observed across other models, it should be interpreted cautiously and may simply reflect shared variance between depressive symptoms and AN severity rather than a distinct moderating mechanism.

The otherwise limited prognostic utility of these baseline reinforcement measures may reflect sample characteristics, illness stage, follow-up duration or developmental status, with prior work also indicating moderating effects of clinical characteristics (e.g. depression) on treatment outcomes in adolescent AN (Le Grange et al., 2022). Baseline indices reflect only one-time assessments, whereas longitudinal changes in affective and motivational measures capture dynamic adaptation during treatment. Indeed, in our longitudinal models incorporating changes in affective symptoms, global model fit improved significantly, and reductions in depressive symptoms consistently emerged as the strongest and most robust predictors of improvement across all outcomes, whereas changes in reinforcement motivation showed more limited associations.

Our findings indicate that depressive symptom reduction is associated with a more balanced sensitivity to punishment and reward, suggesting a link between affective improvement and recovery processes in AN. This interpretation accords with theoretical models emphasizing the role of affective and motivational processes in the maintenance of AN. For example, the transdiagnostic cognitive-behavioral model of ED (Fairburn, Cooper, & Shafran, 2003; Fairburn et al., 2009) proposes that low mood, mood intolerance, and anxiety represent key maintaining mechanisms and that targeting these processes is critical for recovery. Extending this framework, interventions aimed at enhancing emotion-regulation capacity (Sala, Heard, & Black, 2016), increasing positive affect (Haynos et al., 2024), targeting specific facets of anhedonia like anticipatory pleasure and motivational engagement (Ma et al., 2026) and strengthening motivation for change (Denison-Day, Appleton, Newell, & Muir, 2018; Ziser et al., 2021) have been shown to improve emotional functioning and engagement in adaptive, goal-directed behavior.

Strengths of this study include a comparatively large and clinically homogeneous sample of predominantly adolescent patients with AN observed longitudinally, the use of the MROAS as a multidimensional outcome measure, and the simultaneous inclusion of cross-sectional comparisons with recAN and HC. Attrition analyses further indicated that participants who discontinued or had not yet been reassessed at follow-up did not differ from the main longitudinal sample on any variable investigated, minimizing potential bias. Limitations include the reliance on single self-report measures of reinforcement motivation. Also, psychiatric comorbidities were not assessed using standardized structured diagnostic interviews across the full sample and therefore could not be systematically included as covariates in the present analyses, although evidence suggests that remission may vary as a function of comorbidity (Le Grange et al., 2021). As highlighted by a recent study of Jonker et al. (2025), distinguishing between punishment *responsivity* and *avoidance motivation* may be crucial, as only the former appears to predict symptom persistence. Additionally, inpatient treatment duration varied naturally, and subsequent treatment exposure following discharge was naturalistic, potentially introducing variability in outcomes. Furthermore, the predominantly female, adolescent sample may limit generalizability to other age groups, ambulatory care settings, or male patients, while variability in illness duration may also have contributed to heterogeneity in reinforcement-related processes. Although our analyses included multiple covariates, the observational design and use of longitudinal change associations prevent causal inferences regarding the relationship between affective changes, shifts in reinforcement motivation, and recovery. To partially address this, supplementary cross-lagged panel models were evaluated to further inspect directional relationships, though experimental or intervention studies remain necessary to establish true causality. Relatedly, we acknowledge that the use of raw change scores in these longitudinal

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analyses is subject to notable methodological limitations, including measurement error and regression to the mean, which may affect the precision of our estimates.

In sum, the present study revealed that elevated depressive symptoms at admission to treatment for AN and longitudinal reductions in affective symptoms were consistently associated with improvements across core ED symptoms, BMI and multidimensional recovery, emphasizing the central role of affective processes in AN recovery. While changes in reinforcement motivation were observed alongside symptomatic improvement, their impact on clinical outcome was narrower. These findings support the relevance of targeting affective disturbance as a core component of intervention and the integration of emotional and motivational processes within treatment frameworks to optimize outcomes (Haynos et al., 2024; Tchanturia, Doris, Mountford, & Fleming, 2015; Wildes & Marcus, 2011).

Supplementary material. The supplementary material for this article can be found at http://doi.org/10.1017/S0033291726105674.

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Ethical standard. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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