Research report

Heightened sensitivity to punishment and reward in anorexia nervosa

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Objective: The aim of this study was to investigate reinforcement sensitivity in anorexia nervosa (AN). It was tested whether self-reported punishment (PS) and reward sensitivity (RS) differed between adolescents with AN and healthy controls, and/or between AN-subtypes. In addition, the predictive validity of PS and RS was examined for AN symptoms one year later. Method: In total, 165 female adolescents admitted for treatment of AN or eating disorder not otherwise specified resembling AN and 72 controls participated in the study. Participants completed measurements for eating disorder severity and the Sensitivity to Punishment/Sensitivity to Reward Questionnaire (SPSRQ). Percentage of underweight and severity of AN symptoms were measured again after one year in individuals with AN. Results: Individuals with AN scored higher on PS and RS than controls. In addition, the AN purging type showed higher PS than the AN restrictive type, whereas there were no differences in RS between AN-subtypes. Regression analyses indicated that PS and RS were independently associated with the degree of eating disorder symptoms, whereas only PS was related to percentage underweight. Yet, neither RS nor PS were related to percentage age of underweight and AN symptoms after one year. Discussion: Although the present study clearly demonstrated that heightened punishment and reward sensitivity are both linked to AN, there was no evidence that these characteristics are also involved in the course of AN symptoms.

Introduction

Anorexia nervosa (AN) is a severe psychiatric disorder with the highest mortality rate among all psychiatric disorders (Sullivan, 1995). Unfortunately, the underlying processes that cause and maintain AN are still largely unknown. One of the puzzling features of AN is that patients succeed in restricting their food intake, while they actually are in a state of starvation. By nature, food has a high reward value, even more for people that have been deprived of food (e.g., Stroebe, Papes, & Aarts, 2008). So how do individuals with AN overcome their biological drive to eat? One explanation might be provided by the sensitivity of AN patients to environmental cues signaling reward or punishment. According to the Reinforcement Sensitivity Theory (e.g., Gray, 1987) individuals’ behaviors are guided by different brain systems. The behavioral inhibition system reacts to aversive/punishing stimuli and is thought to lead to avoidance behaviors. The behavioral activation system is sensitive to rewarding stimuli and assumed to guide approach behaviors. Heightened punishment sensitivity might be a risk factor for developing AN, because it could ‘help’ individuals to abstain from eating. For individuals who are high in punishment sensitivity and who base their self-esteem largely, or even exclusively, on their body shape and weight (Fairburn, 2008), the idea of gaining weight can become very threatening. As a consequence, they could start to avoid expected punishment (i.e. weight gain) through avoidance behaviors such as restriction of food intake, or excessive exercise, increasing the risk for developing AN.

Prior findings suggest that patients with AN indeed show heightened punishment sensitivity supporting the hypothesis that heightened punishment sensitivity is a risk factor for developing AN (Beck, Smits, Claes, Vandereycken, & Bijnertieber, 2009; Claes, Nederkoorn, Vandereycken, Guerrieri, & Vertommen, 2006; Harrison, O’Brien, Lopez, & Treasure, 2010; Jappe et al., 2011). However, up to now, studies mainly relied on adult samples. Yet, the impact/relevance of reward and punishment sensitivity may be different for younger, adolescent samples (e.g., adolescents generally seem to be more sensitive to appetitive stimuli: Spear & Varlinskaya, 2010; Van Leijenhorst et al., 2010). In addition, and most critical for the present context, the first onset of AN often takes place during early adolescence and the heightened punishment sensitivity...
found in adults with AN might be the consequence of protracted AN rather than a premorbid risk factor. Therefore, the first goal of the present study is to examine whether punishment sensitivity is already heightened in young adolescents with AN. This would further support the idea that heightened punishment sensitivity might indeed be a risk factor for developing AN.

In addition, an alternative explanation for the striking ability of AN patients to resist (or overcome) their drive to eat could be that they differ from healthy controls in their sensitivity to reward. Perhaps lowered reward sensitivity leads to a lower approach motivation to food cues, which increases the risk of developing AN. Results in this respect are still mixed. Some studies showed that individuals with AN of the restrictive type (AN-R) indeed displayed lower levels of reward sensitivity than controls, while individuals with AN of the purging/binging type (AN-P) showed higher levels of reward sensitivity than individuals with AN-R and/or controls (Beck et al., 2009; Claes et al., 2006; Harrison et al., 2010). The latter could help explain why individuals with AN-P sometimes show episodes of binge eating. However, recently, Jappe and colleagues (2011) showed the opposite, namely that both types of women with AN displayed higher sensitivity to reward than healthy controls. They did not find differences in reward sensitivity between AN-R and AN-P. Consequently, the second goal of the present study is to examine reward sensitivity in a large group of AN patients, since in prior studies especially subgroups of AN-P were usually small. Because prior findings with respect to reward sensitivity in AN were mixed, we do not have a clear hypothesis.

Although prior studies suggest that reward and punishment sensitivity generally are independent constructs (see e.g., Franken & Muris, 2006; O’Connor, Colder, & Hawk, 2004), it is still unknown whether these constructs are also independently involved in AN, or whether they should be seen as overlapping risk factors. Furthermore, we explore whether perhaps the relationship between reward sensitivity and the onset of AN is moderated by punishment sensitivity, or vice versa (see e.g., Corr, 2002). Consequently, the third goal of this study is to test whether punishment and reward sensitivity as well as their interaction are independently related to severity of eating disorder symptoms. Our hypothesis is that both factors are independently linked to severity of AN.

Finally, reward and punishment sensitivity might not only be risk factors for the onset of AN, but perhaps they also play a role in the persistence of AN over time. Therefore, the last goal is to examine whether in individuals with AN, punishment sensitivity, reward sensitivity, and/or their interaction predict AN symptoms later in time. More clarity with respect to the potential role of punishment and reward sensitivity as risk factors for the development and maintenance of AN-R and AN-P might be crucial for a better understanding of AN and its underlying processes.

Method

Participants

The clinical group existed of 165 female adolescents who met the criteria of AN or an eating disorder not otherwise specified resembling AN1: 48 of the purging type (AN-P) and 117 of the restrictive type (AN-R). Participants with AN were recruited through the Department of Eating Disorders of Accare, a facility for child and adolescent psychiatry in the Netherlands. In addition, we included 72 female control participants who were recruited via a local high school (Gomarus College in Groningen, the Netherlands). Control participants did not differ significantly from AN participants on age and educational level (see Table 1) and shared the same ethnic background as the AN group (Caucasian). Control participants were screened on eating disorder symptoms with the Eating Disorder Examination–Questionnaire (EDE-Q (Fairburn & Bégin, 1994)). The measurements were part of routine outcome monitoring assessment which was approved beforehand by the local authority of Accare, child- and adolescent psychiatry. Participants of the control group and their parents actively gave informed consent and the study protocol was approved by the Ethical Committee of Psychology of the University of Groningen.

Clinical and measures

Clinical group. Within two weeks after registration participants of the clinical group were diagnosed by health care professionals of Accare using the Dutch child version of the Eating Disorder Examination (ChEDE (Bryant-Waugh, Cooper, Taylor, & Lask, 1996; Decaluwé & Beraat, 1999)). In addition, participants filled in the Dutch version of the EDE-Q (Fairburn & Bégin, 1994). The EDE-Q is a self-report questionnaire which consists of 41 items and reflects the format of the EDE interview including the four subscales and the global severity score. Furthermore, participants completed the Dutch version of the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ (Franken & Muris, 2006; Torrubia, Avila, Molto, & Caceres, 2001)). The SPSRQ is a self-report instrument and contains 48 items in a yes/no format. The questionnaire provides scores for individual sensitivity to punishment (SP), and sensitivity to reward (SR) (range: 0–24).2 The SPSRQ has already been widely used and is designed to index sensitivity to specific cues that signal reward and punishment, in line with Gray’s theory (Torrubia et al., 2001). The wording of some items was slightly adapted to make them appropriate and understandable for the adolescent age group. Psychometric evaluation showed that both subscales of the SPSRQ present satisfactory internal consistency and test–retest reliability as well as convergent and discriminant validity (Torrubia et al., 2001). Furthermore, the validity of the Dutch version of the SPSRQ has been shown to be comparable with the validity of the original version in ED patients (Beck et al., 2009) and the SPSRQ seems to be suitable for use among adolescents (e.g., Matton, Goossens, Braet, & Vervaet, 2013). Finally, weight and height data were collected as well as demographics. Percentage underweight was calculated by dividing the current weight through the target weight appropriate for the length and age of each participant multiplied by one hundred. This number was deducted from 100. The target weight was determined using growth curves (TNO).

Control group. The procedure for participants of the control group was similar to that of the clinical group, with exception of the ChEDE-interview, which was not administered in the control group. Control participants were assessed once.

Study design

The clinical group was measured twice: at baseline the ChEDE, the EDE-Q, the SPSRQ and weight and height data were collected. Percentage of underweight and EDE-Q were measured again after

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1 Participants fulfilled all criteria of AN; 20 participants fulfilled all criteria for AN except criterion A ‘underweight’ which was between 0–15%; 25 participants fulfilled all criteria for AN except criterion B ‘fear of gaining weight’; 2 participants fulfilled all criteria for AN except criterion C ‘disturbance in the way in which one’s body weight or shape is experienced, undue influence of body shape on self-evaluation, or denial of the seriousness of the current low body weight’; 11 participants fulfilled all criteria for AN except criterion D ‘amenorrhea’; 18 participants fulfilled all criteria for AN except criterion A and D; 5 participants fulfilled all criteria for AN except criterion B and D.

2 We also calculated the scales as proposed by O’Connor, Colder and Hawk (2004). However, since the outcomes of the statistical analyses were generally similar to the outcomes of the original calculations, we decided only to report the analyses with the original calculations.
one year. In between baseline and follow-up patients received treatment as usual, which meant that patients received inpatient and/or outpatient treatment which included one or a combination of the following therapies: individual treatment, group treatment, family treatment, cognitive behavioral therapy, dietary advice or medication. For control participants data was collected once for the EDE-Q, the SPSRQ and weight and height.

**Statistical analyses**

ANOVA analyses and post hoc Hochbergs GT2 tests were used to compare SPSRQ scores between the AN groups and the control group. Pearson correlation analyses were conducted to explore the relationships at baseline between the SPSRQ subscales and the percentage underweight and EDE-Q. Linear regression analyses were conducted to test whether punishment and reward sensitivity were independently related to eating disorder symptom severity and percentage underweight at baseline and to test predictive validity over time.

**Results**

**Missing data and drop-out**

Originally, 76 control participants were included, but four control participants scored above the cut-off of 2.3 points on the global severity score on the EDE-Q and were therefore excluded (cf. Mond, Hay, Rodgers, Owen, & Beumont, 2004). This resulted in a final group of 72 controls. Of the 165 participants in the AN group at baseline, 40 individuals had missing data on the SPSRQ and 18 individuals had missing data on the EDE-Q. Of the 72 controls at baseline, 3 individuals had missing data on percentage underweight, 2 individuals had missing data on the SPSRQ, and 1 individual had missing data on the EDE-Q.

Unfortunately, after one year there was a substantial amount of drop-out (41%) in the clinical group. There were various reasons for dropping out: living too far away from the treatment center, doing well and not wanting to be reminded, doing poorly and not feeling able to participate, just not wanting to participate. Furthermore, 15 participants were not measured because they were referred to another treatment center or the follow-up measurement was not conducted yet. Of the 165 participants with AN measured at baseline 88 participated in the 1-year follow-up (t2). Furthermore, 5 individuals had additional missing data for the percentage of underweight at t2 and 3 for the EDE-Q at t2. To test whether patients who remained in the study differed from patients who dropped out, we compared both groups at baseline by means of independent sample t-tests. However, drop-outs and completers did not differ on age, educational level, percentage underweight, scores on the SPSRQ, type of AN, and scores on the ChEDE or EDE-Q.

Missing data were estimated using multiple imputation which was developed as one of the state-of-the-art and preferred methods for dealing with missing data (Schafer & Graham, 2002). Missing data was imputed 40 times with predictive mean matching using IBM SPSS Statistics 20, based on all predictors that were included in the model as well as other variables outside the present study (e.g., depressive symptoms, body image) to impute as accurately as possible. We report the pooled results.

**Descriptives**

In this study internal consistencies of both subscales of the SPSRQ were good (PS: $\alpha = 0.90$; RS: $\alpha = 0.77$). Group characteristics on all variables are reported in Table 1. Furthermore, Pearson’s correlations were calculated between the SPSRQ subscales, the EDE-Q total score and percentage underweight at baseline over all participants ($N = 237$). As expected, the SP-subscale and the SR-subscale did not correlate ($r = 0.10, p = .15$). Percentage underweight and EDE-Q total score were moderately correlated ($r = 0.20, p < .001$). The AN-R group showed significantly lower symptom levels at t2 than at baseline (Table 1).

**Group differences on punishment and reward sensitivity**

A three-group (AN-R, AN-P, controls) ANOVA on the SP-subscale showed a significant main effect for group ($F(2,234) = 39.91, p < .001$, partial $\eta^2 = 0.26$). As expected, both AN-groups showed higher levels of punishment sensitivity than healthy controls (AN-R vs. controls: mean difference = 5.23, $p < .001$, 95% CI [3.35, 7.10]; AN-P vs. controls: mean difference = 8.19 $p < .001$, 95% CI [5.86, 10.52]). Furthermore, the AN-P group showed higher punishment sensitivity than the AN-R group (mean difference = 2.96 $p = .005$, 95% CI [0.82, 5.10]).

A three-group (AN-R, AN-P, controls) ANOVA on the SR-subscale showed a significant main effect for group ($F(2,234) = 4.93, p = .01$, partial $\eta^2 = 0.04$). Both AN-groups showed higher levels of reward

Table 1

<table>
<thead>
<tr>
<th>(A) Controls</th>
<th>(B) AN</th>
<th>AN-R</th>
<th>AN-P</th>
<th>(C) AN T2</th>
<th>t-Test</th>
<th>Paired t-test</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n = 72)</td>
<td>(n = 165)</td>
<td>(n = 117)</td>
<td>(n = 48)</td>
<td>(n = 150)</td>
<td>A vs. B</td>
<td>B vs. C</td>
</tr>
<tr>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>p</td>
<td>p</td>
</tr>
<tr>
<td>Age</td>
<td>14.84 (1.71)</td>
<td>15.02 (1.71)</td>
<td>14.96 (1.78)</td>
<td>15.19 (1.54)</td>
<td>–</td>
<td>.52</td>
</tr>
<tr>
<td>Education$^a$</td>
<td>34:66</td>
<td>43:57</td>
<td>43:57</td>
<td>43:57</td>
<td>–</td>
<td>.47$^a$</td>
</tr>
<tr>
<td>Underweight (%)</td>
<td>-3.19 (12.51)</td>
<td>20.26 (8.47)</td>
<td>21.58 (7.57)</td>
<td>16.85 (9.60)</td>
<td>5.46 (10.16)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>EDE-Q – Restraint</td>
<td>0.82 (0.85)</td>
<td>3.34 (1.74)</td>
<td>2.96 (1.69)</td>
<td>4.27 (1.49)</td>
<td>2.09 (2.03)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>EDE-Q – Eating Concern</td>
<td>0.46 (0.42)</td>
<td>2.91 (1.33)</td>
<td>2.74 (1.39)</td>
<td>3.30 (1.06)</td>
<td>1.71 (1.53)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>EDE-Q – Weight Concern</td>
<td>0.94 (0.85)</td>
<td>3.75 (1.57)</td>
<td>3.47 (1.60)</td>
<td>4.45 (1.24)</td>
<td>2.28 (1.65)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>EDE-Q – Shape Concern</td>
<td>0.96 (0.93)</td>
<td>4.09 (1.55)</td>
<td>3.85 (1.63)</td>
<td>4.69 (1.14)</td>
<td>2.82 (1.70)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>EDE-Q – Total Score</td>
<td>0.80 (0.64)</td>
<td>3.52 (1.32)</td>
<td>3.26 (1.36)</td>
<td>4.17 (0.95)</td>
<td>2.22 (1.40)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>SPSRQ – Punishment</td>
<td>8.35 (5.30)</td>
<td>14.44 (5.30)</td>
<td>13.57 (5.32)</td>
<td>16.54 (4.87)</td>
<td>–</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>SPSRQ – Reward</td>
<td>8.05 (3.28)</td>
<td>9.70 (4.15)</td>
<td>9.55 (4.11)</td>
<td>10.09 (4.25)</td>
<td>–</td>
<td>.001</td>
</tr>
</tbody>
</table>

Note: SPSRQ = Sensitivity to Punishment and Sensitivity to Reward Questionnaire (Torrubia et al., 2001), EDE-Q = Eating Disorder Examination Questionnaire (Fairburn & Bèglin, 1994), AN = Anorexia Nervosa.

$^a$ LS Lower; $^b$ higher education, for 28 participants of the AN-R group and 13 participants of the AN-P group educational level was missing.

$^b$ Chi-square test was performed.
sensitivity than healthy controls (AN-R vs. controls: mean difference = 1.50, p = .044, 95% CI [0.09,2.91]; AN-P vs. controls: mean difference = 2.04, p = .024, 95% CI [0.29,3.80]). However, AN-R and AN-P did not differ in their reward sensitivity (p = .75).

**Predictive value of punishment and reward sensitivity at baseline**

**EDE-Q total scores.** To test whether punishment and reward sensitivity were independently related to the EDE-Q scores at t1 we conducted linear regression analysis with SP and SR as predictors and EDE-Q total score at t1 as dependent variable. The model (F(2,234) = 45.8, p < .001, R² = .28) was significant and both SP and SR showed independent predictive validity for EDE-Q scores (SP: β = .48; SR: β = .20). In a second step, the interaction between SP and SR was added as predictor, but this did not improve the model significantly (Fchange(1,233) = 1.45, p = .26, R²change = .004).

**Percentage underweight.** To test whether punishment and reward sensitivity were independently related to the percentage underweight we conducted linear regression analysis with SP and SR as predictors and the percentage underweight at t1 as dependent variable. The model (F(2,234) = 16.90, p < .001, R² = .13) was significant, but only SP showed independent predictive validity for percentage underweight (SP: β = .35; SR: β = .04). In a second step, the interaction between SP and SR was added as predictor, but this did not improve the model significantly (Fchange(1,233) = 0.40, p = .55, R²change = .001).

**Predictive value of punishment sensitivity and reward sensitivity over time**

**EDE-Q total scores.** To test the predictive validity of SP and SR for severity of eating disorder symptoms after one year we conducted linear regression analysis with EDE-Q total score at t1 as predictor and EDE-Q total score at t2 as dependent variable. The model was shown to be marginally significant (F(1,148) = 5.83, p = .05, R² = .04), indicating that EDE-Q scores at baseline were positively related to EDE-Q scores one year later (β = .19). SP and SR were included as predictors in the second step, but this did not improve the model significantly (Fchange(2,146) = 0.50, p = .66, R²change = .006). In the final step, the interaction between SP and SR was added as predictor. Again this did not significantly improve the regression model (Fchange(1,145) = 0.49, p = .58, R²change = .003).

**Percentage underweight.** Linear regression analysis was used to test the predictive validity of SP and SR for percentage underweight one year later. In the first step, we included percentage underweight at t1 as predictor and percentage underweight at t2 as dependent variable. The model was not significant (F(1,148) = 1.62, p = .30, R² = .01), indicating that percentage underweight at baseline was not related to percentage underweight one year later. SP and SR were included as predictors in the second step, but this did not improve the model significantly (Fchange(2,146) = 0.92, p = .50, R²change = .01). In the final step, the interaction between SP and SR was added as predictor. Again this did not significantly improve the regression model (Fchange(1,145) = 0.75, p = .50, R²change = .005).

**Discussion**

The main goal of the present study was to explore whether self-reported punishment and reward sensitivity might be considered as risk factors for developing AN, and/or the persistence of AN symptoms. In line with prior studies in adults, outcomes showed that adolescents with AN already display higher levels of punishment sensitivity than healthy controls (Harrison et al., 2010; Jappe et al., 2011). However, in contrast to prior studies, individuals with AN-P showed (even) higher punishment sensitivity than individuals with AN-R. The latter finding might be related to greater symptom severity in the AN-P group than in the AN-R group (M difference EDE-Q total score = 0.92), since punishment sensitivity and symptom severity were positively correlated (all participants: r = .49). In addition, adolescents with AN showed higher levels of reward sensitivity than healthy controls. Our findings are in line with recent work among nonclinical adolescents which showed that specifically high sensitivity to punishment in combination with high sensitivity to reward was related to eating problems (Matton et al., 2013). In addition, we found that individuals with AN-P and AN-R did not significantly differ from each other with respect to reward sensitivity. This latter result is similar to the outcomes of Jappe and colleagues (2011), but differs from other studies showing that individuals with AN-R displayed lower levels of reward sensitivity than controls, while individuals with AN-P showed higher levels of reward sensitivity than individuals with AN-R and/or controls (Beck et al., 2009; Claes et al., 2006; Harrison et al., 2010).

The inconsistencies with prior studies in reward sensitivity might be due to different definitions and measurement instruments that are being used to measure reward sensitivity. Studies that showed lowered reward sensitivity in AN-R and heightened reward sensitivity in AN-P typically used the Behavioral Inhibition/Behavioral Activation System Scales (BIS/BAS) of Carver and White (1994) or the Temperament and Character Inventory (TCI) of Cloninger, Svrakic, and Przybeck (1993). Differences in reward sensitivity were only demonstrated for the ‘fun seeking’ subscale of the BIS/BAS and the ‘novelty seeking’ subscale of the TCI which were shown to measure impulsivity rather than reward sensitivity (Franken & Muris, 2006). Although reward sensitivity and impulsivity both are being linked to the behavioral activation system, the concepts are not interchangeable. Impulsivity typically refers to behaviors that are rash and spontaneous, while reward sensitivity reflects a heightened sensitivity to cues that signal reward (cf. Dawe, Gullo, & Loxton, 2004). Perhaps individuals with AN-P do not so much differ from AN-R in their sensitivity to reward, but in their impulsivity. The latter would explain why individuals with AN-P show purging behaviors or engage in episodes of binge eating. Consequently, future studies should include indices of both reward sensitivity and impulsivity to test whether indeed differences in impulsivity explain prior findings of higher reward sensitivity in AN-P than AN-R.

However, it still remains to be explained why individuals with AN typically score similar to controls on the other subscales of the BIS/BAS, ‘reward responsiveness’ and ‘drive’, while they score higher than controls on the RS subscale of the SPSRQ in the present study (and in Jappe et al. (2011)). The latter is especially puzzling, taking into account that Franken and Muris (2006) showed that these BIS/BAS subscales are loading higher on the factor that seemed to reflect reward sensitivity than the RS subscale of the SPSRQ. If individuals with AN would be actually more sensitive to reinforcement, the RS subscale of the SPSRQ measures sensitivity to overt rewarding stimuli, such as money, power, etc. In the present study differences between the AN group and controls were mainly driven by two items regarding appearance (items 6 and 14) and five items regarding interpersonal rewards (items 10, 16, 22, 44, 46). When we excluded these items from the analyses, the difference between the AN group and the control group were no longer

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1 When we repeated the analysis with the total scale score of the ChEDE interview at baseline as dependent variable, only SP showed independent predictive validity.

4 Similar results were obtained when using the total scale score of the ChEDE interview.
significant. The BIS/BAS does not contain any items on appearance or interpersonal reward. Thus, individuals with AN seem to be sensitive to reward within the domains of appearance and interpersonal feedback, instead of reward in general.

Taken together, the present pattern of findings suggests that individuals with AN showed heightened levels of punishment sensitivity and are more sensitive to rewards with respect to appearance and interpersonal feedback. In addition, punishment and reward sensitivity were independently related to the severity of eating disorder symptoms, but only punishment sensitivity was related to percentage underweight. These findings are in line with the idea that heightened punishment and reward sensitivity might be risk factors for developing AN, and that these temperamental factors could ‘help’ individuals to abstain from eating. That is, because of high sensitivity to punishment, fears like ‘becoming fat’ might become so prominent that individuals restrict their food intake, thereby increasing the risk for AN. Furthermore, the focus on appearance and obtaining approval of others might also set individuals at risk for developing anorectic symptoms, such as weight and shape concerns. However, since this study has a cross-sectional design, it is impossible to determine whether punishment and reward sensitivity indeed precede the onset of AN, or whether they are merely correlates of AN.

In addition, as a first step to examine whether reward and punishment sensitivity might be risk factors for the persistence of AN over time, we tested whether punishment and reward sensitivity predicted the course of AN symptoms. Both punishment and reward sensitivity were not related to eating disorder symptoms and percentage underweight one year later. So, although heightened punishment and reward sensitivity seem to be correlates of AN, apparently these characteristics are not related to changes in AN symptoms over time (due to treatment). Additionally, the influence of reward sensitivity on (the course of) AN symptoms was not moderated by punishment sensitivity or vice versa, since we did not find significant interaction effects. However, since AN patients received treatment as usual (without experimental control), it cannot be ruled out that individuals with higher levels of punishment and reward sensitivity received relatively intensive treatment, thereby counteracting the potential inadvertent influence of reinforcement sensitivity on the course of symptoms.

Limitations

The present study suffered from a substantial amount of drop-out (41%) which is unfortunately not exceptional in this field. Although we used missing data imputation, the outcomes of the longitudinal analyses should be interpreted with care. Additionally, it should be mentioned that the Reinforcement Sensitivity Theory meanwhile was updated (Gray & McNaughton, 2000), but that the SPSRQ is still based on the old model. Hereby, it seems that different measurement instruments in this area measure different aspects related to reinforcement sensitivity, which makes it a highly complex matter to compare outcomes across different studies in this field. Finally, it should be mentioned that we did not include measures of (trait) depression/anxiety, or other potentially relevant third variables in our design. Consequently, it is impossible to investigate whether reward and punishment sensitivity might be proxy (or overlapping) risk factors (for more information about different kind of risk factors that can be distinguished see: Kraemer, Kazdin, Offord, & Kessler, 1997; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001).

Future directions

To determine whether punishment and reward sensitivity indeed can be considered pre-morbid risk factors for developing AN, a crucial next step would be to longitudinally study their relationship with onset of AN in a healthy sample of adolescents. Subsequently, to see whether it concerns “fixed” markers or characteristics that are amenable to change, it would be important to examine whether successful treatment leaves reward and punishment sensitivity unchanged. A fruitful way to unravel which (causal) mechanisms underlie the link between punishment and reward sensitivity and AN could be the use of behavioral outcome measures. This might not only give more detail to the interpretation of the present findings, but also might show that behavioral and subjective reward and punishment sensitivity could be (partly) independent risk factors. Germane to this, research in the field of addiction recently showed that substance use in adolescents was related to enhanced attentional engagement with cues that predicted potential reward (van Hemel-Ruiter, de Jong, Oldenhinkel, & Ostafin, 2013). It seems valuable to apply similar behavioral tasks to the context of eating disorders. In addition, it seems important to link the behavioral tasks to functional brain activity, to see whether there exist differences in activation of reward and punishment circuits between AN individuals and healthy controls (e.g., Beaver et al., 2006). Obviously, such processes eventually should be longitudinally studied to determine whether they are pre-morbid risk factors for AN and/or for persistence of AN symptoms. When sensitivity to reward and punishment to punishment do appear to be important factors in the development and/or maintenance of AN, this could provide fresh theory-driven starting points for improving present interventions. Perhaps treatment for AN could be improved by more directly addressing these factors, such as learning patients to focus more on safety information instead of punishing information or using reward sensitivity to find alternative sources for functional rewards as a replacement of the dysfunctional role of the eating disorder (e.g., giving a sense of control, feeling special; cf. Fairburn, 2008).

Conclusion

The present pattern of findings showed that heightened reward and punishment sensitivity are correlates of AN, which is consistent with the view that they might be risk factors for developing AN. Especially punishment sensitivity seems associated with percentage underweight, and heightened punishment sensitivity may therefore be most important as a risk factor for AN. In line with the alleged critical importance of overvalued ideas about weight and shape in AN, specifically reward sensitivity with respect to appearance and interpersonal feedback was heightened in AN. There were no differences in this respect between AN-P and AN-R subgroups, thus enhanced reward sensitivity seems not especially relevant in explaining the presence of binging and purging behaviors in AN-P. The present study found no evidence that individuals’ reward and punishment sensitivity are risk factors for the persistence of AN over time. Thus, although the present study clearly demonstrated that heightened punishment and reward sensitivity are both linked to AN, these characteristics do not seem to make individuals with AN more resistant to treatment.

References


