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Research report

Heightened sensitivity to punishment and reward in anorexia nervosa



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ABSTRACT

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 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.

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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, Papies, & 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

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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, & Bijttebier, 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

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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 & Bèglin, 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.

Procedure 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 & Braet, 1999)). In addition, participants filled in the Dutch version of the EDE-Q (Fairburn & Bèglin, 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, Ávila, Moltó, & Caseras, 2001)). The SPSRQ is a selfreport instrument and contains 48 items in a ves/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

¹ 84 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.

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