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# Abnormal Reward Processing in Eating Disorders and Relevant Interventions

Zhengkun Fu[1, a, *, †] Xiaoyang Liu[2, b, *, †] Tong Su[3, c, *, †]

[1] *Renmin University of China, Beijing, Beijing,100086, China*
[2] *University of California, San Diego, La Jolla, CA 92093, USA*
[3] *University of California, Irvine, Irvine, CA 92697, USA*
[*] *Corresponding author. Email: [a]czh952@163.com, [b]xil067@ucsd.edu, [c]tsu7@uci.edu*
[†] *These authors contributed equally.*

**ABSTRACT**
The reward processing system plays a fundamental role in guiding people's behaviours and maintaining the body's operation. Thus, it is meaningful to concern about the changes in decision-making due to impaired reward processing as a factor of developing mental illness. Most studies of eating disorders emphasize psychological perspectives such as attachment mode, which means that the studies of binge eating disorders that focus on the reward processing system are quite limited. This narrative review focuses on the influence of brain regions and neuroendocrine in reward processing circuits on food reward valuation. Based on the studies of patients with different eating disorders, there are implications on the abnormal reward patterns regarding food appearing among people with binge eating disorder and anorexia nervosa and potential correlation with abnormality in related brain regions such as the orbitofrontal cortex. The human literature and experiments are conducted to test the relationship between abnormal reward processing and eating disorders. The correlation of impairments in flexible reward-based decision-making caused by dysfunction of brain areas and neuroendocrine in reward processing circuits and abnormal eating episodes with the support of neuroimaging evidence are also introduced. The efficacy of several interferences of reward response regarding abnormal eating behaviours as treatment of the eating disorder is discussed with respect to a series of control experiments. However, limited studies focusing on the relationship between the reward system and eating disorders haven't provided sufficient evidence to prove a robust correlation. In the future, more studies are expected to take place in natural settings instead of laboratory settings to improve the low validity of conclusions.

***Keywords:*** *Eating Disorders, Reward Circuitry, Neuroimaging, Neuroendocrinology.*

## 1. INTRODUCTION

Based on past and present literature, reward processing includes many components, like subjective value, reward facilitation, discounting, and learning. We can simply summarize it as the responsivity, including attitude and behaviour, to reward to understand it clearly [1]. But many studies showed it is inadequate to explain abnormalities just by the degree of integral reward responses [2]. In general, reward processing is often explored in psychology because of its high frequency of occurrence in individuals' life. For example, various plans in an individual's daily life are related to reward processing because it is tied to people's motivation and attitude towards obtaining rewards. Therefore, it will contribute to series of changes in decision-making and behaviour if the reward processing of individual changes even becomes abnormal. So, it has essential research value in generation, alterations, or avoiding individuals' behaviour and decision making. In addition, in the studies of psychopathology, the alterations in reward processing are always seen as a characteristic part. Some related symptoms have been regarded as diagnostic criteria for various mental disorders (e.g., depressive disorders, schizophrenia, eating disorders, additive disorders, etc.). Therefore, as for mental disorder, it is necessary to study the specific reward processing of eating disorder because eating is also a sort of behavior related to rewards. So it can provide a deeper understanding of characteristics of eating disorders for researchers and help them to develop more effective and efficient treatments for eating disorders.

The relationship between attachment and eating disorders has been widely investigated. In the review by Zachrisson and Skårderud, the findings regarding attachment and eating disorders are organized into three main categories of theories: retrospective approach, general risk approach, and attachment theoretical approach [3]. In the studies utilizing the retrospective approach, the authors proposed that the affected manifestation of eating disorder symptoms in adulthood, such as the feeling of insecurity and diffidence, might be traced back to early childhood experience, namely, disturbed parent-child relationship. The studies focusing on the general risk approach stress the concurrency of eating disorders and insecure attachment. The main findings highlight the high prevalence of people with eating disorders who also experienced insecure attachments. According to the studies looking at the attachment theoretical approach, the association between specific features of attachment and eating disorders is examined. Several studies find how disordered eating behaviours could be a direct expression of adverse attachment patterns. For example, food restriction can be interpreted as a way to distance from the self, which matches the main feature of the dismissing attachment pattern (i.e., people with dismissing attachment style tend to suppress their feelings). On the other hand, the lack of control feature of bulimic eating behaviour matches the characteristics of the preoccupied attachment pattern (i.e., people with preoccupied attachment style often craves proximity and affection). Another commonly examined topic around eating disorders is their relationship with anxiety disorder. In the review by Swinbourne and Touyz, studies related to the co-morbidity of eating disorders and different anxiety disorders are presented [4]. Several studies have found that OCD has a significant comorbidity rate with an eating disorder, especially with AN, since their symptom manifestations have many psychological and neurobiological overlaps.

The reward processing system plays a fundamental role in guiding people's behaviours and maintaining the body's operation. Thus, it is meaningful to concern about the changes in decision-making due to impaired reward processing as a factor of developing mental illness. Most studies of eating disorders emphasize psychological perspectives such as attachment mode, which means that the studies of binge eating disorders that focus on the reward processing system are quite limited. This narrative review focuses on the influence of brain regions and neuroendocrine in the part of reward processing circuits on food reward valuation. Based on the studies of patients with different eating disorders, there are implications on the abnormal reward patterns regarding food appearing among people with binge eating disorder and anorexia nervosa and potential correlation with abnormality in related brain regions such as the orbitofrontal cortex. The human literature and experiments that are conducted to test the relationship between abnormal reward processing and eating disorders, the correlation of impairments in flexible reward-based decision-making caused by dysfunction of brain areas as well as neuroendocrine in reward processing circuits and abnormal eating episodes with the support of neuroimaging evidence are also introduced. The efficacy of several interferences of reward response regarding abnormal eating behaviours as treatment of the eating disorder is discussed with respect to a series of control experiments. However, limited studies focusing on the relationship between the reward system and eating disorders haven't provided sufficient evidence to prove a robust correlation. In the future, more studies are expected to take place in natural settings instead of laboratory settings to improve the low validity of conclusions.

## 2. ATYPICAL REWARD PROCESSING ACROSS THE EATING DISORDER SPECTRUM

Delay discounting, as an important component of reward processing, which is defined as people's cognition that the value of things is often discounted over time, shows the ability to control desires and the willingness to delay rewards because of more interests. According to the main features of binge eating disorder: lack of ability to control their desire and tend to get rewards immediately, it is important to find out whether patients with binge eating disorder presented differently in the task and abnormal delay discounting compared with others. Manwaring and colleagues compared the different tendencies of delay discounting and probability discounting among obese women with binge eating disorder (BED), obese women without BED, and women in normal BMI (i.e., control group). They combined function and data from their delay and probability discounting tasks and evaluated the degree to individuals' discount by calculating the area under the curves (AuC) [5]. In detail, in the delay tasks, participants were required to make decisions between smaller but immediate obtained rewards and larger but delayed rewards (1 week, 1 month, 6 months, 1 year, and 3 years delayed) in different types of rewards (i.e., money, food, sedentary activity, massage time) [6]. In the probability discounting tasks, the mode of tasks is the same as the former one, while the larger but delayed rewards were replaced by larger but possible getting rewards in 10%, 25%, 50%, 75%, and 90% percentage [7]. Through analyses, they found that BED women showed a steeper trend to discount than obese women and the control group, which means women in the BED group have more impatience and risk aversion. In addition, compared with the other two non-monetary rewards (i.e., sedentary activity and massage), food rewards showed more steep discounting. To sum up, this study suggested different reward processing in women with BED and something special about the discounting of food rewards. So it can

be concluded that the reward processing of patients with binge eating disorders is abnormal.

Moreover, some other studies about sub-type of eating disorders might show the different discounting tendencies in reward processing. Anorexia nervosa (AN) presents different features from BED: it tends to be on a diet excessively. So their delay discounting might differ from patients with BED, which means AN patients may prefer to restrain their desires to get larger rewards and presented another behaviour in the task. For example, in the study by Steinglass et al., the relations between anorexia nervosa and temporal discounting were shown. It required participants to make choices in the intertemporal choice task and titration task: the former task required participants to make choices between smaller sooner (SS) reward and larger later (LL) reward and the later one required people to make choices when they were willing to change their decisions from SS reward to LL by the increasing value of LL reward in fixed delayed time (e.g., 3 months) [8]. After generating 2 variables: discount rate and discount factor, to assess the degree of temporal discounting, the researchers found that the AN group has a significant statistical difference from the control group in the discount rate and discount factor. In more detail, the AN group had a lower discount rate and higher discount factors than the control group, which means the AN group preferred larger-later rewards compared with the control group. In addition, research also suggested less steep discounting of AN was related to the high level of trait anxiety depending on the evidences of high scores in scale (STAI-trait) among AN group and high relations between trait anxiety and both discount rate and factors. Therefore, these results indicate that AN patients are more able to tolerate delayed rewards than ordinary people.

Based on the above behavioural evidences, researchers still persistently find some other evidence in neurology to present its relation more comprehensively. In some research, people found that the orbitofrontal cortex is crucial to lead feeding behaviour. The insular is another region to be considered because of its importance to the taste-related reward system [9]. In the study by Wallace et al., the associations between thickness across the cortical surface and both the degree of drive for thinness, reflected by the scores of EDIThn and bulimia, reflected by the scores of EDIBul in a large sample of young adults, which means scores of EDIThn is positively associated with willingness to thinness and scores of EDIBul is positively associated with the degree of bulimic conditions [10]. It was found that the score of EDIThn and EDIBul were negatively correlated with thickness in orbitofrontal and insular, which is associated with food reward, perception, and an interception. In addition, this research also showed some other cortical regions which have positive relations with those three regions would be altered negatively by the scores of EDIThn and EDIBul (e.g., left prefrontal regions; sensorimotor cortices). Therefore, this study supported the idea of abnormality in related cortical regions. It indicates that eating disorder patients have a structural anomaly in brain regions related to reward processing compared with the ordinary individual.

## 3. NEURAL MECHANISM UNDERLYING THE ABNORMAL REWARD PROCESSING SYSTEM

Profit-seeking behavior-seeking advantages and avoiding disadvantages is the instinct of all animals and the basis for survival and reproduction. In the process of human evolution, the reward processing system has been continuously improved by incorporating higher-order brain regions and neuroendocrine systems to form a complex neural circuit and dominants the process of decision-making. Among this circuit, the medial prefrontal cortex (mPFC) is responsible for the flexible goal-directed behaviour - choosing rewards and predicting the value of actions [11]. The ventro-medial part of the prefrontal cortex (vmPFC), one part of mPFC, guides the fluctuating valuation processes, which rely on incorporating environmental structure. It is intriguing to investigate whether the lack of behavioural control during binge-eating episodes for BN and BED patients correlates with the impairment of flexible behavioural adaption, which is overseeded by vmPFC. In the study by Reiter et al., the impairment in the flexible behavioural adaptation of binge-eating disorder patients was assessed using a computational psychiatry approach (which combines computational modelling with fMRI) [12]. The researchers recruited binge-eating disorder patients as an experimental group and healthy people as the control group. They asked them reward-guided decision-making tasks by choosing cards with different reward and punishment probability, which are anti-correlated, so that flexible behavioural adaptation is required to make effective decisions and maximize their chance of winning. The fMRI images of binge-eating disorder patients show statistically significant evidence of specific impaired behavioural adaptation caused by their decline of blood-oxygen-level-dependent (BOLD) -activation in the vmPFC. As a part of the reward processing system, the dysfunction of the ventro-medial prefrontal cortex suggests a certain correlation with eating disorders.

In the reward processing system, vmPFC regulates the amygdala, which oversees emotional arousal. Both amygdala and anterior cingulate cortex (ACC) play fundamental roles in regulating the cortex-basal nucleus reward circuit. The relationship between dysfunctional emotional regulation and abnormal eating behaviours is proposed and studied by monitoring emotional fluctuation caused by abnormity in key brain areas that can regulate the cortex-basal nucleus reward circuit. Wonderlich and colleagues assessed how acute stress correlates with the binge eating behaviours in a recruited group of women with bulimia nervosa disorder (18-40

years) using the test of momentary ecological assessment (EMA) and neuroimaging of brain areas associated with reward and emotional regulation [13]. Participants first completed their baseline assessment and fMRI scan. During the test, they received stimuli in the order of neutral cues, palatable food cues, stress induction, and food cues, and their brain activities were monitored by fMRI scan during this process. Their fMRI images of regions of interest (ROIs)- predetermined as ACC, vmPFC, and amygdala, were estimated cooperating with EMA analysis to generate computational modelling. Comparing the EMA-fMRI integration analysis with the baseline assessment, a statistically significant decline in BOLD-activation in vmPFC, right ACC, and left amygdala happened when participants viewed food cues after effective stress induction. Then, following binge-eating, the participants report an immediate decrease in negative affect. It suggests that their negative effect increases for female bulimia nervosa patients as their positive effect decreases when they see the food clue under acute stress; their negative effect moderates as while as their positive effect elevates after binge-eating episodes. After experiencing negative emotions, a lower level of amygdala responses is correlated with a higher level of vmPFC signals. For patients with an eating disorder, increased amygdala activity was closely associated with an emotionally aversive or appetitive.

In addition to the dysfunction of related brain areas in reward circuits, the neuroendocrine, which interacts with the mesolimbic dopamine system to mediate reward responses, regulates the homeostasis of maintaining energy. It indicates that disturbances in neuroendocrine might be a possible explanation of abnormal eating behaviours. Berner and colleagues did the literature review of animal and human neuroimaging and biological studies proving the interference in neuroendocrine such as peptide YY(PYY), hypothalamic-pituitary-adrenal (HPA) axis, and gonadal hormones lead to the subsequent disturbances in food reward valuation [14]. PYY is an anorexigenic hormone that generates to respond to food intake and increase satiety. As animal study proved, PYY3−36 (primary form of PYY) infusions in mice decrease the meal size and cause a similar effect in humans with modulation of neuronal activities in homeostasis dominating by the hypothalamus as well as reward circuits dominating by the amygdala, vTA, and insult, which lead to a decline in the reward value in food. As the study in humans predicted, the level of PYY is high for the groups of people with AN and is inconclusive for the groups of people with BN. HPA axis regulates stress and food intake by inducing the adrenal cortex to release glucocorticoids (GCs) which impact appetite and reward valuation of food in response to stress in humans. CHC and cortisol, the primary hormones of the HPA axis neuroendocrine, play an anorexigenic role under acute stress. On the other hand, chronically increased cortisol plays an orexigenic role under prolonged stress. In animal studies, acute elevation in GCs increases food craving and fat gain. In human studies, the HPA axis influences the choice of food intake incorporating with the reward circuit. For people with a higher level of stress, corresponding with a higher level of cortisol, prefer food with greater fat and sugar. Maladaptive HPA axis function leads to eating disorders as it alters the reward valuation system. In conclusion, disturbances in the neuroendocrine system indicate the altered brain reward responses and homeostasis and thus contribute to the behavioral symptoms of anorexia nervosa (AN) and bulimia nervosa (BN).

## 4. RELEVANT TREATMENTS FOR EATING DISORDERS

Patients with AN are found to associate reward feeling with weight loss cues, and such positive feedback seems to be the driving force that perpetuates their persistent pursuit to lower weight. Specifically targeting this misattribution, Haynos et al. propose that Positive Affect Treatment (PAT) could be used to promote reward responsivity in patients with AN [15]. PAT is a type of cognitive-behavioral intervention that targets to treat reward insensitivity in patients with mood and anxiety disorders [16]. Based on breaking the established association patients have with AN symptom, PAT-AN emphasizes eliciting positive, rewarding effect outside of eating disorders. The PAT-AN involves six modules, each with specific plan, and all aim to elicit positive affect. The researchers predict that by therapeutically remove the reward feeling with AN behaviour, patients would be able to stop relying on disordered eating to feel good and further experience more pleasure in other aspects of life. Although the efficacy of PAT-AN needs further investigation, it is an innovative approach that uniquely targets positive affect and reward sensation.

If the neural variation does exist in patients with BED, whether the efficacy of treatment depends on such variation among patients is speculated. In the study by Balodia et al., the relationship between the activation of the brain's reward system and the outcome following treatment of BED is examined [17]. Specifically, the activation of the inferior frontal gyrus (IFG), which is involved in inhibitory control, and the medial prefrontal cortex (mPFC), which is central for self-regulation, are measured [18]. The participants recruited were still engaged in binge eating after four months of sibutramine treatment and cognitive-behavioral-self-help intervention (BEpost-tx) and patients who have achieved remission from binge eating after treatment (NBEpost-tx). During the study, they were asked to complete two trials of monetary incentive delay task (MIDT), in which the participants were presented with either win or lose money option in the anticipatory phases and the revealing of their accumulated earning in the outcome phases, with a 4-6 second delay in between. FMRI measured the

participants' brain activation. The researchers have found that BEpost-tx has shown reduced recruitment of ventral striatal and IFG during reward anticipation and less mPFC activation during the outcome period during reward processing, compared to NBEpost-tx. The result indicates that the efficacy of treatment is closely linked to changed reward neural circuitry in BED patients. Thus, if the related neural area can be specifically targeted prior to treatment using intervention such as neurofeedback, the treatment effect could potentially be bolstered, and disordered eating behaviours such as bingeing could better be mitigated.

It is also worthy of examining the feasibility and efficacy of interference directly with the neural system to treat eating disorders. In the study by Schmidt and Martin, it was previously established that neurofeedback, a treatment in which patients are asked to consciously apply control over bodily responses with the assistance of EEG recording of the specific brain waves, has proven effective for treating disordered eating behaviours [19]. Based on the success of neurofeedback, it is possible that intervening directly with the reward system could also be effective in treating ED. In the study by Park et al., a treatment plan for mitigating severe and enduring anorexia nervosa (SE-AN), using deep-brain stimulation (DBS) on the ventral anterior limb of the internal capsule (ALIC) within NAcc, is proposed [20]. The study mainly applied DBS operation on patients' NAcc area and used magnetoencephalography (MEG) scanning to monitor neural responses. At the pre-operation phase, the patients underwent a whole-brain MRI and MEG scan to establish a baseline. DBS is applied to the ventral ALIC during the operative phase targeting the NAcc with side effects control. Then at the follow-up phase, symptom changes are collected through self-report and interviews. The neural changes are observed through a MEG scan of patients' brains in both resting states and during food wanting tasks. Although this study is at the preliminary testing stage, it provides insights into a potentially effective treatment option for SE-AN.

## 5. LIMITATION AND FUTURE DIRECTION

Most of the studies reviewed focused on observing the relationship between the reward system and one of the eating disorders. Yet only a few studies examine the relationship and connection among eating disorders and how they respond differently to a treatment. The comparison across reward processing manifestations of different eating disorders also remains scarce. Another limitation is that most of the studies occur in a laboratory setting and heavily rely on lab testing to determine treatment efficacy. For future reference, more studies could focus on exploring how intervention parameters would change if multiple eating disorders are considered. Also, to better eliminate response bias in participants and to capture more authentic responses, studies could expand to observations in natural settings. Moreover, observation in a non-lab environment can help determine if the effect of treatment is generated to other aspects of patients' lives. Another future direction studies can take testing to see if other factors, such as stress, trauma, etc., also come into play with reward response in patients with eating disorders.

## 6. CONCLUSION

This article has attempted to sum up relevant studies about abnormal reward processing and eating disorders, and the conclusions were summarized as follows. We focused on the presence of abnormal reward processing in the eating disorder spectrum. We concluded that the reward processing of eating disorder patients is abnormal and further indicated that BED patients could not stand delayed rewards. In contrast, AN patients prefer to control themselves for a longer period to get more rewards, both derived from normal extent. It was also found that people with an eating disorder have an abnormal thickness in the orbitofrontal cortex and insular, related to reward processing. Besides, the impairment of the ventro-medial part of the prefrontal cortex (vmPFC) was suggested to be associated with binge eating disorder patients' abnormal reward processing behaviour. And the degree of amygdala activity also has been suggested to be associated with emotionally aversive or appetitive of patients with bulimia nervosa, which is related to their abnormal reward processing. In addition, the neuroendocrine system is another crucial part of our review, and researchers believe that the abnormal brain reward responses and homeostasis can contribute to its dysfunction, which is related to AN or BN. Furthermore, regarding suitable treatments related to abnormal reward processing in eating disorders, first, PAT was an effective treatment for AN to remove the misattribution between reward feeling and weight loss cues. Second, the researches on a neural area related to the brain's reward system suggested that the effectiveness of treatments for BED patients could be influenced by alteration in reward neural circuitry. So, based on that and according to the studies on neurofeedback, it was suggested that the direct intervention with the reward system could be another effective treatment for ED in the future. Finally, we listed some limitations of past and present studies in this field: the deficiency in comparative researches on reward processing of different eating disorders and a slight disconnection between reality and laboratory testing. And based on these, there are some propositions in the future direction for reference. Researches can focus on the changes of parameters in intervention studies involving different eating disorders. Besides, more realistic experimental designs should be considered by more researchers. In the end, we also suggested another helpful direction, which is observing the influences of other factors in studies on reward response in eating disorders.

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