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Research report

Cognitive interference and a food-related memory bias in binge eating disorder ☆

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ABSTRACT

The present study was concerned with cognitive interference and a specific memory bias for eating-related stimuli in binge eating disorder (BED). Further objectives were to find out under which circumstances such effects would occur, and whether they are related with each other and with reported severity of BED symptoms. A group of women diagnosed with BED and a matched sample of overweight controls completed two paradigms, an *n-back* task with lures and a *recent-probes* task. The BED group generally experienced more interference in the *n-back* task. Additionally, they revealed selectively increased interference for food items in the *recent-probes* task. Findings can be reconciled with the view that control functions are generally impaired in BED, and that there is an additional bias for eating-related stimuli, both of which were related with reported severity of BED symptoms.

Introduction

Nowadays, the environment is characterized by an abundance of highly palatable food. Consequently, individuals are confronted with ongoing temptations motivating food intake. While most individuals can resist these temptations and are well ahead of losing control over their perceptions, cognitions, and behavior, some individuals may experience more difficulty to deal with these temptations, among these, especially individuals affected by binge eating disorder (BED).

BED is characterized by the occurrence of repetitive binge eating episodes in the absence of compensatory behavior (American Psychiatric Association [APA]; 1994). Thereby, these episodes are accompanied by a marked sense of loss of control. Individuals affected by this disorder often describe binge attacks as an urge to eat which is almost impossible to contain. Consequently, this leads to random consumption of whatever food offers itself, while it is of note that these episodes do not necessarily begin with feelings of physical hunger (APA, 1994).

Given narrative reviews (Brownley, Berkman, Sedway, Lohr, & Bulik, 2007; Wilfley & Cohen, 1997; Wilson & Shafran, 2005; Wonderlich, de Zwaan, Mitchell, Peterson, & Crow, 2003) and meta-analyses (Reas & Grilo, 2008; Vocks et al., 2010) on the efficacy of treatments for BED, cognitive-behavioral therapy (CBT) is currently recommended as the treatment of choice with regard to reducing binge eating frequency, over concern with eating, shape and weight and binge eating abstinence rates. However, drop-out rates in empirically-validated treatments still average around 20% (Wonderlich et al., 2003). Additionally, 30–50% of individuals fail to refrain from binge eating in long-term follow-up assessments (Brownley et al., 2007; Vocks et al., 2010; Wonderlich et al., 2003), although somewhat higher, binge eating remission rates of 65% for CBT guided self-help and interpersonal psychotherapy have been reported in a recent study at two year follow-up (Wilson, Wilfley, Agras, & Bryson, 2010). Thus, there is a great need for research identifying factors that may contribute to binge eating in order to enhance the development of effective treatments. According to cognitive theories, eating disorders are determined, in part, by biases of information processing. Specifically, it is assumed that body-related and/or eating-related stimuli are processed differently from other stimuli (Williamson, White, York-Crowe, & Stewart, 2004). This has been accounted for in terms of schema activation and schema-consistent information processing, and has been deemed responsible for the development

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and maintenance of eating disorders (Vitousek & Hollon, 1990; Williamson, Muller, Reas, & Thaw, 1999). However, most of the research on biases has been conducted in anorexia (AN) and bulimia (BN) nervosa.

Contrary to AN and BN, binge eating in BED rarely results from physiologically induced hunger. Furthermore, while overvaluation of shape and weight is an inherent part of AN and BN, a substantial number of BED patients has normal levels of shape and weight concerns when compared to their obese counterparts without BED (Grilo, Crosby, et al., 2009). As such, most of the studies assessing biased information processing in BED focus on eating-related stimuli. For instance, in one study (Svaldi, Tuschen-Caffier, Peyk, & Blechert, 2010), individuals with BED compared to overweight controls displayed an enhanced processing of high-caloric food pictures, as indexed by larger long latency EEG potentials. By contrast, no group differences were found for low-caloric food pictures. Hence, attentional processes seem to be affected in BED.

But also control functions may be impaired in BED. In line with this notion, individuals with binge eating characteristics indicate to be generally more impulsive than control participants in self-report questionnaires (Galanti, Gluck, & Geliebter, 2007; Nasser, Gluck, & Geliebter, 2004). Similarly, there is behavioral evidence of impaired response inhibition compared to obese controls (Mobbs, Iglesias, Golay, & Van der Linden, 2011). Additionally, BED patients were shown to discount delayed rewards more strongly and to engage in risky decisions neglecting long-term goals compared with obese controls (Davis, Levitan, Muglia, Bewell, & Kennedy, 2004; Davis, Patte, Curtis, & Reid, 2010; Svaldi, Brand, & Tuschen-Caffier, 2010). Of note, current models of executive control posit that control functions operating at different stages of processing are only moderately correlated (Friedman & Miyake, 2004; Hasher, Lustig, & Zacks, 2007; Nigg, 2000; Stahl et al., in press). To conclude, despite the evidence of early attentional biases as well as late-stage inhibitory deficits, it is difficult to say to what extent the central stage of cognitive representation and manipulation is affected in BED.

This limited attention given to cognitive processes (i.e. after stimulus encoding and prior to selecting a response) is surprising, given that binge attacks are neither solely driven by palatable stimuli in the environment nor by an inability to control behavioral impulses alone. We suggest eating-related cognitions may play a role as well by interfering with current cognitive processing. This would be particularly the case if BED patients have difficulty to counteract such eating-related cognitive interference. The current study was designed to test this prediction.

To this end, we employed two standard paradigms used in cognitive interference research, namely the *n-back task with lures* (e.g., Schaefer et al., 2006) and the *recent-probes task* (Monsell, 1978; Nee, Wager, & John, 2007). The *n-back* task may be considered a more general task tapping the level of functioning of working memory, requiring the updating of working memory, shielding of relevant information and interference control. In contrast, the recent probes paradigm can be considered a more specific measure of proactive interference, i.e., to what extent previously processed information interferes with current processes.

In each task, we presented an equal number of eating-related and neutral stimuli. Our reasoning was that increased effects across stimuli of all categories would indicate generally increased cognitive interference (or impaired cognitive interference control, respectively), whereas particularly increased cognitive interference for eating-related stimuli relative to neutral stimuli would be indicative of a specific eating-related bias. Given both effects occur, we also wanted to test the correlations of general interference and an eating-related bias in BED as well as the relationships with clinically relevant symptoms of BED.

To sum up, in the current study we tested the following hypotheses: (1) Cognitive interference is generally increased in BED, as indicated by interference effects across stimuli of all categories. (2) There is a specific eating-related bias in BED, as indicated by specifically increased interference effects for eating-related stimuli relative to neutral stimuli. (3) The magnitude of general cognitive interference is correlated with the magnitude of an eating-related bias. Additionally, both are predictive of clinically relevant symptoms of BED.

Method

Participants

BED ($n = 31$) and the overweight control group (CG; $n = 36$) were recruited in separate announcements in newspapers and television. Additionally, BED participants were recruited from the affiliated outpatient clinic (waiting list). BED announcements asked for women suffering from binge attacks. CG announcements asked for overweight women. Inclusion criteria for the BED group was a current diagnosis of BED according to criteria of the *Diagnostic and statistical manual of mental disorders* (DSM-IV-TR; Association, 2000). Inclusion criteria for the CG was a Body Mass Index (BMI = weight/height^2) $\geqslant 25$ in the absence of a current or lifetime diagnosis of an eating disorder. Exclusion criteria for both groups were the presence of current substance abuse or addiction (except sustained full remission), bipolar disorder, current or past psychosis, schizophrenia and current suicidal ideation. All participants had to have an age of $\geqslant 18$ years.

In total, 121 participants were screened via a detailed telephone interview. If eligible ($n = 79$), they were invited for a formal diagnostic interview. All diagnoses were determined by means of the Structured Clinical Interview (SCID) for DSM-IV Axis I (Spitzer, Williams, Gibbon, & First, 1992) and the Eating Disorder Examination (Fairburn & Cooper, 1993). The interviews were conducted by three students at Ph.D. level and at the end of their training as cognitive-behavioral therapists. One additional interviewer was at Msc. level and at the end of her training as a cognitive-behavioral therapist. They had all previously undergone a two-day SCID and a three-day EDE training. Furthermore, all of them were employed at the affiliated outpatient clinic, which has a strong emphasis on eating disorders. Additionally, the first author (JS) checked the first five audiotaped interviews of each interviewer for diagnostic validity to ensure reliability of diagnoses. Of the 79 participants invited for the diagnostic assessment, seven invited participants did not show up to the scheduled assessment and were afterwards unreachable. Another five participants had to be excluded during the formal diagnostic sessions, not fulfilling the established inclusion and exclusion criteria. Our final sample consisted of 31 women with BED and 36 women in the CG. All participants were German native speakers. They gave informed consent prior to study participation. They received 25€ for study participation. The study was approved by the local ethical committee.

Groups did not differ significantly on BMI and age (see Table 1 for descriptive statistics), as well as on educational level, marital status, and income (all $ps > 0.41$). As expected, participants with BED scored significantly higher on scales assessing severity of eating pathology and severity of depression. As the sample in the *n-back* task with lures was slightly smaller (see procedure for further details), sociodemographics and overall pathology are presented separately for the two paradigms.

Women with BED had a mean of 15.73 ($SD = 9.21$; range = 0–36) binges per week over the four weeks prior to testing. Consistent with other studies (Wilfley, Schwartz, Spurrell, & Fairburn, 2000), comorbidity in the BED sample was high: 32.3% ($n = 10$) had no comorbid disorder, 35.5% ($n = 11$) had one, 16.1% ($n = 5$) had two and 16.1% ($n = 5$) had three or more comorbid disorders. Thereby,