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2 **Research** report

Cognitive interference and a food-related memory bias in binge eating 6 4 7 disorder ☆

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Introduction 45

46 Q3 Nowadays, the environment is characterized by an abundance 47 of highly palatable food. Consequently, individuals are confronted 48 with ongoing temptations motivating food intake. While most 49 individuals can resist these temptations and are well ahead of los-50 ing control over their perceptions, cognitions, and behavior, some 51 individuals may experience more difficulty to deal with these temptations, among these, especially individuals affected by binge 52 eating disorder (BED). 53

BED is characterized by the occurrence of repetitive binge eat-54 ing episodes in the absence of compensatory behavior (American 55 Psychiatric Association [APA]; 1994). Thereby, these episodes are 56 accompanied by a marked sense of loss of control. Individuals af-57 fected by this disorder often describe binge attacks as an urge to 58 eat which is almost impossible to contain. Consequently, this leads 59 60 to random consumption of whatever food offers itself, while it is of

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ABSTRACT

The present study was concerned with cognitive interference and a specific memory bias for eatingrelated stimuli in 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 39 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 41 related with reported severity of BED symptoms.

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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; 64 Wonderlich, de Zwaan, Mitchell, Peterson, & Crow, 2003) and 65 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 71 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

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84 processed differently from other stimuli (Williamson, White, York-85 Crowe, & Stewart, 2004). This has been accounted for in terms of 86 schema activation and schema-consistent information processing. 87 and has been deemed responsible for the development and main-88 tenance of eating disorders (Vitousek & Hollon, 1990; Williamson, 89 Muller, Reas, & Thaw, 1999). However, most of the research on 90 biases has been conducted in anorexia (AN) and bulimia (BN) 91 nervosa.

92 Contrary to anorexia nervosa (AN, binge-purge type) and buli-93 mia nervosa (BN), binge eating in BED rarely results from physiologically induced hunger. Furthermore, while overvaluation of 94 95 shape and weight is an inherent part of AN and BN, a substantial number of BED patients has normal levels of shape and weight 96 concerns when compared to their obese counterparts without 97 98 Q4 BED (Grilo, Crosby, et al., 2009). As such, most of the studies assess-99 ing biased information processing in BED focus on eating-related 100 stimuli. For instance, in one study (Svaldi, Tuschen-Caffier, Pevk, 101 & Blechert, 2010), individuals with BED compared to overweight 102 controls displayed an enhanced processing of high-caloric food pictures, as indexed by larger long latency EEG potentials. By contrast, 103 104 no group differences were found for low-caloric food pictures. 105 Hence, attentional processes seem to be affected in BED.

But also control functions may be impaired in BED. In line with 106 107 this notion, individuals with binge eating characteristics indicate 108 to be generally more impulsive than control participants in self-re-109 port questionnaires (Galanti, Gluck, & Geliebter, 2007; Nasser, 110 Gluck, & Geliebter, 2004). Similarly, there is behavioral evidence 111 of impaired response inhibition compared to obese controls (Mobbs, Iglesias, Golay, & Van der Linden, 2011). Additionally, BED pa-112 113 tients were shown to discount delayed rewards more strongly and to engage in risky decisions neglecting long-term goals com-114 115 pared with obese controls (Davis, Levitan, Muglia, Bewell, & Kennedy, 2004; Davis, Patte, Curtis, & Reid, 2010; Svaldi, Brand, & 116 117 Tuschen-Caffier, 2010). Of note, current models of executive con-118 trol posit that control functions operating at different stages of pro-119 cessing are only moderately correlated (Friedman & Miyake, 2004; 120 Hasher, Lustig, & Zacks, 2007; Nigg, 2000; Stahl et al., in press). To 121 conclude, despite the evidence of early attentional biases as well as 122 late-stage inhibitory deficits, it is difficult to say to what extent the 123 central stage of cognitive representation and manipulation is af-124 fected in BED.

125 This limited attention given to cognitive processes (i.e. after stimulus encoding and prior to selecting a response) is surprising, 126 127 given that binge attacks are neither solely driven by palatable stimuli in the environment nor by an inability to control behavioral 128 129 impulses alone. We suggest eating-related cognitions may play a 130 role as well by interfering with current cognitive processing. This 131 would be particularly the case if BED patients have difficulty to 132 counteract such eating-related cognitive interference. The current 133 study was designed to test this prediction.

134 To this end, we employed two standard paradigms used in cognitive interference research, namely the *n*-back task with lures (e.g., 135 Schaefer et al., 2006) and the recent-probes task (Monsell, 1978; 136 Nee, Wager, & John, 2007). The *n*-back task may be considered a 137 138 more general task tapping the level of functioning of working memory, requiring the updating of working memory, shielding of 139 140 relevant information and interference control. In contrast, the recent probes paradigm can be considered a more specific measure 141 of proactive interference, i.e., to what extent previously processed 142 143 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) 165 were recruited in separate announcements in newspapers and 166 television. Additionally, BED participants were recruited from the 167 affiliated outpatient clinic (waiting list). BED announcements 168 asked for women suffering from binge attacks. CG announcements 169 asked for overweight women. Inclusion criteria for the BED group 170 was a current diagnosis of BED according to criteria of the Diagnos-171 tic and statistical manual of mental disorders (DSM-IV-TR; Associa-172 tion, 2000). Inclusion criteria for the CG was a Body Mass Index 173 $(BMI = weight/height^2) \ge 25$ in the absence of a current or lifetime 174 diagnosis of an eating disorder. Exclusion criteria for both groups 175 were the presence of current substance abuse or addiction (except 176 sustained full remission), bipolar disorder, current or past psycho-177 sis, schizophrenia and current suicidal ideation. All participants 178 had to have an age of ≥ 18 years. 179

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.

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