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Research report

The independent and interacting effects of hedonic hunger and executive function on binge eating ☆

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ABSTRACT

Poor executive function (EF; pre-frontal cognitive control processes governing goal-directed behavior) and elevated hedonic hunger (i.e., preoccupation with palatable foods in the absence of physiological hunger) are theoretical risk and maintenance factors for binge eating (BE) distinct from general obesity. Recent theoretical models posit that dysregulated behavior such as BE may result from a combination of elevated appetitive drive (e.g., hedonic hunger) and decreased EF (e.g., inhibitory control and delayed discounting). The present study sought to test this model in distinguishing BE from general obesity by examining the independent and interactive associations of EF and hedonic hunger with BE group status (i.e., odds of categorization in BE group versus non-BE group). Treatment-seeking overweight and obese women with BE ($n = 31$) and without BE (OW group; $n = 43$) were assessed on measures of hedonic hunger and EF (inhibitory control and delay discounting). Elevated hedonic hunger increased the likelihood of categorization in the BE group, regardless of EF. When hedonic hunger was low, poor EF increased the likelihood of categorization in the BE group. Results indicate that the interplay of increased appetitive drives and decreased cognitive function may distinguish BE from overweight/obesity. Future longitudinal investigations of the combinatory effect of hedonic hunger and EF in increasing risk for developing BE are warranted, and may inform future treatment development to target these factors.

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Introduction

Binge eating (BE) is defined as consuming large amounts of food within a discrete time period, accompanied by a sense of loss of control (LOC). BE is a key symptom of binge eating disorder (BED), and is linked to serious psychological and physical co-morbidity (Latner, Hildebrandt, Rosewall, Chisholm, & Hayashi, 2007) and impaired social functioning (Rieger, Wilfley, Stein, Marino, & Crow, 2005; Robinson et al., 2006). A majority of those with BED are overweight or obese (Hudson, Hiripi, Pope, & Kessler, 2007); however, individuals with BED demonstrate increased functional impairment compared to non-binge eating overweight and obese peers with similar BMIs (Wilfley, Wilson, & Agras, 2003).

Although the current diagnostic criteria for BED require that an individual's binge episodes consist of an "objectively large" amount of food, mounting evidence indicates that the presence of LOC is the primary indicator of BE severity and associated psychosocial impairment. Indeed, empirical research has shown that the presence and severity of LOC eating (Colles, Dixon, & O'Brien, 2008), and frequency of LOC eating episodes (Latner et al., 2007; Picot & Lilenfeld, 2003), are closely related to eating disorder psychopathology, even when the amount consumed in a given episode fails to reach an objectively large size. Individuals with BED or sub-threshold BED appear to be similar in terms of psychosocial and quality of life impairment, weight outcomes, and psychological distress (Mond, Latner, Hay, Owen, & Rodgers, 2010). Thus, for the purposes of the current study, we refer to BE pathology as recurrent episodes LOC eating, including objective and subjective binge sizes.

Despite the fact that BE and BED are much more common among those who are overweight and obese, most overweight and obese individuals do not endorse BE pathology (Hsu et al., 2002). Thus, research has begun to examine variables involved in the development and maintenance of BE as distinct from those associated with being overweight or obese. Deficits in cognitive function have been investigated as possible risk and maintenance factors for BE – particularly deficits in executive function (EF), which represent higher-order control processes that govern goal-directed behavior (Van den Eynde et al., 2011). A large body of evidence has linked EF deficits

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with obesity in the absence of BE (Smith, Hay, Campbell, & Trollor, 2011); however, research has also suggested that BE may present with distinct and more pronounced cognitive deficits (Duchesne et al., 2010; Manasse et al., 2014). Deficits along two dimensions of EF show compelling theoretical and empirical associations with BED: 1) inhibitory control, manifested as reduced inhibition of prepotent responses (Duchesne et al., 2010; Manasse et al., 2014; Svaldi, Naumann, Trentowska, & Schmitz, 2014), and 2) monetary delay discounting, or the preference for immediate, smaller reward over delayed, larger reward (Manasse et al., 2015; Manwaring, Green, Myerson, Strube, & Wilfley, 2011). Although preliminary studies have implicated deficits in these EF domains in BED, the evidence remains mixed (Van den Eynde et al., 2011). For example, three (Duchesne et al., 2010; Mobbs, Iglesias, Golay, & Van der Linden, 2011; Svaldi et al., 2014) of six total studies have detected inhibitory control deficits in individuals with binge eating compared to weight-matched controls. Similarly, one study found no differences in delay discounting between obese BED and non-BED subjects, (Davis, Patte, Curtis, & Reid, 2010), although another reported steeper discounting (i.e., choosing smaller, short-term rewards in preference to larger, long-term rewards) in obese BED versus non-BED subjects (Manasse et al., 2015). Although inhibitory control and delay discounting reflect dimensions of impulsivity broadly, research suggests that inhibitory control measured via tasks such as the Stroop or stop-signal paradigms are classified as measures of "impulsive inhibition" (i.e., late-stage inhibition of a prepotent response), whereas delayed discounting may be classified as a measure of "impulsive decision-making" (i.e., deliberate choice of a smaller short-term over a larger, long-term reward) (Reynolds, Ortengren, Richards, & de Wit, 2006). Thus, these distinct constructs that theoretically underlie impulsive behavior each warrant investigation as variables that play a role in the maintenance of BE.

However, one possible reason for inconsistent results across studies is a failure to consider potential moderating variables. In fact, an influential theory of self-control posits that poor EF interacts with *increased appetitive drive* to predict dysregulated behavior, such as BE or alcohol use (Hofmann, Friese, & Strack, 2009; Hofmann, Rauch, & Gawronski, 2007). According to this theory, EF processes are necessary to override persistent and difficult-to-control urges or impulses. Thus, when appetitive desire is high, a well-functioning EF system may be essential to prevent dysregulated behavior from occurring; however, if appetitive desire is low, only minimal EF capabilities may be necessary to regulate behavior (e.g., overeating) (Nederkoorn, Houben, Hofmann, Roefs, & Jansen, 2010; Rollins, Dearing, & Epstein, 2010).

One common conceptualization of increased appetitive desire for food is "hedonic hunger," which refers to a preoccupation with highly palatable food when not physically hungry (Lowe & Butryn, 2007). Overweight individuals with BE may be distinguished from overweight counterparts without BE by a *combination* of elevated hedonic hunger and reduced EF, and emerging evidence shows that this combination predicts palatable food intake in overweight and obese samples without BE when they are energy replete (Appelhans et al., 2011). One study found that hedonic hunger, as measured by the Power of Food Scale (Cappelleri et al., 2009; Lowe et al., 2009), is positively related to BE frequency in those with anorexia nervosa or bulimia nervosa (Witt & Lowe, 2014), but no studies have directly compared hedonic hunger between obese samples with and without BED. Additionally, no studies have tested the interacting effect of hedonic hunger and executive dysfunction on the presence of BE, over and above overweight and obesity.

As such, the current study sought to test Hofmann's model of self-control in predicting the presence of BE in an overweight and obese sample. First, we hypothesized that elevated hedonic hunger, poorer inhibitory control and increased delayed discounting would each be independently associated with BE status. We additionally hypothesized that hedonic hunger would moderate the association of executive dysfunction (specifically, inhibitory control and delayed discounting) with membership in BE and non-BE groups. Specifically, we hypothesized that EF deficits would be most strongly associated with the presence of BE at the highest levels of hedonic hunger.

Methods

Participants and procedure

The current study included overweight and obese (BMI = 26–50 kg/m^2) females who endorsed BE in the preceding three months (BE group) and a group of overweight and obese women without any past or present BE (OW group). Participants were seeking treatment for weight loss and/or BE. All participants provided informed consent.

Participants in the OW group ($n = 43$) met the following criteria: 1) no LOC eating episodes in the past 3 months and 2) no current or past history of BE or eating disorder (e.g., anorexia nervosa, bulimia nervosa, BED). Participants in the BE group (BE; $n = 31$) must have endorsed an average of at least one subjective or objective binge episode per week over the past three months (12 total binge episodes over the past 3 months), and must not have met criteria for bulimia nervosa. We chose to include those with subjectively large binge episodes (i.e., subthreshold BED) given evidence that neurocognitive factors (Manasse et al., 2014) and functional impairment associated with binge eating is most associated with presence of LOC, rather than size of binge episodes (Latner et al., 2007; Mond et al., 2006).

Recruitment took place over the course of one year (June 2013–May 2014). A neuropsychological battery and BE screening were included as part of a baseline assessment prior to entry in either intervention. A licensed clinical psychologist supervised all neuropsychological assessments. Order of administration of tasks was randomly generated for each participant to control for order effects. Participants received free treatment from either of the trials and also received $50 for completion of the assessments. The study protocol was approved by Drexel University's Institutional Review Board.

Measures

Binge eating

The Eating Disorders Examination (EDE) version 16D is the gold-standard semi-structured interview for assessing for BE (Grilo, Masheb, Lozano-Blanco, & Barry, 2004; Wilfley, Schwartz, Spurrell, & Fairburn, 1997). The Overeating section ("Questions for Identifying Bulimic Episodes and Other Episodes of Overeating") was administered to all participants to examine for presence of LOC eating and BE. The EDE has high inter-rater reliability and test–retest reliability (Rizvi, Peterson, Crow, & Agras, 2000) and good internal consistency (Cooper, Cooper, & Fairburn, 1989).

IQ

Wechsler Test of Adult Reading (WTAR) (Wechsler, 2001): The WTAR is a single-word oral reading test used to estimate verbal intelligence; scores were converted to Full Scale IQ estimates. The WTAR has strong correlations (.70–.80) with WAIS-III FSIQ scores for a wide age range of WTAR scores (Wechsler, 2001).

Inhibitory control

The Delis–Kaplan Executive Functioning System Color–Word Interference Task (D-KEFS) (Delis, Kaplan, & Kramer, 2001): Color–Word Interference is a modified Stroop task assessing response inhibition in the presence of distractors. This modified Stroop task contained four trials: 1) Participants were presented with blocks

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