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Research report

The influence of restrained and external eating patterns on overeating

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Abstract

Eating in response to an increasingly obesogenic environment has been strongly implicated as a salient aspect of eating behaviour, arguably influenced by learning and experience. Interindividual differences in susceptibility to weight gain may be due, in part, to variability in response to environmental triggers. The phenomenon of food craving may also be an important factor influencing appetite control. The present study tested a model, in which food craving was hypothesised to be an intervening causal variable, on a causal pathway between responsivity to environmental cues and the development of obesity. One hundred and twenty four participants (aged 21–71 years, 83 females and 41 males) completed the study. Participants completed the Dutch eating behaviour questionnaire (DEBQ), measuring external eating (externality), emotional eating (emotionality) and restrained eating behaviour (restraint), and an adapted form of the food craving inventory (FCI), assessing cravings for carbohydrate, fats, sweets and fast food fats, in addition to total food cravings. Initial analysis showed positive correlations between FCI-tot and body mass index (BMI), FCI-fats and BMI and FCI-fast food fats and BMI in both men and women, and between FCI-carbohydrates and BMI in men only. Multiple regression analyses showed externality as the principal predictor of food craving, which was greater in males compared to females, but differential for different food groups between genders. Restrained eating and cravings for fats and fast food fats were negatively associated in women only. As predicted, total cravings, and cravings for fats and fast food foods mediated the positive association between external eating and BMI. It is concluded that appetitive response to external cues as an important risk factor in appetite control is mediated through cravings for particular food groups and is gender-dependent.

Keywords: Food craving; Externality; Mediation; Restraint; Body mass index (BMI); Carbohydrates; Sweet foods; Fats; Fast-food fats

Introduction

The phenomenon of food craving (“an irresistible urge to consume a specific food”) (Waters, Hill, & Waller, 2001; Weingarten, 1990) has been implicated as an important factor influencing appetite control (Blundell & Finlayson, 2004; Strachan, Ewing, Frier, Harper, & Daery, 2004; Waters et al., 2001). Positive associations have been shown between food cravings and excessive overeating (Hetherington & Macdiarmid, 1995), BMI (Delahanty, Meigs, Hayden, Williamson, & Nathan, 2002; Rodin, Manuso, Granger, & Nelbach, 1991), snacking behaviour (Basdevant, Craplet, & Guy-Grand, 1993), binge eating and bulimia (Greeno, Wing, & Shiffman, 2000; Waters, Hill, & Waller, 2001) and low compliance with weight reducing programmes (Delahanty et al., 2002). In relation to dietary compliance, sustained adherence to a diet, rather than diet type, has been shown to be the key predictor of weight loss and cardiac risk (factor) reduction (Dansinger, 2005) and this is important in the context of the ever-changing diet climate and industry. Increasing adherence to any one diet shows that the efficacy of weight loss and maintenance programmes will vary between individuals (Hill, 2005) and is a major challenge for public health interventions. Thus, explanations of interpersonal differences in dietary adherence are of importance.

There is evidence of a link between dietary restraint and food craving, with restrained eaters often reporting higher food craving scores and showing higher levels of disinhibition and binge eating than unrestrained eaters (Cepeda-Benito, Fernandez, & Moreno, 2003; Hill, Weaver, &

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Blundell, 1991; Nammi, Saisudha, Chinnala, & Boini, 2004). The interpretation of scores of restraint, however, has depended very much upon the choice of restraint scale, with different scales reflecting unsuccessful (Three factor eating questionnaire, TFEQ; Revised restraint scale, RSS) or successful (Dutch eating behaviour questionnaire, DEBQ) restraint (Heatherton, Herman, Polivy, King, & McGree, 1988; Lowe, 1993; Mela & Rogers, 1998; Rogers & Smit, 2000). Moreover, the correlation between restraint and food craving incorporates the usual complication of uncertainty over cause and effect. I.e. food cravings may be either a consequence of or part of the aetiology of long-term dietary restraint and/or short term dieting.

The ability of external cues to elicit physiological reactivity, leading to increased food intake, has been extensively investigated (Nederkoorn, Smulders, Havermans, & Jansen, 2004; Nederkoorn, Smulders, & Jansen, 2000; Van Strien & Ouwens, 2003) and cue reactivity/externality is recognised as a salient aspect of eating behaviour, arguably influenced by learning and experience (Gibson, 2001). There is an ongoing discussion of the link between obesity trends and the environment (Cordain et al., 2005; Rogers, 1999) and it is becoming increasingly difficult to resist environmental cues, which may override any homeostatic control of food intake. The interindividual differences in susceptibility to weight gain (Blundell et al., 2005; Maes, Neale, & Eaves, 1997) may be due, in part, to variability in response to environmental triggers (Blundell & Finlayson, 2004). For example, it has been demonstrated that response to a variety of external cues is heightened in restrained eaters (Cools, Schotte, & McNally, 1992; Ogden & Wardle, 1990).

Incentive-associated stimuli are a powerful stimulus for desire, and often effective in reinitiating motivated behaviour. It has been suggested that environmental cues associated with foods that are craved produce neural representations of the craved foods together with a desire to seek and consume the food, explained by Pavlovian and/or instrumental models of food craving (Zellner & Edwards, 2005). For instance, the sight of food can lead to craving, even when in a state of satiation (Cornell, Rodin, & Weingarten, 1989), supporting a conditioned-incentive model of food craving. Alternatively, stimuli that signal food delivery have also been suggested to cause withdrawal and other compensatory-like responses in the same way as drug cues, for example in conditioned cephalic phase insulin release (Brand-Miller, Holt, de Jong, & Petocz, 2003; Teff, 2000; Teff, Levin, & Engelman, 1993), supporting a conditioned-drive model. Moreover, extinction studies where the conditioned stimulus (food cue) is repeatedly presented without the unconditioned stimulus (food) support Pavlovian models. It is well established that highly palatable foods are associated with high hedonics (Blundell et al., 2005; Drewnowski & Greenwood, 1983); thereby providing a rewarding pleasure response. Therefore, anticipation of hedonics (the reinforcing outcome) would be expected to increase craving, supporting an instrumental component (Zellner & Edwards, 2005). The link between externality, conditioning and food craving has been supported by recent evidence of hyperactivity in the orbitofrontal cortex, an area of the cortex involved in stimulus–stimulus association learning, concomitant with the experience of food craving (Rolls, 1999; Uher et al., 2005).

In the light of the above, this study aimed to demonstrate a greater association between food craving and external eating behaviour scores, in comparison with emotional and restrained eating scores, as assessed by the DEBQ. The use of the White FCI (White, Whisenhunt, Williamson, Greenway, & Netemeyer, 2002) as a reliable and valid measure of general and specific food cravings facilitates the investigation of which food groups are paramount in such positive associations. In addition, the study aimed to test a mediation model, in which we expected to see a positive statistical relationship between externality and BMI, explained by a positive association between food craving and both externality and BMI. Thus food craving is hypothesised to be an important intervening causal variable, on a causal pathway between responsivity to environmental cues (which may be of genetic or learned behaviour aetiology) and the development of obesity.

Methods

Sample size

As the aim of the current study was to test the individual predictors within a regression model, a sample size of $104 + k$ was estimated, where k is the number of predictors (Green, 1991). In this study, the influences of BMI and gender, along with the DEBQ-measured restraint, external and emotional eating as predictors were explored; thus our sample size was sufficient when considered as a whole. It is clear that this is an oversimplification, as the sample size required will depend on the effect size. However, since this was an exploratory study, the effect size was not predictable from previous studies.

Participants

One hundred and sixty-seven volunteers from Oxford Brookes University and the wider community were recruited through advertisements placed around the University and in community newspapers and bulletins. Thirty-nine volunteers were excluded from the study due to ill health, clinically abnormal glucose metabolism, lack of compliance in filling out questionnaires, or participation in a current weight loss programme; a further four female participants were later excluded due to recent major changes in physical activity levels. Thus, 124 participants, mean age 44.0 (SD 14.4) years, mean BMI 24.7 (SD 4.3) kg/m^2, completed the study.