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Reward sensitivity and food addiction in women

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Abstract

Sensitivity to the rewarding properties of appetitive substances has long been implicated in excessive consumption of palatable foods and drugs of abuse. Previous research focusing on individual differences in reward responsiveness has found heightened trait reward sensitivity to be associated with binge-eating, hazardous drinking, and illicit substance use. Food addiction has been proposed as an extreme form of compulsive-overeating and has been associated with genetic markers of heightened reward responsiveness. However, little research has explicitly examined the association between reward sensitivity and food addiction. Further, the processes by which individual differences in this trait and excessive over-consumption has not been determined. A total of 374 women from the community completed an online questionnaire assessing reward sensitivity, food addiction, emotional, externally-driven, and hedonic eating. High reward sensitivity was significantly associated with greater food addiction symptoms (r = .31). Bootstrapped tests of indirect effects found the relationship between reward sensitivity and food addiction symptom count to be uniquely mediated by binge-eating, emotional eating, and hedonic eating (notably, food availability). These indirect effects held even when controlling for BMI, anxiety, depression, and trait impulsivity. This study further supports the argument that high levels of reward sensitivity may offer a trait marker of vulnerability to excessive over-eating, beyond negative affect and impulse-control deficits. That the hedonic properties of food (especially food availability), emotional, and binge-eating behavior act as unique mediators suggest that interventions for reward-sensitive women presenting with food addiction may benefit from targeting food availability in addition to management of negative affect.

Keywords: Food Addiction; reward sensitivity; personality; hedonic eating; Reinforcement Sensitivity Theory

In recent years, there has been growing interest in the 'addictive' qualities of high caloric foods. In a series of empirical and review papers, Davis and colleagues have convincingly argued that overeating in today's "obesogenic environment" falls along a spectrum of eating behavior that ranges from "passive overeating" to binge-eating disorder, and at the most extreme level, to food addiction (Carlier, Marshe, Cmorejova, Davis, & Muller, 2015; Davis, 2013a, 2013b). Food addiction is characterized by the excessive overeating of high calorie food accompanied by loss of control and intense food cravings (Gearhardt, Corbin, & Brownell, 2009). The impact of the concept in the area of addiction and eating is further supported by a 9-fold increase in the number of journal articles referring to food addiction from 2006 to 2010 (Gearhardt, Davis, Kushner, & Brownell, 2011). Following from these comprehensive reviews, there is a current call "to think more mechanistically in the evaluation of food addiction by examining the contribution of biological, psychological, and behavioral circuits implicated in addiction to problematic eating behaviors." (Meule & Gearhardt, 2014, p. 3665). To that end we investigate a biologically-based trait of reward sensitivity that has been used to better understand individual differences in the vulnerability to addiction.

Reward sensitivity - general approach motivation

Beyond the role of basic metabolic processes, there is growing evidence that psychological factors and brain chemistry regulate eating behavior. A burgeoning avenue of enquiry in this area has focused on a personality trait referred to as Reward Sensitivity (Gray & McNaughton, 2000). Reward sensitivity is a biologically-based, normally-distributed, predisposition to seek out rewarding substances and to experience enjoyment in situations with high reward potential. Reward sensitivity is proposed as the expression of an underlying

Behavioural Approach System (BAS); the mesolimbic dopamine "reward" pathways have been proposed as the key biological basis of this trait (Gray & McNaughton). Both highly palatable foods and potent drugs of abuse have long been known to activate the dopaminergic "reward pathways" of the mid-brain, and are clearly implicated in the pursuit of natural (and now, quite unnatural) rewards in the environment (Davis, 2013a). A core theme of recent research has been the proposal that highly reward-sensitive individuals are more attuned to the rewarding properties to the reinforcing properties of drugs of abuse and high fat/high sugary "tasty" food (Dawe & Loxton, 2004; Hennegan, Loxton, & Mattar, 2013). Indeed, there has been a rapidly increasing body of evidence supporting the association between reward sensitivity and a range of addictive behaviors including alcohol abuse and ilicit drug use (Bijttebier, Beck, Claes, & Vandereycken, 2009; Dawe et al., 2007; Smillie, Loxton, & Avery, 2011). Heightened reward sensitivity has also been consistently associated with binge-eating, a motivated approach response towards dessert images, having a preference for foods high in fat and sugar, and a preference for colorful and varied food (Davis et al., 2007; Guerrieri, Nederkoorn, & Jansen, 2007; Loxton & Dawe, 2006; May, Juergensen, & Demaree, 2016; Schag, Schonleber, Teufel, Zipfel, & Giel, 2013). Activation of the reward pathways to images of food correlates strongly with self-report measures of reward sensitivity (Beaver et al., 2006). As such, heightened responsiveness to the rewarding properties of highly palatable foods and drugs of abuse has been proposed as a common factor to over-eating and the abuse of other substances (e.g., Loxton & Dawe, 2001; Loxton & Dawe, 2006).

Food Addiction and Reward Responsiveness

Food addiction or addictive-like eating has been operationalised in recent years by the Yale Food Addiction Scale (YFAS) – a 25 item measure based on the diagnostic criteria for substance dependence (Gearhardt et al., 2009). This scale, which assesses tolerance,

withdrawal, loss of control over eating, inability to stop eating, and negative impact on social and occupational function, derives both a symptom count score (0 to 7) and a diagnosis (meet 3 or more criteria and clinical impairment). Both symptom count score and diagnostic status classification have been used in research examining the validity, prevalence, and correlates of food addiction (e.g., Davis et al., 2011; Davis & Loxton, 2014; Davis et al., 2013). Although controversial, there is growing support for addictive-like eating behavior as assessed by the YFAS (e.g., Carlier et al., 2015; Schulte, Joyner, Potenza, Grilo, & Gearhardt, 2015).

Differences in the responsiveness of the "reward" circuits of the mid-brain in the vulnerability to food addiction have been supported by studies using fMRI and genetics. Gearhardt, Yokum, et al. (2011) found the activation of brain regions involved in the expectation of reward and attention and planning of food reward (when anticipating the receipt of a chocolate milkshake) to be associated with food addiction symptom scores. Taking a different approach, Davis et al. (2013) found a quantitative multilocus genetic profile score, based on six polymorphisms related to elevated dopamine function (Nikolova, Ferrell, Manuck, & Hariri, 2011), was positively associated with food addiction. This same profile score was associated with a number of addictive behaviors (Davis & Loxton, 2013). Using a computer task (Go/No-Go task), Meule, Lutz, Vogele, & Kubler (2012) found college women with high food addiction symptom scores responded more quickly (pressed a computer key) to high calorie food pictures than those with low scores. Together, such studies suggest greater reward responsiveness are involved in food addiction.

Mediators of reward responsiveness and food addiction

In a previous study we found the association between genetic vulnerability and food addiction to be mediated by binge-eating and food cravings (Davis et al., 2013). A composite “hedonic responsiveness” (hedonic eating, food cravings, and a preference for high fat/sugary

foods) was found to mediate the association between a genetic variant linked with opioid (pleasure) signaling and food addiction symptom scores (Davis & Loxton, 2014). We have also found self-reported reward sensitivity to be associated with greater attention to food stimuli, and a greater desire to eat when presented with food images (Hennegan et al., 2013). Thus, potential mediators include an attraction to the hedonic properties of food, and a tendency to notice and respond to food cues in the environment.

Hedonic eating

A key component of reward sensitivity is noticing and seeking out of appetitive substances (Corr, 2008). While reward sensitivity is underpinned by a system involved in seeking out appetitive substances more generally, hedonic eating refers to noticing and seeking of food specifically. As such, hedonic eating is potentially a food-specific form of reward-driven outcomes. Lowe et al. (2009) developed a scale to assess the motivation of individuals to consume food beyond homeostatic need; i.e., hedonic eating. The Power of Food Scale (PFS) assesses three aspect of hedonic eating based on proximity of food, 1) food available but not present, 2) food present but not tasted, and 3) food tasted but not consumed. The scale assesses the desire for food rather than the response to the consumption of food (as would be captured by binge-eating measures). Thus, we would anticipate that reward sensitivity and hedonic eating aspects would be positively associated, with reward sensitivity being an enduring trait and hedonic eating a specific arena in which this desire for appetitive substances is played out. In two previous studies, we found hedonic eating to be associated with food addiction (Davis & Loxton, 2014; Davis et al., 2013). However, in these studies we used the total PFS score. In the current study we were interested in the subscale scores (each with increasing proximity to food) as Gray and McNaughton (2000) argue that those high in reward sensitivity will notice and approach appetitive substances. However, reward sensitivity

is not associated with pleasure when consuming the substance (Corr, 2008). Using the PFS subscale scores may provide greater insight into the specific aspects of hedonic eating associated with reward sensitivity and food addiction.

External and Emotional eating

Smells and images associated with tasty foods (e.g., the smell of hot chips, pictures of chocolate cake) activate the reward pathways even more strongly than the consumption of food itself and have been linked with eating when otherwise sated (Cappelleri, Bushmakin, Gerber, Leidy, Sexton, Lowe, et al., 2009; Schultz, 1998). Individuals high in reward sensitivity show stronger associations (e.g., believe that eating is a good way to celebrate) and external eating (eating in response to external food cues) than less reward-sensitive individuals (Hennegan et al., 2013). The association with food addiction is mixed - external eating was associated with food addiction diagnostic status in one sample of obese individuals (Pepino, Stein, Eagon, & Klein, 2014) but not in another (Davis et al., 2011). Relatedly, emotional eating reflects the tendency to eat in order to assuage negative emotional states. While the association tends to be weaker than with external eating, emotional eating was associated to reward sensitivity (Davis et al., 2007; Hennegan et al., 2013) and more recently with food addiction (Davis et al., 2011; Pepino et al., 2014). Thus, we test external eating and emotional eating as additional mediators of reward sensitivity and food addiction.

Binge eating

Binge-eating has also been implicated in the progression from a preference for palatable foods to food addiction. For instance, in a sample of 72 obese adults, Davis et al. (2011) found 25% met criteria for food addiction. Seventy percent of those who met criteria for food addiction, also met criteria for Binge Eating Disorder, leading some to suggest that food addiction is simply another term for Binge Eating Disorder (see Davis et al. 2013, for a

review of this issue). However, while there was considerable overlap, half of the participants who met criteria for BED did not meet criteria for food addiction. A recent systematic review found reward sensitivity played a key role in binge-eating disorder in obese samples (Schag et al., 2013). Davis et al. (2013) has argued that binge-eating is a eating-related sub-phenotype that plays a role in mediating high reward responsiveness and food addiction. This was supported by binge-eating mediating the association between a multilocus genetic profile of reward responsiveness and food addiction diagnosis (Davis et al. 2013). However, to our knowledge this indirect effect of binge-eating has not been tested when investigating reward sensitivity.

Aims of the study

The present study aims to extend the research investigating the association between individual differences in reward sensitivity and food addiction via binge-eating, hedonic, emotional, and externally-driven eating. We used an online survey to collect data from a large sample of women from the community to test the model shown in Figure 1. Only women were recruited in keeping with previous research investigating reward sensitivity and eating behavior (Hennegan et al., 2013; Loxton & Dawe, 2001; Loxton & Dawe, 2006). It was hypothesized that 1) higher levels of reward sensitivity would be associated with more food addiction symptoms, 2) the association between reward sensitivity and food addiction would be mediated via a) hedonic eating, b) external eating, c) emotional eating, and d) binge-eating. Given previous research that food addiction has been associated with body mass, negative affect, and trait impulsivity (Davis et al., 2011), we also tested whether the proposed model continued to be supported when also controlling for these variables.

Method

Participants

A total of 382 women completed the online survey as part of a study investigating food addiction, over-eating and reward sensitivity in women. Following the deletion of women with substantial missing data or identified as multivariate outliers, 374 participants were included in the subsequent analyses. Ninety-five percent were Caucasian, with the remainder Asian, Indigenous Australian, or other ethnicity. Mean age was 30.58 years (*SD* = 12.70, range 17-70 with 70% aged under 32 years). Body mass was in the normal range (*M* = 24.00, *SD* = 5.95).

Procedure

The questionnaires were administered online using Qualtrics (www.qualtrics.com: Qualtrics Labs Inc., Provo, UT). Participants were recruited from undergraduate Psychology students and via advertisements on social media. Psychology students were given course credit for participation. The questionnaire took approximately 30-40 minutes to complete. Following completion, participants were given the option of leaving their email address on a separate secure webpage should they wish to be contacted with the results of the study and if they were interested in completing a subsequent study. Ethics clearance was obtained through the University's Behavioural and Social Sciences Ethical Review Committee.

Measures

The Sensitivity to Reward Scale (SR; Torrubia, Avila, Molto, & Caseras, 2001) was used to assess reward sensitivity. The SR scale consists of 24 dichotomously-scored items and includes situations in which individuals may strive for reward (e.g., "Does the prospect of obtaining money motivate you strongly to do some things?"). Positively endorsed scores are summed to create a total score. Internal consistency for the scale was .80.

The Power of Food Scale (PFS; Lowe et al., 2009) was used to assess hedonic eating. This 15-item questionnaire differentiates between motivations and drive to obtain food from the tendency to over-eat. All questions are answered on a 5-point Likert scale ranging from 1 (Strongly Disagree) to 5 (Strongly Agree). A total mean score represents a greater responsiveness to the food environment. Three subscale scores can be derived: 1) Food availability, e.g., "It seems like I have food on my mind a lot", 2) Food Present, e.g., " If I see or smell a food I like, I get a powerful urge to have some.", and 3) Food tasted, e.g., "Just before I taste a favorite food, I feel intense anticipation". Cronbach's alphas in the current study were (total = .82; Food available = .89; Food present = .88; Food Tasted = .82). Mean scores for the three subscales (Food available = 2.03; Food present = 2.63; Food Tasted = 2.48) were higher than that found in Cappelleri, Bushmakin, Gerber, Leidy, Sexton, Karlsson, et al. (2009) web-based survey of non-obese participants, although the mean total score (2.33) was similar to Lowe (2009).

The Dutch Eating Behavior Questionnaire (DEBQ; Van Strien, Frijters, Bergers, & Defares, 1986) was used to assess external and emotional eating. The external eating subscale consists of 10 items using a 5-point Likert scale from 1 (never) to 5 (very often). The scale is a measure of disinhibited eating triggered by external cues such as taste, smell and others behavior (e.g., ''If you see or smell something delicious, do you have a desire to eat it?''). The emotional eating scale consists of 13 items and is a good measure of eating cued by emotional events (e.g., "Do you have a desire to eat when you are feeling lonely?"). Mean scores were used to assess responsiveness to external food cues and using food to manage negative emotions. Cronbach's alphas in the current study were .85 for external eating and .96 for emotional eating.

The Binge Eating Questionnaire (BEQ; Halmi, Falk, & Schwartz, 1981). The five items of the BEQ that assess binge eating (rather than purging) were used in the current study. This was done to help better capture the study's goals of measuring eating behavior. Example items include, "Are there times when you are afraid you cannot stop voluntarily eating. Cronbach's alpha in the current study was .76.

Yale Food Addiction Scale (YFAS; Gearhardt et al., 2009). The 25-item YFAS was used to assess food addiction symptoms. Similar to the DSM-IV substance-dependence criteria, a diagnosis of food addiction can be given if the respondent experiences three or more symptoms over the past year, and if the "clinically significant impairment" criterion is met. A continuous, symptom count score is obtained by summing the number of symptoms endorsed, and can range from 0 to 7. Kuder-Richardson test of internal reliability in the current study was .83. Using the diagnostic scoring, 5.5% of the sample (n = 20) met criteria for food addiction, which is lower than that typically found in normal weight samples (Pursey, Stanwell, Gearhardt, Collins, & Burrows, 2014). However, the mean (1.56) was similar to the mean found in non-clinical populations (1.70, Pursey et al., 2014).

Covariates. Depressed mood, stress, and anxiety are frequently associated with eating problems, including food addiction (e.g., Davis et al 2011), and thus were assessed as possible covariates. The 21-item, Depression, Anxiety and Stress Scale (DASS; Lovibond & Lovibond, 1995) includes a depression scale, an anxiety scale, and a stress scale. Higher scores reflect higher levels of psychological distress and is well established for use in research. The internal reliability of the scales in the present study were: Depression = .87, Anxiety = .74, Stress = .86.

While reward sensitivity has previously been referred to as "impulsivity" there is consensus that reward sensitivity is conceptually and neurologically distinct from impulsivity

as typically conceptualized (e.g., Dawe & Loxton, 2004). However, given there is some overlap between these traits (typically correlating .3), we also assessed "trait impulsivity" as a potential covariate. The total score of the 30-item Barratt Impulsiveness Scale (BIS-11; Patton, Stanford, & Barratt, 1995) was used to measure “trait impulsivity”. Alpha for this scale was .82.

Analysis plan

Associations between reward sensitivity, food addiction, binge-eating, hedonic eating, external eating, and emotional eating were first tested using bivariate correlations. To test binge-eating, external eating, and hedonic eating as mediators of reward sensitivity and food addiction, a multiple mediation model was conducted according to procedures described by Hayes (2013). Binge-eating, hedonic subscales, emotional and external eating were entered as mediators as shown in Figure 1. Bias-corrected bootstrap confidence intervals (n = 10000, confidence intervals set at 95%) were used to assess the significance of the indirect effects. An advantage of the bootstrapping approach relevant to the current study is that the assumption of normality is not required. The SPSS "PROCESS" macro, model 4, v2.16 (Hayes, 2013) was used to test the significance of the overall indirect effects. The absence of zero within the confidence intervals suggests a significant indirect effect. This approach provides an estimate of the overall indirect effect of the mediators as a group (analogous to R in multiple regression) as well as estimates of each mediator (controlling for the other mediators; analogous to b weights in multiple regression, e.g., in Figure 1 the product of a1 and b1 is the specific indirect effect of reward sensitivity on food addiction via binge-eating, controlling for the other mediators).

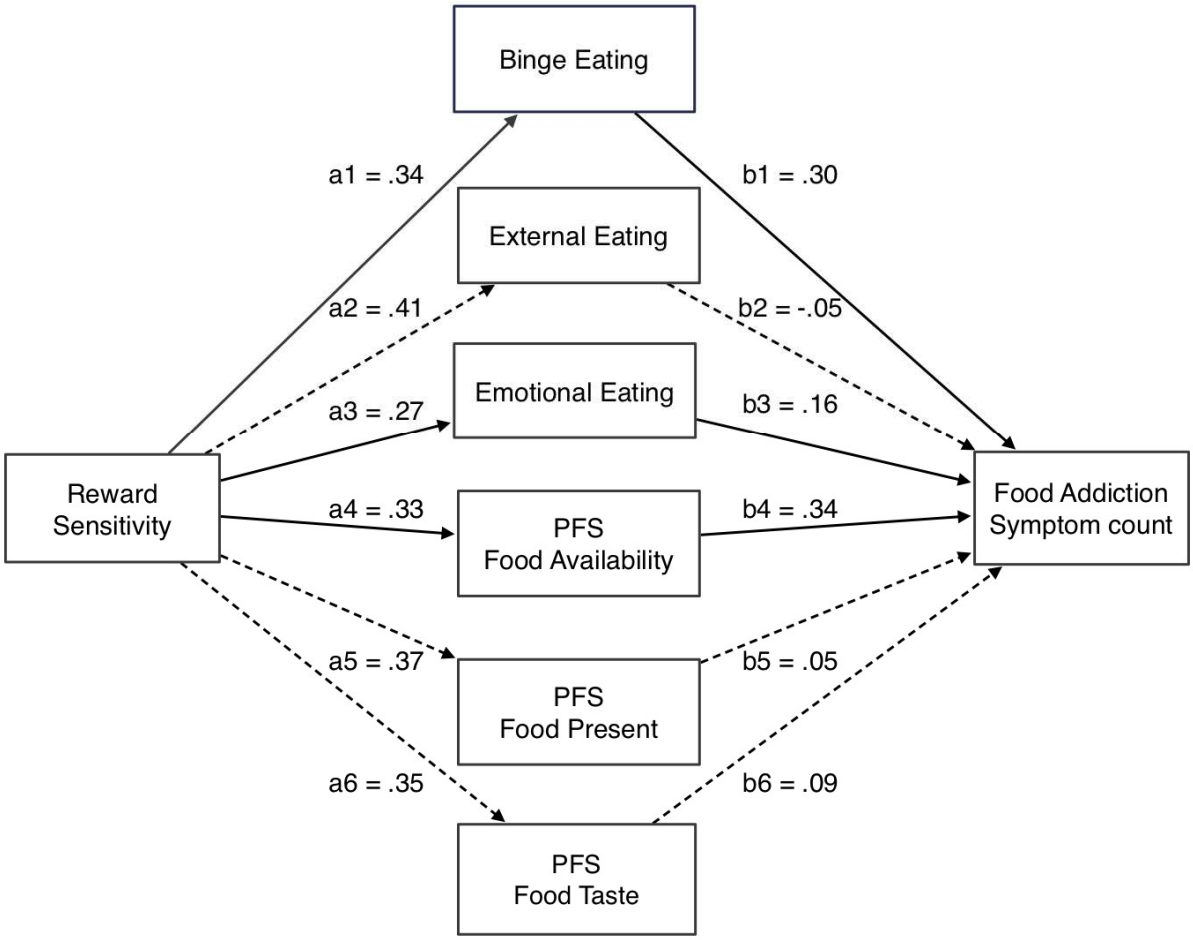

Figure 1. Indirect effects of reward sensitivity and YFAS symptom count via binge-eating, external eating, emotional eating, and hedonic eating.

Note. All values are standardized regression coefficients. Each 'a' path is the effect of reward sensitivity on the mediating variables. The 'b' paths represent the associations between the mediating variables and YFAS symptom score. Solid lines represent significant indirect effects. Dashed lines represent non-significant indirect effects.

Results

Descriptives

While there was positive skew in all the eating variables (as expected in a community sample) this is accounted for in the bootstrapped tests and thus were not transformed. Descriptive statistics and correlations between all variables are shown in Table 1. Reward sensitivity was significantly associated with food addiction, binge-eating, emotional eating, external eating, and hedonic eating subscales. The correlations between the PFS subscales and the DEBQ external eating scale were of a similar magnitude to that found in Lowe et al (2009). Reward sensitivity was moderately correlated with the total PFS score (r = .38). YFAS scores were significantly associated with age (r = -.12), BMI (r = .20), trait impulsivity (r = .21), anxiety (r = .34), depression (r = .34), and stress (r = .37). As such we tested the mediation model without and without these covariates.

Tests of Indirect Effects on YFAS symptom scores

As shown in Figure 1, binge-eating, emotional eating, externally-driven eating, and hedonic eating subscales were entered as parallel mediators. Table 2 provides the total and specific indirect effects when using the YFAS symptom scores. The overall total indirect effect of reward sensitivity and food addiction via the mediating variables (i.e., the indirect effect via the six mediators combined) was significant. However, when controlling for the shared variance between the mediators (i.e., the specific indirect effects), only the binge-items of the BEQ, the DEBQ Emotional Eating subscale, and the “Food Availability” subscale of the PFS were significant. There was no difference in the magnitude of the significant indirect effects. The overall model (reward sensitivity, hedonic eating subscales, binge-eating, emotional and external eating) accounted for over 48% of the variance in food addiction symptom count. See Figure 1 for standardized coefficients. When using the total PFS score rather than the three subscale scores in the model, there was a significant indirect effect of

reward sensitivity and YFAS symptom count via overall hedonic eating, controlling for binge-eating, external, and emotional eating (unique indirect effect = .05; SE = .01; 95CI = .03; .07).

Covariates

To assess whether the associations between reward sensitivity, YFAS, and the mediating variables were due to shared variance in negative affect (i.e., depression, anxiety, stress), trait impulsivity, age, or weight, a subsequent model was tested in which DASS depression, anxiety, and stress, BIS-11, age, and BMI, were included as covariates. There was virtually no change to any coefficients and the indirect effects via binge-eating, emotional eating and PFS food availability remained significantly different from zero.

Ancillary Tests of Indirect Effects using YFAS diagnosis scores

Although there were relatively few participants who met diagnostic criteria for food addiction (n = 20) we ran ancillary analyses to assess whether the same pattern of results was found for the association between reward sensitivity and YFAS diagnosis status as the outcome variable. Reward sensitivity was significantly higher in the YFAS diagnosis group (M = 12.30) than the no YFAS diagnosis group (M = 8.36; t[365] = 4.10, p < .001). In the first model with the PFS subscales, binge-eating, external eating, and emotional eating as the mediators, the overall total indirect effect of reward sensitivity and food addiction via these variables was still significant (indirect effect = .18, SE = .10, 95CI: .07; .30). However, when controlling for the shared variance between the mediators only the binge-eating showed a significant unique indirect effect (95CI: .02; .26). Unlike in the previous analysis, there was no significant effect via emotional eating (95CI: -.10 ; .09). The indirect effect via PFS Food Availability subscale (95CI: -.02; .20) also dropped to non-significance. The external eating scale and other PFS subscales remained non-significant. In a second model using the total PFS

341 score instead of the subscales, there was a significant indirect effect via hedonic eating (95CI:

342 .03; .21) as well as via binge-eating (95CI: .04; .24).

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Table 1

Descriptive statistics and correlations among the variables

	M	*SD*	1	2	3	4	5	6	7
1. Reward Sensitivity	8.58	4.26	-						
2. Binge Eating	1.23	1.43	.33***	-					
3. External Eating	3.06	.60	.41***	.35***	-				
4. Emotional eating	2.54	.95	.27***	.52***	.53***	-			
5. PFS: Food Available	2.03	.95	.33***	.61***	.57***	.63***	-		
6. PFS: Food present	2.63	1.02	.37***	.49***	.72***	.52***	.74***	-	
7. PFS: Food tasted	2.48	.92	.35***	.35***	.54***	.32***	.66***	.69***	-
8. Food Addiction Symptoms	1.56	1.34	.31***	.56***	.37***	.49***	.61***	.50***	.42***

Note. PFS = Power of Food Scale. *** $p < .001$

Table 2

Unstandardized Indirect effects of reward sensitivity and food addiction symptom scores via binge eating, external eating, emotional eating, and hedonic eating subscales

	Bootstrap estimate	SE	BC 95% CI lower	BC 95% CI upper
Binge eating	.028*	.007	.015	.044
External eating	-.013	.008	-.031	.002
Emotional eating	.014*	.006	.005	.027
PFS: Food Available	.032*	.010	.016	.055
PFS: Food Present	.006	.008	-.009	.023
PFS: Food Tasted	.008	.006	-.003	.021
Total Indirect effect	.076*	.013	.051	.102

Note. PFS = Power of Food Scale. Based on 10000 bootstrap samples. BC = bias corrected; CI = Confidence Interval,

* Indirect effect is significantly different from zero. Unstandardized indirect effect reported.

Discussion

The results of the study supported the hypothesis that reward sensitivity was associated with greater food addiction symptoms. Further, tests of indirect effects found the relationship between reward sensitivity and food addiction to be uniquely mediated by binge-eating, emotional eating, and hedonic eating (notably, food availability). These indirect effects held even when controlling for BMI, anxiety, stress, depression, and trait impulsivity. When using YFAS diagnostic status as the outcome, binge-eating and hedonic eating (as a total score) mediated the association between reward sensitivity and food addiction.

The association between reward sensitivity and food addiction symptom scores, and the higher reward sensitivity score in those meeting food addiction diagnosis is in accord with research showing an association between food addiction and a genetic profile linked to reward responsiveness (Davis et al. 2013). Reward sensitivity has also been consistently found to be associated with overeating (Bijttebier et al., 2009) and mid-brain responsiveness to appetitive food cues (Beaver et al., 2006). This association, however, is somewhat at odds with two previous studies that have found minimal association between YFAS scores and reward sensitivity (Clark & Saules, 2013; Gearhardt et al., 2009). This may be due to differences in the measures used to assess reward sensitivity. Both earlier studies used the total BAS scale score from the Carver and White (1994) BIS/BAS scale, whereas in this study we used the Torrubia et al. (2001) Sensitivity to Reward Scale. The BIS/BAS scale consists of a single Behavioural Inhibition System (BIS) scale (a measure of punishment sensitivity) and three BAS scales (fun-seeking, drive, reward responsiveness). Confirmatory factor analyses have consistently supported the use of separate subscale scores, rather than a total BAS score (e.g., Heubeck, Wilkinson, & Cologon, 1998; Jorm et al., 1999). More importantly, the BAS subscales also tend to correlate differentially with over-eating, hazardous drinking, and illicit drug use (Loxton & Dawe, 2001; Loxton et al., 2008; May et al., 2016; Voigt et al., 2009). For example, Loxton and Dawe (2001) found only two of these subscales (fun-seeking and drive) to be associated with hazardous drinking and only one subscale (fun-seeking) to be associated with dysfunctional eating. Voigt et al. similarly found the fun-seeking scale to be associated with greater alcohol and drug use, and the reward responsiveness scale to be associated *lesser* alcohol and drug use. Using the total BAS score may therefore miss significant associations with specific subscales. Future research may benefit from using measures that include BAS subscales to compare results.

A recent analysis of current measures of reward sensitivity found that a (short version) of the Sensitivity to Reward Scale captures trait impulsivity as well as reward sensitivity (Krupić, Corr, Ručević, Križanić, & Gračanin, 2016). As such, the associations we find between the Sensitivity to Reward Scale and YFAS may reflect both reward sensitivity and trait impulsivity. However, even when we controlled for trait impulsivity, the model still held suggesting that impulsivity alone does not account for the association found in the current study. Nevertheless, in future studies alternative measures of reward sensitivity (e.g., Corr & Cooper, 2016) may assist in better understanding the association of reward sensitivity and food addiction.

This is the first study to examine the association between reward sensitivity and the subscales of the Power of Food scale (Davis et al., 2011; 2013). Reward sensitivity was moderately associated with all three subscales and the total score. While the indirect effect via hedonic eating was supported using the total score, when using the subscale scores only the "food available" subscale showed a significant unique indirect effect. This subscale assesses the tendency to be aware of and drawn towards food that could be obtained but is not currently present. The use of the multiple mediation approach is similar to the use of multiple regression whereby there was a unique indirect effect of "food availability" when controlling for the other mediators. This adds to the literature on hedonic eating and food addiction with the more distal component (i.e., being aware of the availability of food) playing a unique factor in food addiction symptoms in generally normal weight women. Given this is the only study to explicitly examine the PFS subscale, these findings need replication.

In an earlier study we found reward sensitivity to be associated with external and emotional eating (Hennegan et al., 2013). In that study the association between external eating, but not, emotional eating, was mediated via the expectations that eating is rewarding.

In the current study, reward sensitivity was again associated with both external eating and emotional eating. However, in this study only emotional eating showed a significant unique indirect effect when using the YFAS symptom count score. The indirect effect was non-significant when using diagnostic status. This reflects a previous study (Davis, et al., 2013) where emotional eating did not show a unique indirect effect of a genetic profile score of dopamine responsiveness and YFAS diagnosis. The difference in the finding that emotional eating was associated with YFAS symptom count, but not YFAS diagnostic status may reflect lower power when using the categorical clinical score relative to the continuous symptom count - in both studies, the number of participants meeting diagnostic criteria was small (20 in the current study, 21 in Davis et al.). Alternatively, emotional eating may be associated with subclinical levels of addictive-like eating, but not in the development of clinically severe levels of food addiction. To tease out these differences requires samples with larger numbers of participants with clinical significant food addiction.

The association between external eating and food addiction has been mixed, with one study of obese individuals finding no difference in external eating between those meeting diagnostic criteria for food addiction and those that did not (Davis et al., 2011), while another sample of obese patients undergoing bariatric surgery has found a difference (Pepino, et al., 2014). In this study, there was an association between external eating and food addiction symptoms. However, this became non-significant when controlling for the other eating variables.

As previously found, reward sensitivity was associated with a measure of binge-eating (Bijttebier et al 2009). Binge-eating was again supported as a mediator of an index of reward responsiveness and food addiction. The current study adds further support to Davis's (2013a) contention that "food addiction is a reward-responsive phenotype of obesity" and proposal of

"a reward-based process model whereby an inherent biological susceptibility contributes to increased risk for overeating, which in turn may promote addictive tendencies toward certain highly palatable foods" (p. 173). We extend this proposal by explicitly linking a biologically-based personality trait as a phenotypic risk factor for binge-eating and hedonic-eating; eating-related behaviors that may lead to food addiction (and potentially obesity).

Limitations

We note that this is the first study to find an association between reward sensitivity and food addiction. In other studies in which this trait has been measured there have been non-significant associations. While we have suggested that the difference may reflect the use of different measures of reward sensitivity, another possibility is that the association found in this study may be a spurious finding. However, in a number of other (unpublished) studies we have performed using similar samples and the same measure, we have consistently found associations of a similar magnitude. As noted, given the different measures of reward sensitivity are used in the study of addictive-like eating, future research should include additional scales to determine whether the association with food addiction is only found with this specific measure.

As with any cross-sectional study, causal effects cannot be established and prospective studies are required. This is critical in this area as there is evidence using animal models that a diet of hyper-palatable foods changes the reward pathways in the mid-brain - the very region underpinning individual differences in reward sensitivity. We also used an online survey that was promoted as a study of "health in women", which may have targeted participants with an interest in health more generally. We note that the prevalence of women who met criteria for food addiction was lower than that have found in other samples collected in Australia (e.g., Pursey, Collins, Stanwell, & Burrows, 2015). We also note that the study only used women

and so the associations found in this study may not generalise to men. However, we note that in our previous studies of a genetic index of reward responsiveness and food addiction that there were no apparent differences between men and women (Davis et al., 2013). Nevertheless, this is a significant limitation that would need to be addressed in future research examining reward responsiveness and addictive-like eating.

Conclusions

This study further supports the argument that high levels of reward sensitivity may offer a trait marker of vulnerability to excessive over-eating, beyond negative affect and impulse-control deficits. That the hedonic properties of food (especially food availability) and binge-eating behavior act as unique mediators suggest that interventions for reward-sensitive women presenting with food addiction may benefit from targeting food availability. There is growing evidence that public health interventions on obesity, such as provision of dietary guidelines, are largely ineffective, in part, due to the failure to account for individual differences in people's response to food availability and the promotion of unhealthy foods in the environment. Binge-eating behavior also plays a key role in the development and maintenance of food addiction symptoms. An impulsivity-focused treatment program has recently been proposed (Schag et al., 2015). Such personality-targeted interventions have had promising results in the reduction of binge-drinking and drug use in adolescents (e.g., Conrod, Castellanos, & Mackie, 2008). Given the clear links between food addiction and traditional addictions, such approaches may be effective with reward-driven over-eating.

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