Research report

From motivation to behaviour: A model of reward sensitivity, overeating, and food preferences in the risk profile for obesity

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Abstract

The reinforcing effects of addictive drugs and palatable foods are regulated, at least in part, by a common biological mechanism. The reactivity or sensitivity of these brain reward regions have been found to correlate significantly with the risk for a variety of drug addictions. Sensitivity to Reward (STR) is conceptualised as a psycho-biological personality trait rooted firmly in the availability of dopamine in the mesocorticolimbic (‘common reward’) pathways, and as such is a good candidate for studying motivational factors and eating behaviours. The purpose of the present study was to examine whether STR was related to behaviours that contribute to excess body weight. Structural equation modelling procedures were used with a sample of healthy adult women (n = 151). We hypothesised that STR would positively predict overeating and a preference for foods high in fat and sugar; and that these two behaviours would, in turn, predict a higher Body Mass Index. Results provided an excellent fit of the model to our data confirming our view that a personality trait like STR can only influence a physical condition like body weight indirectly by the way it co-varies with behaviours that contribute directly to variation in the outcome variable.

Keywords: Sensitivity to reward; Overeating; Food preferences; BMI

Introduction

The reinforcing effects of addictive drugs and palatable foods are regulated, at least in part, by a common brain mechanism depending crucially on the level of dopamine (DA) activation in mesocorticolimbic regions (e.g. Di Chiara et al., 2004; Risinger, Freeman, Rubinstein, Low, & Grandy, 2000). Indeed, the sub-cortical brain does not seem to differentiate among rewards provoked by natural reinforcers like food, illicit drugs like cocaine, or behaviours like gambling (Kelley, Schiltz, & Landry, 2005). For example, two recent studies found that obese women had lower rates of alcohol (Kleiner et al., 2004) and marijuana use (Warren, Frost-Pineda, & Gold, 2005) than their normal weight, age-matched counterparts, and that, in both cases, an inverse relationship existed between body mass index (BMI) and drug use. The authors concluded that overeating competes with pharmacologic agents for brain reward sites, and thereby may serve as a buffer for the use and abuse of other addictive behaviours. Complementary to this viewpoint is evidence that appetite suppression is a major pharmacological effect of chronic drug use (see Cochrane, Malcolm, & Brewerton, 1998).

Clearly many factors influence the kinds of pleasure we pursue in our daily lives. The context—through classical and operant conditioning processes—is particularly important in determining the choices we make and the frequency of their use (Corwin & Hajnal, 2005). In most Western societies, the convenience, the availability, and the
relatively low cost, make tasty foods a highly salient reward for many people. Aggressive marketing by the fast-food industry further enhances the temptation to overindulge.

In view of the many commonalities between food and drug reward, some have argued that chronic overeating can be modelled as an *addictive* behaviour similar to other substance-dependent disorders (e.g. Corwin, 2006; Davis, Strachan, & Berkson, 2004; James, Gold, & Liu, 2004; Wang, Volkow, Thanos, & Fowler, 2004). Consequently, there is a growing interest in examining the role of DA neurotransmission in the risk profile for obesity—especially how its variation in the population affects individual differences in vulnerability to overeating.

Undoubtedly there are many routes to obesity so it is reasonable to assume that weight-gaining individuals possess a variable set of biological and behavioural susceptibility factors (Blundell et al., 2005). On the one hand, there is evidence that *hypo*-dopaminergic functioning—what has been called a Reward Deficiency Syndrome (RDS)\(^1\)—underlies a range of addictions including alcoholism, cocaine abuse, and pathological gambling (see Bowirrat & Oscar-Berman, 2005). Some have recently argued that RDS is also a factor in the development of obesity (e.g. Wang et al., 2004). On the other hand, enhanced DA functioning, which is characterized by a heightened hedonic capacity and greater behavioural activation (e.g. Cohen, Young, Baek, Kessler, & Ranganath, 2005), fosters strong appetitive responses to the natural pleasures in life. For instance, amplification of the DA signal in human participants via a small dose of oral methylphenidate\(^2\) increased their desire to eat in response to a palatable food cue (Volkow et al., 2002). There is also evidence that obese individuals have enhanced sensitivity in brain areas associated with the sensory (e.g. lips, tongue, mouth) processing of food (Wang et al., 2002).

Regrettably, our current obesigenic environment can exploit those with a high sensitivity to reward (STR) by promoting consumption beyond caloric need. Non-homeostatic eating can take several forms including eating that is driven by emotional states (‘comfort’ eating) or by environmental cues such as the sight and smell of food stimuli. It is also characterised by frequent snacking and episodes of binge eating. In addition, hedonic processes—commonly regarded as the pleasure that is associated with food—are of central relevance to other aspects of eating. A primary target of such influence is an enhanced preference for foods that are fat and sweet since they typically provide a greater reinforcement value than bland food (Epstein & Leddy, 2006). In a recent study of high and low-fat phenotypes—those habitually consuming a diet containing >43% or <32% fat respectively—the former group comprised a significantly greater number of obese individuals (Blundell et al., 2005). However, among the high-fat group there was considerable variability in BMI, and results indicated that those who were prone to weight gain reported higher hedonic responsiveness to eating, and a greater intensity of pleasurable sensations from the taste of food. We predicted that those who are highly sensitive to reward would therefore also be more responsive (than their anhedonic counterparts) to the perceived palatability of sweet and fatty foods.

To date, only a handful of studies has investigated the relationship between reward sensitivity, eating behaviours, and body weight, and are all supportive of positive links (Davis et al., 2004; Franken & Muris, 2005; Loxton & Duwe, 2001). For example, Franken and Muris (2005) found that young women who were more sensitive to reward reported stronger food cravings and had a higher BMI. Likewise, Davis et al. (2004) found that anhedonic women were less likely to overeat, after controlling for depression in their regression model. However, as a body of work these studies are limited by the general use of small samples of primarily young, normal-weight women, by self-report instead of objective measures of body weight, and by single markers of overeating.

The present study expands on this research by using a more comprehensive set of measures to reflect the constructs of interest, and by testing a large sample of women who are more representative of the adult population in terms of BMI and (pre-menopausal) age. We used structural equation modelling (SEM) to test the prediction that sensitivity to reward is a phenotypic positive influence on weight gain—especially in an environment that proliferates with tempting and available foods—via its influence on overeating and food preferences (see Fig. 1). The first three constructs in the path diagram were modelled as multi-factorial latent variables. *Sensitivity to Reward, Overeating, Food Preferences,* and *Body Mass Index* in a sample of adult women.

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\(^1\)When there is sub-optimal functioning of the brain reward cascade, which could be caused by certain genetic variants, especially in the DA system, the brain of that person is likely to need a DA ‘fix’ to feel good and to improve the DA deficit (Blum & Braverman, 2001).

\(^2\)Methylphenidate (a.k.a. Ritalin) is a psychomotor stimulant that has a mechanism of action similar to cocaine. It binds to the DA transporter increasing neurotransmitter availability in the synapse.
Measures

Measures had a BMI of 18.4. and (BMI 1998), 38.5% of the sample were normal weight-status categories (World Health Organization, 14 mean BMI was 27.6 (SD 5.9). Based on accepted BMI weight-status categories (World Health Organization, 1998), 38.5% of the sample were normal weight (BMI >18.5 and <25); 27% were overweight (BMI >25 and <30); and 34% were obese (BMI >30). One subject had a BMI of 18.4.

Methods

Subjects

One hundred and fifty-one healthy pre-menopausal women between the ages of 25 and 50 years (mean = 33.5 years; SD = 7.1) took part in the study. They were solicited from posters placed at two university campuses, and at various hospital and community centres in the urban core of a large Canadian city, asking for volunteers to participate in a “health psychology study”. Participants were screened initially during a structured telephone interview and excluded if they had any serious medical condition, were underweight (based on their reported height and weight), were pregnant or had recently given birth, or were currently being treated for any psychiatric disorder including an eating disorder. They were also required to be fluent in English. Eighty-four percent of the participants were born in Canada, or an English speaking Western country like the United States or Britain. Their mean BMI was 27.6 (SD = 5.9). Based on accepted BMI weight-status categories (World Health Organization, 1998), 38.5% of the sample were normal weight (BMI >18.5 and <25); 27% were overweight (BMI >25 and <30); and 34% were obese (BMI >30). One subject had a BMI of 18.4.

Measures

1. Sensitivity to Reward was measured by 2 self-report questionnaires, both designed to assess individuals’ capacity for pleasure and reward—i.e. their degree of appetitive motivation and response to signals or reinforcement and/or non-punishment.

(i) The STR subscale of the SPSR Questionnaire (Torrubia, Avila, Molto, & Caseras, 2001) comprises 24 forced-choice items which assess the respondent’s approach responses under various conditions of reward such as physical sensations (“Is it easy for you to associate tastes and smells to very pleasant events?”). This scale has shown good internal consistency, temporal stability, and concurrent validity (e.g. Caseras, Avila, & Torrubia, 2003). The alpha coefficient in the present study was 0.77.

(ii) The Behavioural Activation (BAS) subscale of the BIS/BAS questionnaire (Carver & White, 1994) comprises 13 items which assess one’s persistent pursuit of desired goals, the desire for new and pleasing stimuli, and the positive anticipation of rewarding events in the future. In this study, we used the total score instead of the three subscales to decrease the number of parameters to be estimated (relative to the sample size) in the SEM, and because there was high internal consistency of the total score. The z coefficient was 0.88.

2. Overeating was assessed by three separate scales, each reflecting the tendency to overeat in response to certain triggers:

(i) The Dutch Eating behaviour Questionnaire [DEBQ] (Van Strien, Frijters, Bergers, & Defares, 1986) assesses three aspects of eating behaviour. In this study only the Emotional Eating subscale (e.g. the degree to which eating is prompted by emotional states like tension and worry rather than by hunger) and the External subscale (e.g. the degree to which one tends to overeat if food looks and smells good) were used. The Dietary Restraint subscale was not deemed a useful index of overeating given its focus on dieting and calorie restriction.

(ii) The Binge Eating Questionnaire [BEQ] (Halmi, Falk, & Schwartz, 1981) assesses the frequency and severity of symptoms associated with binge eating (such as loss of control over eating, and negative affect following a binge) and with purging (e.g. self-induced vomiting). Binge eating was quantified by summing the responses to 5 (yes–no) questions tapping aspects of the behaviour (e.g. “Are there times when you feel you cannot voluntarily stop eating?”).

3. Food Preferences were assessed by The Food Preference Questionnaire (Geiselman et al., 1998). This scale was designed as a 2 [FAT: high vs. low] × 3 [CARBOHYDRATE: high simple, high complex, low carbohydrate/ high protein] measure of preference for various kinds of macronutrients. It contains 72 common foods in each of the six cells listed in random order. Respondents indicate their preference of each on a 9-point Likert scale. A high-fat preference score is obtained by summing the 36 high-fat items and calculating the mean. The high-sugar preference score comprises the 24 high-sugar items. The authors report good reliability and validity of these measures, and the z coefficients for our sample were 0.96 and 0.87, respectively.

4. BMI (weight[kg]/height[m^2]) was calculated from height and weight measured with the participant wearing indoor clothing and standing in stocking feet.

Procedure

For those who passed the telephone screening interview a testing appointment was arranged at the closest of two
available research facilities. After giving signed informed consent, a more detailed screening interview took place to confirm eligibility criteria. The questionnaire package was then completed, and height and weight were measured. At the end of the study, each participant was paid a small stipend for her time and out-of-pocket expenses.

Results

The proposed model (see Fig. 1) was tested using SEM and Amos 6.0 software. SEM is a useful statistical procedure for researchers who want to test a theory involving causal processes, and therefore is well suited to the management of cross-sectional data for inferential purposes (Byrne, 2001). The kurtosis and skew of the eight variables in the model were between −1.04 and 0.50 and −0.53 and 0.96, respectively—values which are well within the acceptable range to proceed with SEM according to West, Finch, and Curran (1995).

In this study, STR was modelled as a latent variable with two measured variables. Overeating and Food Preferences were also latent variables comprising three and two measured variables, respectively. Prior to analysing the structural model, the measurement model was tested using exploratory factor analysis with Promax rotation. As expected, a clear three-factor solution emerged, and the correlations among factors were modest (0.27–0.39). It is noteworthy, however, that although the external eating variable had a strong loading on the Overeating factor, as predicted, it also had a modest relationship with the STR factor (0.27). This cross-loading is understandable since external eating reflects an appetitive response to food cues in one’s environment and STR describes one’s general capacity for pleasure, as well as the motivation to seek out reinforcing stimuli. A list of the factor loadings is shown in Table 1. Table 2 presents a bivariate correlation matrix of all the measured variables in the study, including BMI, and the sample means and standard deviations for these variables.

Table 1
Factor loadings for the measurement model

<table>
<thead>
<tr>
<th></th>
<th>Factor 3</th>
<th>Factor 2</th>
<th>Factor 1</th>
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<tbody>
<tr>
<td>Sensitivity to reward</td>
<td></td>
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<tr>
<td>Reward Sensitivity Scale (SPSRQ)</td>
<td>0.87</td>
<td>−0.01</td>
<td>−0.02</td>
</tr>
<tr>
<td>BAS scale (BIS/BAS)</td>
<td>0.88</td>
<td>−0.04</td>
<td>−0.02</td>
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<tr>
<td>Overeating</td>
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<tr>
<td>Binge eating</td>
<td>−0.02</td>
<td>0.85</td>
<td>−0.14</td>
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<tr>
<td>Emotional eating</td>
<td>−0.12</td>
<td>0.89</td>
<td>0.04</td>
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<tr>
<td>External eating</td>
<td>0.27</td>
<td>0.58</td>
<td>0.15</td>
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<tr>
<td>Food preferences</td>
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<tr>
<td>High fat preference</td>
<td>0.02</td>
<td>−0.01</td>
<td>0.95</td>
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<tr>
<td>High sugar preference</td>
<td>−0.05</td>
<td>−0.03</td>
<td>0.97</td>
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Table 2
Intercorrelations, means, and standard deviations for all measured variables

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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>Mean</th>
<th>SD</th>
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<tr>
<td>BMI</td>
<td>0</td>
<td>0.16*</td>
<td>0.28**</td>
<td>0.09</td>
<td>0.25**</td>
<td>0.18*</td>
<td>0.13</td>
<td>27.6</td>
<td>5.92</td>
<td></td>
</tr>
<tr>
<td>RS</td>
<td>0.50**</td>
<td>0.22**</td>
<td>0.39**</td>
<td>0.22**</td>
<td>0.27**</td>
<td>0.21**</td>
<td>10.85</td>
<td>4.25</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BAS</td>
<td>0.20*</td>
<td>0.36**</td>
<td>0.22**</td>
<td>0.26**</td>
<td>0.22**</td>
<td>39.56</td>
<td>5.58</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>EmEat</td>
<td>0.50**</td>
<td>0.49**</td>
<td>0.22**</td>
<td>0.20*</td>
<td>2.85</td>
<td>0.95</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ExEat</td>
<td>0.39**</td>
<td>0.37**</td>
<td>0.26**</td>
<td>3.22</td>
<td>0.57</td>
<td></td>
<td></td>
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<tr>
<td>Binging</td>
<td>0.11</td>
<td>0.11</td>
<td>1.38</td>
<td>1.58</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>High Fat</td>
<td>0.82</td>
<td>5.99</td>
<td>1.33</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>High Sugar</td>
<td>6.28</td>
<td>1.15</td>
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BMI = Body Mass Index. RS = Sensitivity to Reward subscale of the SPSR questionnaire. BAS = Behavioral Activation Scale of the BIS/BAS questionnaire. EmEat = Emotional Eating subscale of the DEBQ questionnaire. ExEat = External Eating subscale of the DEBQ questionnaire. Binging = scores on the BEQ. High Fat = High fat preference subscale of the Food Preference questionnaire. High Sugar = High sugar preference subscale of the Food Preference questionnaire. * = < 0.05. ** = < 0.01.
\( \chi^2 \) analysis tested the hypothesis that the relationships proposed in the model are a reasonable explanation of those existing in the data. We obtained a significant \( \chi^2 = 33.91, \text{df} = 17, p = 0.009 \). However, a non-significant \( \chi^2 \)—indicating a good absolute or overall model fit—is frequently not obtained. This may occur either because the model is slightly mis-specified or does not account for all the measurement error. Therefore, it is important to examine other indices that have been developed to assess the fit of the model. These values (e.g. the root-mean-square error [RMSEA] was 0.081 and should be <0.06; and the goodness-of-fit index [GFI] was 0.947 and should be >0.95), also suggested that the model was not a particularly good fit to the data.

**Model modification**

Each postulated modification to a model should be defensible primarily from a theoretical perspective (Boomsma, 2000; Kaplan, 1990). In other words, purely data-driven decisions are inappropriate in SEM. Given the theoretical association (as discussed above) and the modest cross-loading of the external eating indicator and the sensitivity to reward factor, a path between sensitivity to reward and external eating was freed. This simple modification reduced the \( \chi^2 \) by 13.81 and produced a non-significant value \( \chi^2 = 20.11, \text{df} = 16, p = 0.215 \) indicating a good absolute or overall model fit. We also confirmed the goodness-of-fit of the model by examining other indices developed for this purpose (see Boomsma, 2000; Byrne, 2001). Table 3 lists five commonly reported fit indices and the values considered representative of a good fit. The **Comparative Fit Index** evaluates the fit of the estimated model relative to the fit of the independent model (where no relationships are estimated between variables). The **Standardized Root Mean Squared Residual** is an index of the average differences between the sample variances and covariances and the estimated (model) variances and covariances. The **Root Mean Square Error of Approximation** is a popular measure that also takes into consideration the complexity of the model (i.e. the degrees of freedom). The **Goodness of Fit Index** is a measure of the proportion of variance and covariance that the proposed model is able to explain (similar to \( R^2 \) in regression). And finally, the **Adjusted Goodness of Fit Index** is the GFI adjusted for the degree of parsimony in the model. It can be seen from the values we obtained in the present analysis that in every case the observed value indicates a good fitting model.

Path coefficients assess the magnitude of the relationships among the latent and measured variables in the model. An examination of critical ratios\(^3\) (CR) for each coefficient indicated that all paths were statistically significant and the CR values ranged between 2.10 \( (p = 0.036) \) and 5.59 \( (p < 0.0001) \). **Fig. 2** shows the standardized regression weights for each path tested in the revised model.

**Discussion**

In a large sample of healthy adult women, we tested the theory that STR would predict (i) the tendency to eat beyond caloric need and in the absence of hunger and,

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\(^3\)The critical ratio is calculated by dividing the unstandardized estimate by its standard error.
Nowadays BMI values among the obese typically span a broad range from 30 to over 60, and the prevalence of morbid obesity is rising at twice the rate of milder forms (Freedman, Khan, Serdula, Galuska, & Dietz, 2002). Clearly it will behove future researchers to consider obesity as a heterogeneous set of conditions (defined largely by degree of severity) which are likely to differ in their clinical symptoms, their profile of risk factors, and their response to treatment strategies. Indeed, from the perspective of the present study, it may be that the relationships we found among STR, overeating, food preferences, and BMI are only positive in the range from normal weight to mildly obese, and that a different set (and direction) of relationships may exist in those with morbid obesity. For example, food may serve as a positive reinforcer for some individuals and as a negative reinforcer (i.e. a ‘self-medicating’ behaviour) for others, thereby influencing the frequency and magnitude of their overeating, and subsequent weight gain.

**Overeating**

The specified path between STR and overeating was strongly positive, supporting our earlier work (Davis et al., 2004) and that of other researchers (e.g. Loxton & Dawe, 2001)—albeit in the present study, using a more comprehensive set of markers for each construct. It is generally agreed that variation in reward sensitivity (and related constructs like behavioural activation and novelty/sensation seeking) is regulated by DA and influenced by the level of activation in reward-sensitive regions of the midbrain (Cohen et al., 2005; Depue & Collins, 1999; Evans et al., 2006). Therefore, appetitive responses to food should be enhanced in more reactive individuals—as we found—and contribute to a greater level of consumption and the tendency to overeat.

Our findings are, however, conceptually at variance with the RDS view of overeating and obesity. For example, Wang et al. (2001) found that obese individuals had a significant reduction in DA D2 receptor availability. Others have also found a higher prevalence of the Taq1A allele (thought to be linked with lower receptor levels) in obese individuals (e.g. Blum et al., 1996; Noble et al., 1994). One reason for this apparent contradiction may be the sample differences in BMI. The RDS studies have generally used morbidly obese subjects, typically recruited from obesity treatment clinics. For example, in the study by Wang et al. (2001), the obese adults all had a BMI > 40 (Class III obesity). By contrast, in the study by Franken and Muris (2005), which showed a positive link between STR, food cravings, and body weight, 83% of the sample were normal weight young women. Since none of the participants in the present study had a BMI > 40, it is important to emphasis that our results cannot extrapolate to women at the very high end of the weight range for example, when palatable foods are consumed trace amounts of endogenous opiates are released in the brain (e.g. Drewnowski, Krohn, Demitrack, Nairn, & Gosnell, 1992), which in turn, increase DA transmission in brain reward regions. The ‘pleasure’ experienced from this behaviour should therefore be enhanced in those with a greater sensitivity in brain reward regions. On the other hand, the social and psychological context is also of great importance in forming our food preferences. Since highly palatable foods are often associated with pleasurable social interactions and celebrations, hedonic individuals may be more likely to partake in, and enjoy, social gatherings, and therefore to form more positive conditioned responses to tasty food cues in their environment.

Finally, it is important to acknowledge that although the association between Food Preferences and BMI was statistically significant, it was weaker than the other relationships in the model. One explanation could be a relatively restricted range on the measured markers of this latent variable (viz. high fat and high sugar preference) because fat and sweet foods have a great universal appeal. The high means and small standard errors for these two scales confirm this possibility.

**Conclusions**

Personality can only influence a physical condition like body weight *indirectly* by the way it co-varies with behaviours that contribute *directly* to its variation in the
population. STR is a biologically-based trait rooted firmly in the reactivity of the mesocorticolimbic (‘common reward’) DA pathways and as such is a good candidate for study in the area of ingestive behaviours. As predicted, a high STR was related to overeating as well as an heightened preference for sweet and fatty foods; and these two factors were positively correlated with BMI. Future studies should extend this research to those with severe obesity, and should also test the model in a sample of adult men.

References


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