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How is reward sensitivity related to bodyweight in children?

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6 Sandra Verbeken[a*]

7 Caroline Braet[a]

8 Jan Lammertyn[b]

9 Lien Goossens[a]

10 Ellen Moens[a]

11 [a]*Ghent University, Ghent (Belgium)*

12

13 [a] Department of Developmental, Personality and Social Psychology, Ghent University,
14 H. Dunantlaan 2, 9000 Ghent, Belgium.

15 [b] Faculty of Psychology and Educational Sciences Ghent University, H. Dunantlaan 1,
16 9000 Ghent, Belgium.

17

18 *Corresponding author. Department of Developmental, Personality and Social
19 Psychology, Ghent University, H. Dunantlaan 2, 9000 Ghent, Belgium.

20 *E-mail address:* Sandra.Verbeken@UGent.be (S. Verbeken)

21 " not for publication": Tel.: +32 9646412. Fax: +32 9646499

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Abstract

Previous research assumes that there are two seemingly opposing hypotheses for the relation between reward sensitivity (RS) and bodyweight: hyper-responsiveness model and reward deficiency syndrome (RDS), leading to the proposition of a feed forward process of weight gain. High RS may contribute to overeating and weight-gain among normal weight individuals. Over time the excessive food-intake may evolve in a down-regulation of dopamine (RDS), resulting in overeating as a form of self-medication and the progression to obesity. This process was evidenced in adults showing a curvi-linear relationship between self-reported RS and BMI. The aim of the current study was to investigate the association between self-reported RS and BMI in children (10-15y). The results confirm the non-linear relationship between RS and body weight and support the suggestion of the same feed forward process in children. These findings imply that it is crucial to reduce the intake of high palatable foods in high RS children to prevent the decrease in RS and reduce the risk for future weight gain.

Keywords: Reward sensitivity, overweight, obesity, children, food intake, dopamine

The recent boom of childhood obesity challenges worldwide public health (Orsi, Hale, & Lynch, 2011). Besides the recognized genetic predisposition, the impact of the modern food-environment is well-established. A greater food palatability, a wide variety of foods, the high and easy availability (in the home and workplace), the stimulation by advertising, the food saliency, a larger portion size, and a higher energy density of food, all contribute to an increased reward value of foods, which overrides existing satiety signals and fosters overeating (Rolls, 2011). Consequently, in an increasing part of western society a positive energy balance is likely, which could lead to weight gain. Although, the fact that not everyone in the same high rewarding food-environment becomes overweight points also at the role of interacting individual factors. It is reasonable to propose that individual differences in reward sensitivity (RS) or the tendency to engage in motivated approach behavior in the presence of rewarding stimuli may be one of the factors that contribute to a vulnerability to overeat and become obese (Small, 2009).

Obese individuals find palatable foods more rewarding than non-obese (McGloin et al., 2002; Rissanen et al., 2002), but it remains unclear *why* this is so (Lowe et al., 2009). According to Gray's Reinforcement Sensitivity Theory (RST, Gray, 1994), RS reflects functional outcomes of the behavioral activation system (BAS), which is organized primarily by the neurotransmitter dopamine (DA) (Di Chiara, 1995; Pickering & Gray, 1999). Additionally, it has been postulated that dopamine deregulation contributes to the development of obesity and binge eating (Davis et al., 2008; Davis et al., 2009; Geiger et al., 2009; Mathes et al., 2010).The dual vulnerability theory of dopamine deregulation presents two opposing hypotheses (Davis et al., 2008; Stice, Spoor, Janet, & Zald, 2009). The first hypothesis, the hyper-responsiveness model, states that hypersensitivity to reward due to increased dopaminergic functioning, may motivate individuals to seek rewarding stimuli simply because the reinforcement value of the reward is so great (Davis, Strachan, & Berkson, 2004; Davis

et al., 2008; Dawe & Loxton, 2004). Alternatively, Reward Deficiency Syndrome (RDS), states that individuals with relative insensitivity to reward because of low dopaminergic functioning, seek more rewarding substances to increase endogenous dopamine levels and improve mood (Wang, Volkow, & Fowler, 2002; Volkow, Wang, Fowler, & Telang, 2008).

The **hyper-responsiveness model** was supported by experimental research in healthy volunteers. In a large group of adults who habitually consume a high-fat diet, Blundell et al. (2005) identified individual differences which make some people susceptible to weight gain and others resistant. The results clearly indicate that hedonic attraction to palatable foods and eating could significantly differentiate between individuals who gained weight and those who remained lean. This finding was further supported in children by Guerrieri, Nederkoorn, and Jansen (2008). They identified high and low RS-children based on their performance on a behavioural task and measured their caloric intake via a Bogus Taste Test. Interestingly, when varied food was offered, the high RS children ingested significantly more calories than their low RS counterparts. When monotonous food was offered, RS did not really affect caloric intake.

Similarly imaging research in adults found that RS as measured with the BIS/BAS self-report scale (Carver & White, 1994), significantly predicted activation to appetizing foods (relative to bland foods) in brain areas implicated in food reward (Beaver et al., 2006; Schienle, Schäfer, Hermann, & Vaitl, 2009). Additionally, fMRI data indicate that obese children and adolescents versus their lean counterparts showed greater activation in brain reward areas in response to visual food stimuli (Bruce et al., 2010; Batterink, Yokum, & Stice, 2010) and in response to food consumption (Stice, Spoor, Bohan, & Small, 2008). Especially relevant is the fact that it was previously shown in mice that activation of these brain areas produces overeating and increases the preference for foods high in fat and sugar (Kelley, 2004).

The assumed initial vulnerability may be a generalized hyper-responsiveness to various reward types as opposed to a specific deficit within the eating domain (Stice, Yokum, Burger, Epstein, & Small, 2011). Stice et al. (2011) found that adolescents at high-risk versus low-risk for future obesity by virtue of parental obesity not only showed greater activation in reward regions in response to palatable food, but also to monetary reward. Similarly, compared to lean individuals, obese children continue to play a rewarded computer game longer (Nederkoorn, Braet, Van Eijs, Tanghe, & Jansen, 2006; Verbeken, Braet, Claus, Nederkoorn, & Oosterlaan, 2009) and report higher generalized RS (Davis et al., 2004; Kane, Loxton, Staiger, & Dawe, 2003; Mobbs, Crépin, Thiéry, Golay, & Van der Linden, 2010). Furthermore, substantial longitudinal research indicates that children with higher generalized RS (measured with a self-regulation task) were more likely to be classified as overweight or obese several years later (Francis & Susman, 2009; Graziano, Calkins, Keane, 2009; Seeyave et al., 2010). However the paradigm used in these longitudinal studies provides a mixed measure of RS and inhibitory control, and therefore it is impossible to support the unique predictive value of RS.

The second hypothesis, here labeled as **Reward Deficiency Syndrome** (RDS) rests on the premise that palatable food can be used in the same manner as addictive drugs, and that risk for its overuse should therefore be greater among those at the anhedonic end of the RS continuum. In other words decreased dopamine activity reduces the sensitivity to natural rewards, and this deficit might temporally be compensated for by overeating (Davis et al., 2004). In obese children, imaging data indeed show a lower activation of a part of the dopaminergic reward system in response to food cues (Davids et al., 2010) and to food receipt compared to normal-weight children (Stice et al., 2008). These findings suggest that food may be experienced as less rewarding by obese children.To our knowledge, there is until now no evidence for this model based on behavioral measures or self-report data in

children. Few studies in adults and adolescents found evidence for the relation between reduced self-reported RS and uncontrolled eating, emotional eating, binge eating, and obesity (Davis et al., 2004; Davis & Fox, 2008; Goldfield et al., 2010; Keränen, Rasinaho, Hakko, Savolainen, & Lindeman, 2010; Pagoto, Spring, Cook, McChargue, & Sneider 2006).

These seemingly opposing data might reflect a **dynamic vulnerability (DV) model** for obesity that may evolve and change over time in response to overeating (Stice et al., 2011). The DV-model states that it is possible that heightened generalized RS is an initial risk factor for excessive food intake among normal weight individuals resulting in a positive energy balance and weight gain. However, the excessive food-intake can overload the DA system in such a way that it reduces the DA activity. An adaptive decrease in dopamine D2 receptor (D2R) is suggested to contribute to the reduced responsiveness of the striatum to palatable food (for the biochemical mechanisms see review Kenny, 2011). Hence, excessive overeating is assumed in the long run to lead to an insensitive reward system, which enhances further overeating to reach an acceptable level of hedonic satisfaction (Davis et al., 2004; Lowe et al., 2009; Stice et al.,2011).

This DV-model was already evidenced in adults showing a curvi-linear relationship between BMI and RS, based on self-report (Davis & Fox, 2008), but has never been examined in children. Such knowledge seems however pivotal in unraveling differential mechanisms leading to overeating but also in tailoring early intervention.

The current study aimed to investigate the DV model in children by analysing the association between self-reported RS and bodyweight in children. From the age of 10 years, it seems relevant to assess RS via self-report. Based on the findings of Davis and Fox (2008), we expect a positive association between self-reported RS and bodyweight, which will change to a negative association among children with obesity.

Method

Participants

Participants (10-15y) were recruited from two schools in the Dutch-speaking part of Belgium. Passive informed consent was obtained from parents. Parents received a letter explaining the purpose and method of the study two weeks prior to the data collection and they were asked to fill out a form if they did not want their child to participate in the study. Less than 2% of the parents did not allow their child to participate. Moreover, active informed consent was obtained from the children whose parents gave permission to participate in the study. All children agreed to participate. The questionnaires were administered during a class period. Children had approximately 15 minutes to complete the survey. This procedure resulted in a sample of 438 children (52.5% female) with a mean age of 12.07 years (*SD* = 1.51; range = 10-15 years). All participants were following a regular academic track. This study was approved by the ethics committee of the department of Developmental, Social, and Personality Psychology of the Ghent University.

Measures

Body weight. Each participant reported on his or her own height and weight. The Body Mass Index (BMI) (weight/height^2) was determined for each child. In order to make BMI comparisons between children of different ages, this study uses the adjusted BMI ((actual BMI/ Percentile 50 of BMI for age and gender) x 100). The 50th percentiles of the BMI for age and gender are based on normative data (Fredriks, van Buuren, Wit, & Verloove-Vanhorick, 2000). An adjusted BMI score equal to or smaller than 85% is considered as underweight, a score equal to or greater than 120% as overweight, and a score equal to or greater than 140% as obese (Van Winckel & Van Mil, 2001). In the current sample 10.5% of the children were classified as underweight, 67.1% as average weight, 9.4% as overweight and 13.0% as obese.

Data on the validity of self-reported weight and height suggest that preadolescents and adolescents provide information on their weight and height that is as valid as the information provided by adults (with correlations between self-reported and objectively measured data up to r = .98 for weight and r =.73 for height) (Field et al., 1999).

Reward Sensitivity The BIS/BAS self-report scale was administered (Carver & White, 1994). This scale measures affective responses to impending rewards (Behavioural Approach System, BAS) or punishments (Behavioural Inhibition System, BIS) and contains 20 items, scored on a 4-point Likert scale. The BAS items are divided into three subcategories: items tapping strong pursuit of appetitive goals (BAS Drive) (e.g., "I go out of my way to get things I want"), positive affect/excitability (BAS Reward Responsiveness) (e.g., "When good things happen to me, it affects me strongly"), and the inclination to seek out new rewarding situations (BAS Fun Seeking) (e.g. "I'm always willing to try something new if I think it will be fun"). In accordance with Dawe and colleagues (Dawe, Gullo, & Loxton, 2004; Dawe & Loxton, 2004), the BAS Drive scale is the main focus in the current study. These authors suppose that relative to the other BAS scales, BAS Drive is the best predictor of appetitive motivation and approach behaviour and is purported to closely reflect individual differences in the activity of brain reward circuitry (Pickering & Gray, 1999). This assumption was also underscored by imaging research (Beaver et al., 2006) examining the relationship between the BAS Drive scale and neural responses to appetizing foods (e.g. chocolate cake, pizza) using fMRI in healthy volunteers. They found that BAS Drive scores significantly predicted activation to appetizing foods (relative to bland foods) in the brain areas of reward. Relative to the other BAS scales, it has a unique predictive quality to such cues over and above that offered by the other two scales (Beaver et al., 2006).

The alpha coefficients in the present study were .88 for the BAS Drive scale, .80 for the BAS Reward Responsiveness scale and .72 for the BAS Fun Seeking scale.

Statistical analyses

To investigate the predicted inverted U-relationship of reward sensitivity with adjusted BMI, we fitted a quadratic regression model of BAS Drive on adjusted BMI. To control for gender and age effects, we also added these variables to the model as well as second order interactions between adjusted BMI, age, and gender. An orthogonal polynomial basis was used for the quadratic component in adjusted BMI. Gender was added to the model as a dummy variable, coded 1 for males and 0 for females. To follow the example of Davis and Fox (2008), we also fit a non-parametric regression model to the data to find out whether the predicted non-linear relationship is different from the one we predicted. To validate if the fitted model is indeed quadratic, we selected the equivalent degrees of freedom for the smoothing spline (as control for our main analysis) in adjusted BMI by means of generalized cross-validation (Wood, 2000).

RESULTS

Descriptive statistics

Table 1 presents the means, standard deviations, quartiles and range values for adjusted BMI and the BAS Drive subscale, split up for boys (n = 208) and girls (n = 230). In terms of adjusted BMI, there were no significant differences between boys and girls (t = 0.02, df = 429, p = 0.98). For the BAS Drive subscale scores varies between 1 and 4 whereby, boys had a marginal higher average score compared to girls (t = 1.93, df = 406, p = .05).

Table

Regression analysis

In Table 2, the analysis of variance is presented for the quadratic regression, with BAS Drive score as dependent variable. None of the second order interaction

terms is significant (Sex X adjusted BMI: p = .91, Age X adjusted BMI: p = .65 and Sex X Age: p = .98). There is however an effect of adjusted BMI (p = .013). More specifically the quadratic component of adjusted BMI is significant (p = 0.011). Table 3 represents the coefficients for the additive quadratic regression of BAS Drive on adjusted BMI, sex and age. The additive model accounts for almost 3% of the variation (R^2 = 0.028).

Table 2

Table 3

In Figure 1a, the partial quadratic relationship between BAS Drive and adjusted BMI is displayed by means of an effect plot (Fox, 1987, Fox & Hong, 2009). Similarly, Figure 1b shows an effect plot for the additive non-parametric regression presented earlier. The equivalent degrees of freedom for the adjusted BMI spline term in the model is 2.23 which is very close to the 2 degrees of freedom used in quadratic regression. The similarity between the non-parametric and the quadratic regression model is also expressed by the high correlation of .95 between the fitted values of both models, which underscores the validity of the model.

Figure 1a

Figure 1b

As depicted in the Figure 1a there is a positive relationship between adjusted BMI and the BAS Drive score until an adjusted BMI score of 133% is reached. Above this change point, the direction of the relationship is negative.

Discussion

This is the first study to demonstrate the quadratic association between RS and bodyweight in a group of healthy children with a wide variety in bodyweight. It was shown that self-reported generalized RS shows a great level of individual differences covering the wide range of the BAS drive scale and interestingly this is significantly associated with adjusted BMI. Consistent with the findings in adults (Davis & Fox, 2008) the results show a positive association in the normal weight and overweight children, which changed to a negative association among the children with obesity, suggesting that the DV model is also feasible in children.

As predicted, the children with overweight report high levels of RS, which is consistent with previous research in adults showing that high RS is correlated with increased body weight (Davis et al., 2004; Davis, Levitan, Muglia, Bewell, & Kennedy, 2004; Franken & Muris, 2005, Mobbs et al., 2010; but Pagoto et al., 2006). This finding accords with the premise that high RS individuals are more likely to approach and take pleasure form natural rewards like food. Consequently, these individuals will probable eat more when palatable foods are omnipresent. Evidence for this account was already found in adult studies proving a significant positive association between RS and overeating, the preference for high fat and sweet food (Davis, Patte, Tweed, & Curtis, 2007; Guerrieri et al., 2008), binge eating (Davis & Woodside, 2002; Loxton & Dawe, 2001), and food cravings (Franken & Muris, 2005).

Additionally, the current finding of a turning point resulting in decreased RS in obese children accords with previous research in adults (Davis & Fox, 2008; Davis et al., 2004) and with the hypothesis that RDS is also at play in childhood obesity. Others already showed in a decrease of activation in reward areas in obese versus lean children, implying that food cues may be experienced as less rewarding by obese children (Davids et al., 2010). Animal studies found the same reward dysfunction induced by drugs of abuse and hypothesized that this deficit may contribute to the

transition from controlled to uncontrolled drug use by providing a new source of motivation to consume the drug in order to alleviate the persistent state of diminished reward (Ahmed & Koob, 2005; Kenny et al., 2006). Therefore, it is possible that low RS may perpetuate pathological eating as a mean of compensating for decreased activation of reward circuits as a way to alleviate the persistent state of negative reward (Kenny, 2011; Berridge & Robinson, 1998; Wang et al., 2001).

Overall, the current study suggests that already in children individual differences in RS were remarkable. The actual observations can help us understand why seemingly conflicting findings in overweight and obese individuals can index one model. Though still speculative, the results subscribe the dynamic vulnerability (DV) model for obesity (Davis, et al;, 2004; Lowe et al., 2009; Stice et al., 2011): in a food-abundant environment, high RS leads to excessive food-intake which may trigger neurobiological adaptations resulting in anhedonia and further overeating as compensation. That this feed forward process may already be observable at young age was suggested by animal studies. In mice, the exposure to high fat, high sugar diets during *early periods in life*, might induce changes in the DA brain reward circuits, resulting in subjects that are exceptionally vulnerable to environmental factors that contribute to obesity (Shalev et al., 2010).

The actual findings also underpin the importance of the clinical cut off points overweight versus obese. In research these groups are too often examined as a whole: overweight/obese versus average weight, and this way possible diversities between the groups are disguised.

Although, the focus in the current study was on BAS Drive as an index of individual sensitivity to both hedonic and motivational aspects of reward, we also analyzed the data of the other BAS scales. A linear relationship was found between

adjusted BMI and the BAS Reward Responsiveness which measures the positive hedonic value obtained from rewards (Carver & White, 1994; Beaver et al., 2006). This finding is in line with the findings of Pagoto et al. (2006) revealing diminished enjoyment (positive affect) of rewarding activity in young women with higher BMI. Furthermore, the absence of a significant relationship between BMI and BAS Fun Seeking in the current data could be expected since BAS Fun Seeking is about seeking new rewarding experiences as opposed to rewards in the immediate environment. Generally, our findings accords with the observation that the motivation to obtain palatable food increases during the development of obesity (BAS Drive, curvilinear) while the hedonic value obtained from consuming palatable food decreases (BAS Reward Responsiveness) (Kenny, 2011) and underline the importance of the specific assessment of potential subcomponents within BAS in future studies.

The current research involved children, but since the adolescent is characterized by a heightened RS, it seems also worthwhile to examine in future research the specific association between RS and BMI in adolescents. Reward-related processes appear to develop in a curvilinear manner with a peak during adolescence, while inhibitory processes show a protracted linear development throughout adolescence, leaving the adolescent with highly sensitive, reward-driven processes that can only be moderately regulated by gradually developing inhibitory processes (for a review, see Hardin, 2010). We performed a first analyses on a small group of 34 adolescents age 16-17 years and found that the association between RS and BMI was indeed attenuated with age. Compared to the younger children, the lower scores at the extremes disappeared among the adolescents; they all reported relative high RS, independent of the BMI.

However, the present study's cross-sectional design constrains the interpretation of the findings. It remains unclear if the responsiveness of brain reward

systems is influenced by intrinsic or diet-induced alterations. Future longitudinal research is needed to clarify this remarkable and promising hypothesis, as direction of effects cannot be determined from the current study. A second limitation is the use of self-reported data on weight and height. Although research has generally confirmed the validity of using self reported physical measures on a group level, the possibility still exists that self-reports of these physical measures may be biased or incorrectly represented among those suffering from overweight or obesity. If possible, future research may therefore use objective measures of weight and height instead of self-reports. Otherwise, a way to improve the accuracy of the self-reported data, may be the inclusion of questions that help concretize the actual moment of measurement (e.g. ask participants when they were last weight and their height recorded). Finally, since recent twin research in children showed that the genetic influence on childhood obesity persists despite the obesogenic environment (Wardle, Carnell, Haworth, & Plomin, 2008) it is recommended in future research to include data on parental BMI.

In sum, the current study provide evidence that individual differences in RS may play a critical role in the vulnerability to overeat and becoming overweight or obese and suggest that initial high RS over time may decrease due to diet-induced alterations in the brain fostering further overeating. This means that in treatment it seems promising to focus on alteration of food reward value or the offer of reward alternatives (Volkow et al., 2003; Volkow et al., 2008). Furthermore, the DV model implies that prevention programs should strive to reduce intake of high-fat and high-sugar foods during development to avoid the decrease in RS and reduce the risk for future weight gain in vulnerable populations (Stice et al., 2011)

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Table 1 - Distribution of adjusted BMI and Reward Sensitivity scores (BAS Drive).

	Mean	SD	Minimum	Q1	Median	Q3	Maximum
Adjusted BMI							
Males	109.48	28.25	76.05	91.13	99.41	118.72	216.98
Females	109.43	27.46	67.21	91.20	102.53	118.11	222.92
BAS Drive							
Males	2.53	0.86	1.00	2.00	2.50	3.25	4.00
Females	2.38	0.72	1.00	2.00	2.25	2.75	4.00

Table 2 - Analysis-of-variance for the regression model fit to BAS Drive

Source	*Sum of squares*	Df	*F*	*p*
Sex	2.37	1	3.77	0.053
Age	< 0.01	1	< 0.01	0.977
BMI	5.54	2	4.41	0.013
Linear	0.02	1	0.04	0.849
Quadratic	4.10	1	6.53	0.011
Sex x BMI	0.12	2	0.09	0.912
Linear	0.02	1	0.04	0.842
Quadratic	0.09	1	0.15	0.702
Age X BMI	0.54	2	0.43	0.651
Linear	0.01	1	0.01	0.923
Quadratic	0.52	1	0.84	0.361
Sex X age	< 0.01	1	< 0.01	0.984
Error	268.73	428		

Table 3 - Coefficients and standard errors for the additive quadratic regression BAS Drive on BMI, Sex and Age

	Coefficient	Standard Error
Intercept	2.54	0.31
BMI		
Lineair	0.45	0.80
Quadratic	-2.32	0.79
Sex (Female = 1)	-0.15	0.08
Age	-0.00	0.03
R squared = 0.028		

Figure Caption

Figure 1. Fitted BAS Drive scores by adjusted BMI, averaging for other terms in the model. For each panel a rug plot at the bottom shows the distribution of adjusted BMI. The broken lines around the fit represent the 95-percent confidence interval.

Figure 1a

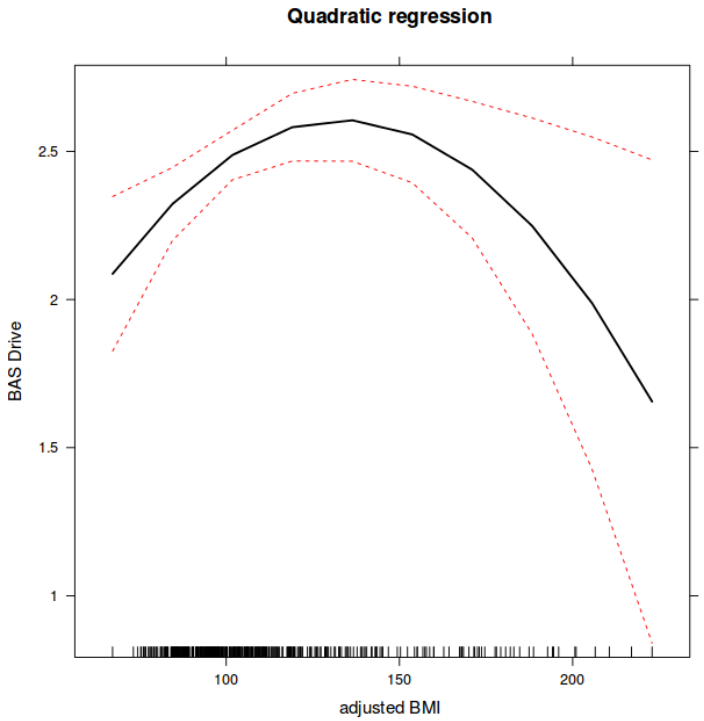
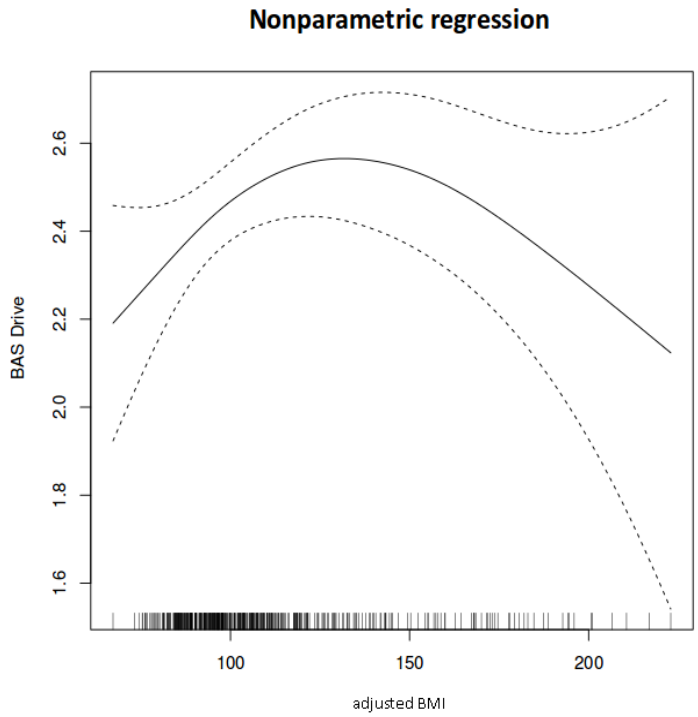

Quadratic regression
BAS Drive
2.5
2
1.5
1
100
150
200
adjusted BMI

Figure 1b


Nonparametric regression
BAS Drive
1.6
1.8
2.0
2.2
2.4
2.6
100
150
200
adjusted BMI