Published in final edited form as:
Eat Disord. 2013 ; 21(3): 265–274. doi:10.1080/10640266.2013.779190.

What Contributes to Excessive Diet Soda Intake in Eating Disorders: Appetitive Drive, Weight Concerns, or Both?

Tiffany A. Brown and **Pamela K. Keel**
Department of Psychology, Florida State University, Tallahassee, Florida, USA

Abstract

Excessive diet soda intake is common in eating disorders. The present study examined factors contributing to excessive intake in a sample of individuals with lifetime eating disorders based on proposed DSM-5 criteria (n=240) and non-eating disorder controls (n=157). Individuals with eating disorders, particularly bulimia nervosa, consumed more diet soda than controls. Eating disorder symptoms that reflect increased appetitive drive or increased weight concerns were associated with increased diet soda intake. Increased weight concerns were associated with increased diet soda intake when levels of appetitive drive were high, but not when they were low. Results highlight the importance of monitoring diet soda intake in individuals with eating disorders and may have implications for the maintenance of dysregulated taste reward processing in BN.

Introduction

Individuals with eating disorders consume excessive amounts of diet soda (Klein, Boudreau, Devlin, & Walsh, 2006). Excessive consumption of soda has been linked to various health problems, such as dental carries and erosion due to the high concentration of phosphoric and citric acids in soda (Pallavi & Rajkumar, 2011), and increased risk of bone loss and bone fractures (Wyshak, 2000; Wyshak & Frisch, 1994). Given that women with eating disorders already have problems with dental erosion (Mehler, Crews, & Weiner, 2004) and reduced bone density (Mehler, Gray, & Schulte, 1997), the addition of excessive quantities of diet soda may exacerbate already existing health problems. Thus, understanding possible explanations for excessive diet soda intake is both theoretically and clinically relevant.

Both biological and psychological explanations have been proposed to explain excessive intake of diet soda including, increased appetitive drive and/or increased weight concerns. In regard to biological explanations, recent studies have examined artificial sweetener intake, such as those in diet soda, as an indicator of appetitive drive in eating disorders. Under this paradigm, diet soda consumption can be seen as a model of sham feeding in which orosensory stimulation (i.e., taste stimuli) is separated from postingestive feedback (i.e., physiological satiation). As individuals with eating disorders often have concerns about consuming caloric content, sham feeding studies allow researchers to separate the drive to consume from the potential cognitive avoidance of caloric stimuli. Using a modified sham feeding paradigm, Klein and colleagues (2009) found that women with Bulimia Nervosa (BN) consumed more artificially sweetened solutions than controls, which the authors attributed to a higher appetitive drive in BN. In a second study, Klein and colleagues (2010) found that after controlling for consumption of unsweetened solutions, women with Anorexia Nervosa (AN) consumed the same amount of artificially sweetened solutions as

Address correspondence to Tiffany A. Brown, Department of Psychology, Florida State University, 1107 W. Call Street, Tallahassee, FL, 32306, USA. brown@psy.fsu.edu.

controls. The authors attributed this finding to a possible decreased appetitive drive in AN relative to BN or to a learned consequence of the pattern of acute starvation and restriction. Thus, findings from studies of sham feeding highlight possible differences between specific eating disorders in diet soda consumption, which may reflect potential biological differences in appetitive drive. However, these studies have not made direct comparisons between diagnostic groups on intake.

In regard to psychological explanations of excessive diet soda intake, both AN and BN are associated with increased weight concerns, which are often translated into attempts to avoid highly caloric or energy dense foods by substituting low calorie foods. Given the commonly held belief that consumption of diet soda facilitates weight management (Tordoff & Alleva, 1990), excessive diet soda intake may reflect consumption of a “safe” food that mimics taste associated with nutritive substances in a non-caloric product. If increased weight concerns drive diet soda intake, then intake may be particularly elevated in eating disorders defined by high concerns about weight (i.e. AN, BN, Eating Disorder Not Otherwise Specified (EDNOS)), but not in disorders that do not require weight concerns (i.e. Binge Eating Disorder (BED) and some forms of EDNOS).

Thus, high appetitive drive, weight concerns, or their combination may contribute to excessive consumption of diet soda. The purpose of the present study was threefold. First, we sought to confirm that diet soda intake was higher in individuals with eating disorders compared to controls. Second, we sought to compare specific DSM-5 diagnostic groups on diet soda intake and to explore the influence of specific DSM criteria on diet soda intake, as symptoms may differentially reflect appetitive drive and weight and shape concerns. We elected to use DSM-5 criteria due to the heterogeneity in symptom presentation within DSM-IV EDNOS, which may mask potential differences between groups. Specifically, BED, which is designated as EDNOS in DSM-IV, may reflect different levels of appetitive drive and weight/shape concerns than someone with atypical or subthreshold AN or BN. Studies have demonstrated that draft DSM-5 criteria successfully reduce the heterogeneity associated within EDNOS, while maintaining the diagnostic validity of AN and BN (Keel, Brown, Holm-Denoma, & Bodell, 2011; Machado, Goncalves, & Hoek, 2013). Third, we sought to explore the influence of continuous indicators of appetitive drive and weight/shape concerns on diet soda intake. We hypothesized that individuals with eating disorders, and particularly those with BN, would have greater diet soda intake compared to controls based on prior studies. Further, we expected diagnostic items associated with appetitive drive (objective binge eating episodes (OBE), overeating) and weight concerns (fear of gaining weight, overvaluation of weight/shape) to be associated with greater diet soda intake. Finally, individuals high in both appetitive drive and in weight/shape concerns would report the greatest diet soda consumption.

Method

Participants

Participants came from an epidemiological study examining eating and health attitudes and behaviors and completed self-report surveys during the springs of 1982, 1992, or 2002 (Heatherton, Nichols, Mahamedi, & Keel, 1995; Keel, Heatherton, Dorer, Joiner, & Zalta, 2006; Zuckerman, Colby, Ware, & Lazerson, 1986). In 2002, participants from the 1982 and 1992 cohorts were recruited for 10- and 20-year follow-up, respectively. Participants whose survey responses indicated the presence of an eating disorder at any assessment wave were invited to participate in interviews (n=294, 11.8% of the full sample of n=2491), which were conducted between 2003–2007. Of cases recruited for the interviews, 196 (67%) participated. A case-controlled design was used to match these participants on age, gender, and ethnicity with individuals whose survey data did not indicate presence of an eating

disorder. Data for the current study came from individuals diagnosed with a lifetime eating disorder according to proposed DSM-5 criteria including: AN (n=48), BN (n=44), BED (n=20), EDNOS (n=128), and non-eating disorder controls (CTRL, n=157). Lifetime eating disorder diagnoses included individuals who were either currently ill at the time of the interview or had a past history of an eating disorder prior to the interview (at some point during their lifetime). Of the individuals with a lifetime eating disorder diagnosis, 38.8% were currently ill at the time of the interview. Diagnostic groups did not differ on age ($F(4,368)=1.318$, $p=.263$) or ethnicity ($\chi^2(16)=12.045$, $p=.741$), with the majority of the sample being Caucasian (76.0%). Groups did differ on current BMI, with individuals with a lifetime diagnosis of BED having a higher current BMI than individuals with a lifetime diagnosis of AN, BN, EDNOS, and controls (all p-values <.041). Additionally, individuals with a lifetime diagnosis of EDNOS had a higher current BMI than individuals with AN ($p=.003$).

Procedures and Measures

All participants completed written informed consent prior to participation for each stage of the two-stage epidemiological study.

Stage 1: Survey Data—*Soda (Diet and Regular) Intake* was assessed by two, single-item questions asking "How many cans of (Diet) Soda do you consume per day?" consistent with methods used in other epidemiological studies (Fowler, et al., 2008; Pettinato, Loud, Bristol, Feldman, & Gordon, 2006).

The *Eating Disorder Inventory* (EDI; Garner, Olmstead, & Polivy, 1983) is a well-validated self-report, 6-point forced choice inventory that assesses behavioral and psychological traits common in BN and AN, including Bulimia and Drive for Thinness subscales. Internal consistency in the present sample was high (Cronbach's alpha: Drive for Thinness = .93; Bulimia = .89). The Bulimia subscale was used as a proxy for appetitive drive given that it primarily measures binge eating, or perhaps more specifically, the overconsumption of food (Garner, et al., 1983). Thus, the Bulimia subscale reflects the behavioral component of appetitive drive: the tendency towards overconsumption of food. Consistent with this premise, previous research has found associations between the Bulimia subscale and measures of appetitive drive (Ochner, Green, van Steenburgh, Kounios, & Lowe, 2009). The Drive for Thinness subscale was used as a proxy for weight concerns given its significant associations with other measures of weight concerns (Winzelberg, et al., 2000).

Stage 2 Interview Data—The *Structured Clinical Interview for DSM-IV Axis I Disorders* (SCID-I; First, Spitzer, Gibbon, & Williams, 1995) was used to assess lifetime eating disorders. Within the eating disorders module, interviewers administered all questions to each participant, omitting skip rules. For example, all questions within the AN section were asked, regardless of the participant's response to the item regarding low weight. Further, questions regarding frequencies of bingeing and purging behaviors were coded, regardless of whether they met the threshold for twice per week for three months. This allowed us to capture subthreshold forms of DSM-IV AN and BN that are proposed to be relegated to full threshold diagnoses under DSM-5 draft criteria. Interrater reliability for lifetime eating disorders was $\kappa=0.82$. Diagnostic categories generated by the use of proposed DSM-5 criteria represent a hierarchy in which a lifetime diagnosis of AN ruled out a lifetime diagnosis of BN, which ruled out a lifetime diagnosis of BED, which ruled out a lifetime diagnosis of EDNOS.

Data Analyses

Parametric analyses were performed using PASW 18.0. Bonferroni adjusted p-values were used to evaluate the statistical significance of post-hoc comparisons.

Results

Individuals with a lifetime eating disorder consumed more diet soda (Mean (SD)= 4.48 (.49)) than did controls (Mean (SD)= 2.48 (.59); F(1,363)= 6.712, p =.010). Table 1 presents mean comparisons on diet soda and soda intake by DSM-5 diagnosis. Diagnostic groups did not differ on regular soda intake. Individuals with BN drank more diet soda per week than controls (p =.010), but did not differ from the other eating disorder groups (all p-values >.10). No other diagnostic groups differed from controls (all p-values >.17). Further, comparing the control group's consumption of *both* diet and regular soda (3.56 cans of soda total, per day) to the BN group's consumption of diet soda *only* (6.90 cans of diet soda, per day) illustrates the excessive nature of diet soda intake in BN.

Table 2 presents associations between diet soda intake and endorsement of specific DSM criteria. Endorsement of each DSM criterion was associated with significantly greater diet soda intake than non-endorsement, with the exception of the low weight criterion. Examining effect sizes, items associated with appetitive drive (objectively large amount of food, loss of control, and objective binge episodes) were of small effect size. In contrast, fear of gaining weight was associated with a medium effect size for diet soda intake. Inappropriate compensatory behaviors were also associated with significantly greater diet soda intake but the association was of a small effect size.

In regard to the relationship between appetitive drive (EDI Bulimia) and weight concerns (EDI Drive for Thinness) on diet soda intake, no main effect was found for Bulimia (Beta= .067, t(365)=.816, p =.415); however, a main effect was found for Drive for Thinness, such that individuals high on Drive for Thinness consumed more diet soda than individuals low on Drive for Thinness (Beta= .178, t(365)=2.471, p =.014). In addition, there was a significant Bulimia by Drive for Thinness interaction effect (Beta=.196, t(365)=3.185, p =.002). Follow-up tests revealed that at high levels of Bulimia, increases in Drive for Thinness were associated with increases in diet soda consumption (Beta=.333, t(365)=3.785, p <.001). No such relationship was found at low levels of Bulimia (Beta=.024, t(365)=.276, p =.782).

Discussion

Results from the present study demonstrated that individuals with a lifetime eating disorder, specifically those with BN, consume more diet soda than controls. Further, diet soda intake in BN was indeed excessive, and not just a function of replacing what would be regular soda with a diet product. Additionally, endorsement of diagnostic items associated with appetitive drive or weight concerns were associated with increased diet soda intake, with fear of gaining weight or becoming fat having a medium effect size. Finally, increases in Drive for Thinness were associated with increased diet soda intake. No such relationship was found for Bulimia. However, there was a significant interaction between Bulimia and Drive for Thinness, such that individuals high on both measures consumed the greatest amount of diet soda.

Our findings are consistent with results from previous sham feeding studies (Klein, et al., 2009; Klein, et al., 2010) and provide a potential explanation for why individuals with BN (Klein, et al., 2006; Klein, et al., 2009), but not AN (Klein, et al., 2010) or BED, consume more artificially sweetened solutions than controls. Results extend Klein and colleagues' (2009) rationale that increased appetitive drive in BN drives excessive artificial sweetener

consumption by highlighting the importance of weight concerns. Indeed, the importance of high weight concerns in addition to high appetitive drive appears to be an important factor associated with excessive diet soda intake. While individuals with AN and BN share concerns regarding weight and shape, individuals with AN appear to have a relatively lower appetitive drive than those with BN (Klein et al., 2010), which may help explain why individuals with AN did not consume more diet soda than controls. Similarly, individuals with BED, who, like individuals with BN, have high appetitive drive, did not consume more diet soda than controls. Given that weight concerns are not necessary for a diagnosis of BED, appetitive drive in BED may be expressed through consumption of foods with a high caloric content, as opposed to consumption of non-nutritive substances. Supporting this, the BED group consumed approximately 2 cans of regular soda per day, compared to 1 can in the control group, and less than 1 can per day in the other eating disorder groups. Thus, it appears that the combination of both high appetitive drive *and* high weight concerns in individuals with BN may help explain why this group alone consumed excessive amounts of diet soda.

Results from the current study may potentially be explained by dysregulation of taste-reward processing in individuals with eating disorders, particularly BN. Previous neurobiological studies have found that sucralose, an artificial sweetener often used in diet soda, stimulates the same taste reward circuitry as sucrose but at a lower magnitude (Frank, et al., 2008). Thus, sucralose activates sweet taste reward circuits but may not fully satisfy desire for sweet caloric ingestion, which may contribute to excessive food intake. Consistent with this, another study found that consuming more than three artificially sweetened beverages per day prospectively predicted a two-fold increase in weight compared to not drinking such beverages (Fowler, et al., 2008). Further, studies have found evidence that artificial sweeteners may actually enhance postingestive hunger (Mattes & Popkin, 2009). If artificial sweetener intake contributes to the overconsumption of food, then this may imply that diet soda consumption may be particularly problematic for individuals with BN, given that they are already prone to loss of control eating episodes and have reduced reward-processing to nutritive stimuli (Frank, et al., 2006). Future studies should examine whether diet soda consumption may play a role in maintaining dysregulated taste processing and appetite in BN.

The present study had several strengths that are worth noting. First, lifetime eating disorder diagnoses were established through diagnostic interview by interviewers demonstrating high interrater reliability. The present study also utilized well-validated measures of eating pathology that demonstrated good internal consistency, increasing power for analyses and reducing the risk of type II error. Further, to our knowledge, this is the first study to examine factors influencing diet soda intake and make direct comparisons among eating disorder diagnoses.

Although the present study had several strengths, there were also limitations that merit discussion. First, diet soda consumption was based on current self-report at the time of the survey in the first stage of the study, while diagnoses were assessed for lifetime occurrence during the second stage of the study. Although these methods are consistent with other epidemiological studies using a two-stage design (Hudson, Hiripi, Pope, & Kessler, 2007), this procedure introduces a time lapse between survey and interview assessments. Further, the use of lifetime diagnoses may not fully represent the behaviors of individuals presenting for treatment of a current eating disorder. However, we still found differences between individuals with lifetime BN and those without a history of an eating disorder, which may imply that excessive diet soda intake in BN persists after state of the illness. In addition, assessment of appetitive drive and weight concerns were concurrent with diet soda intake, allowing us to examine the relevance of these features. Further, the sample size for the BED

group was small, which may have reduced power to detect significant group differences. In contrast, the EDNOS group was large but the heterogeneous nature of this group may have masked associations between specific forms of EDNOS and excessive diet soda intake. Finally, the results of this study are cross sectional, and thus, no causal or temporal inferences can be made.

Results from the present study also have some potential implications for clinical practice. Recent research has found that among patients with AN, consumption of higher energy density foods predicted treatment success (Schebendach, et al., 2008), highlighting the importance of attending to food intake patterns throughout treatment. As diet soda represents a low energy density beverage, monitoring diet soda intake in patients with eating disorders may be clinically beneficial. This may also be relevant for patients with BN, as excessive diet soda intake may not only exacerbate the medical consequences of the disorder (e.g. dental, esophageal, skeletal), but also may maintain or contribute to dysregulated eating patterns. However, prior to more definitive recommendations regarding monitoring diet soda consumption, future studies should examine whether and how excessive diet soda consumption specifically affects treatment outcome.

In summary, results from the present study highlight a phenomenon that is commonly acknowledged among the clinical community, but often understudied empirically. Results demonstrate that individuals with a lifetime diagnosis of BN consume excessive amounts of diet soda compared to non-eating disorder controls. These patients may use diet soda as a way to satiate their appetitive drive, or “fill up”, without adding calories. Indeed, this pattern may be due to the combination of high appetitive drive and high weight concerns in BN.

Acknowledgments

This work was supported by a grant from the National Institute of Mental Health (R01MH63758; PI: Pamela K. Keel). Portions of this work were presented at the 2011 Annual Meeting of the International Conference on Eating Disorders in Miami, FL.

References

First, M.; Spitzer, RL.; Gibbon, M.; Williams, JBW. Structured Clinical Interview for DSM-IV Axis I Disorders--Patient Edition (SCID/P). Biometrics Research Department: New York State Psychiatric Institute; 1995.

Fowler SP, Williams K, Resendez RG, Hunt KJ, Hazuda HP, Stern MP. Fueling the obesity epidemic? Artificially sweetened beverage use and long-term weight gain. Obesity (Silver Spring). 2008; 16(8):1894–1900. [PubMed: 18535548]

Frank GK, Oberndorfer TA, Simmons AN, Paulus MP, Fudge JL, Yang TT, et al. Sucrose activates human taste pathways differently from artificial sweetener. Neuroimage. 2008; 39(4):1559–1569. [PubMed: 18096409]

Frank GK, Wagner A, Achenbach S, McConaha C, Skovira K, Aizenstein H, et al. Altered brain activity in women recovered from bulimic-type eating disorders after a glucose challenge: a pilot study. Int J Eat Disord. 2006; 39(1):76–79. [PubMed: 16254868]

Garner DM, Olmstead MP, Polivy J. Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. International Journal of Eating Disorders. 1983; 2(2):15–34.

Heatherton TF, Nichols P, Mahamedi F, Keel P. Body weight, dieting, and eating disorder symptoms among college students, 1982 to 1992. Am J Psychiatry. 1995; 152(11):1623–1629. [PubMed: 7485625]

Hudson JI, Hiripi E, Pope HG Jr, Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. Biol Psychiatry. 2007; 61(3):348–358. [PubMed: 16815322]

Keel PK, Brown TA, Holm-Denoma J, Bodell LP. Comparison of DSM-IV versus proposed DSM-5 diagnostic criteria for eating disorders: reduction of eating disorder not otherwise specified and validity. Int J Eat Disord. 2011; 44(6):553–560. [PubMed: 21321984]

Keel PK, Heatherton TF, Dorer DJ, Joiner TE, Zalta AK. Point prevalence of bulimia nervosa in 1982, 1992, and 2002. Psychol Med. 2006; 36(1):119–127. [PubMed: 16202192]

Klein DA, Boudreau GS, Devlin MJ, Walsh BT. Artificial sweetener use among individuals with eating disorders. Int J Eat Disord. 2006; 39(4):341–345. [PubMed: 16523474]

Klein DA, Schebendach JE, Brown AJ, Smith GP, Walsh BT. Modified sham feeding of sweet solutions in women with and without bulimia nervosa. Physiol Behav. 2009; 96(1):44–50. [PubMed: 18773914]

Klein DA, Schebendach JE, Gershkovich M, Smith GP, Walsh BT. Modified sham feeding of sweet solutions in women with anorexia nervosa. Physiol Behav. 2010; 101(1):132–140. [PubMed: 20438741]

Machado PP, Goncalves S, Hoek HW. DSM-5 reduces the proportion of ednos cases: Evidence from community samples. Int J Eat Disord. 2013; 46(1):60–65. [PubMed: 22815201]

Mattes RD, Popkin BM. Nonnutritive sweetener consumption in humans: effects on appetite and food intake and their putative mechanisms. Am J Clin Nutr. 2009; 89(1):1–14. [PubMed: 19056571]

Mehler PS, Crews C, Weiner K. Bulimia: medical complications. J Womens Health (Larchmt). 2004; 13(6):668–675. [PubMed: 15333281]

Mehler PS, Gray MC, Schulte M. Medical complications of anorexia nervosa. Journal of Womens Health. 1997; 6(5):533–541.

Ochner CN, Green D, van Steenburgh JJ, Kounios J, Lowe MR. Asymmetric prefrontal cortex activation in relation to markers of overeating in obese humans. Appetite. 2009; 53(1):44–49. [PubMed: 19426775]

Pallavi SK, Rajkumar GC. Soft drinks and oral health- A review. Indian Journal of Public Health Research & Development. 2011; 2(1):135–138.

Pettinato AA, Loud KJ, Bristol SK, Feldman HA, Gordon CM. Effects of nutrition, puberty, and gender on bone ultrasound measurements in adolescents and young adults. J Adolesc Health. 2006; 39(6):828–834. [PubMed: 17116512]

Schebendach JE, Mayer LE, Devlin MJ, Attia E, Contento IR, Wolf RL, et al. Dietary energy density and diet variety as predictors of outcome in anorexia nervosa. Am J Clin Nutr. 2008; 87(4):810–816. [PubMed: 18400701]

Tordoff MG, Alleva AM. Effect of drinking soda sweetened with aspartame or high-fructose corn syrup on food intake and body weight. Am J Clin Nutr. 1990; 51(6):963–969. [PubMed: 2349932]

Winzelberg AJ, Eppstein D, Eldredge KL, Wilfley D, Dasmahapatra R, Dev P, et al. Effectiveness of an Internet-based program for reducing risk factors for eating disorders. Journal of Consulting and Clinical Psychology. 2000; 68(2):346–350. [PubMed: 10780136]

Wyshak G. Teenaged girls, carbonated beverage consumption, and bone fractures. Arch Pediatr Adolesc Med. 2000; 154(6):610–613. [PubMed: 10850510]

Wyshak G, Frisch RE. Carbonated beverages, dietary calcium, the dietary calcium/phosphorus ratio, and bone fractures in girls and boys. J Adolesc Health. 1994; 15(3):210–215. [PubMed: 8075091]

Zuckerman DM, Colby A, Ware NC, Lazerson JS. The prevalence of bulimia among college students. Am J Public Health. 1986; 76(9):1135–1137. [PubMed: 3461714]

Table 1

Mean Comparisons on Diet Soda and Soda Intake by DSM-5 Diagnosis

	AN N=45		BN N=36		BED N=19		EDNOS N=116		CTRL N=149			
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**F(4,360)** *	***p***
Diet soda	5.37[a,b]	1.07	6.90[b]	1.20	4.27[a,b]	1.65	3.41[a,b]	.67	2.48[a]	.59	3.535	.008
Soda	.73	.53	.88	.58	1.84	.80	.75	.32	1.08	.29	.508	.730

* F-test df for: Soda analyses= 355

Note: Superscripts that differ represent significant differences of p<.05 between groups after Bonferroni correction.

Table 2

Associations Between Diet Soda Intake and Endorsement of Specific DSM Criteria.

		Absent			Present					
Measure	**Item**	***n***	**Mean Intake**	**SD**	***n***	**Mean Intake**	**SD**	**t(df)**	***p***	**d**
Appetitive Drive	Objectively large	230	2.75	6.29	133	5.12	8.36	−2.837(219.1)	.005	.32
	Loss of control	195	2.92	6.47	170	4.55	8.07	−2.118(322.9)	.035	.22
	OBE	234	2.75	6.25	121	5.50	8.64	−3.114(186.7)	.002	.37
Influence of W/S	Fear of gaining weight	266	2.57	5.47	99	6.66	10.29	−3.791(119.5)	<.001	.52
	Influence of W/S	122	1.97	4.24	238	4.40	8.16	−3.720(357.9)	<.001	.39
Other Symptoms	ICB	188	2.58	5.38	178	4.82	8.74	−2.928(291.4)	.004	.32
	Low weight	273	3.31	6.51	91	4.83	9.23	−1.451(121.2)	.149	.19

Note: ICB= Inappropriate compensatory behaviors; Intake = Cans of Diet Soda per day; Objectively large= Objectively large amount of food; OBE= Objective binge episode W/S= Weight and Shape