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Associations among sugar sweetened beverage intake, visceral fat, and cortisol awakening response in minority youth

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Abstract

Context—Abdominal adiposity has long been associated with excess caloric intake possibly resulting from increased psychosocial stress and associated cortisol dysfunction. However, the relationship of sugar-sweetened beverage (SSB) intake specifically with cortisol variability and visceral adipose tissue (VAT) is unknown.

Objective—To examine the relationships between SSB intake, VAT, and cortisol response in minority youth.

Design—A cross-sectional analysis.

Setting—The University of Southern California.

Participants—60 overweight/obese Non-Hispanic Black and Hispanic adolescents ages 14–18 years.

Main Outcome Measures—VAT via Magnet Resonance Imaging (MRI), cortisol awakening response (CAR) via multiple salivary samples, and SSB intake via multiple 24-hour diet recalls. SSB intake was divided into the following: low SSB consumers (< 1 servings per day), medium SSB consumers (1 - <2 servings per day), high SSB consumers (≥ 2 servings per day). Analysis

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of covariance were run with VAT and CAR as dependent variables and SSB intake categories (independent variable) with the following a priori covariates: sex, Tanner stage, ethnicity, caloric intake, and body mass index.

Results—The high SSB intake group exhibited a 7% higher VAT compared to the low SSB intake group ($\beta=0.25$, CI:(0.03, 0.33), $p=0.02$). CAR was associated with VAT ($\beta=0.31$, CI:(0.01,0.23), $p=0.02$). The high SSB intake group exhibited 22% higher CAR compared to the low SSB intake group ($\beta=0.30$, CI:(0.02,0.48), $p=0.04$).

Conclusion—This is the first study exploring the relationship between SSB, VAT, and CAR. SSB consumption appears to be independently associated greater abdominal adiposity and higher morning cortisol variability in overweight and obese minority youth. This study highlights potential targets for interventions specifically to reduce SSB intake in a minority youth population.

Keywords

Adolescences; cortisol awakening response; visceral adipose tissue; sugar-sweetened beverage

1. Introduction

Adolescence is a key time to address obesogenic behaviors as children transition into adulthood and begin to make independent dietary choices[1]. Adolescent weight gain has been shown to be persistent over the life cycle, overweight teens are four times more likely to become overweight or obese adults than normal weight peers [2]. Increasingly, the role of sugar sweetened beverage (SSB) consumption has been highlighted as a modifying factor for overweight and obese adolescents. SSB intake has been shown to cluster with multiple other unhealthy lifestyle and eating behaviors in adolescents, such as: intake of fried foods, desserts, increased sedentary activities, and decreases in fruit and vegetable intakes [3]. A recent meta analysis indicated a strong association between SSB consumption and risk of becoming obese in children [4]. In general, high school students are more likely to consume SSB compared to middle and elementary school children [5]. Moreover, Non-Hispanic Blacks (NHB) and Hispanic high school students consume more carbonated and non-carbonated SSBs respectively than non-Hispanic white high school students [5]. This coincides with NHB and Hispanic minorities also being disproportionately at risk for obesity, with approximately 1 in 3 NHB and Hispanic youth (12–19 years) being overweight or obese [6].

Independent of BMI, SSB intake has been implicated as a determinant of type 2 diabetes, and metabolic syndrome [7], however SSB intake is one of many factors associated to metabolic risk. Visceral adipose tissue (VAT) has been shown as risk factor for metabolic syndrome in adolescents [8]. SSB intake was shown to be positively associated with waist circumference and proportion of visceral to subcutaneous fat with no change in total body fat [9], indicating a restructuring of body fat depots. Furthermore, in a six month intervention study, one liter of SSB per day resulted in higher liver fat, skeletal muscle fat, blood triglycerides, and VAT in adults [10]. A minority cohort study demonstrated that VAT accumulation is greatest during young adulthood in NHB and Hispanic populations [11], and

therefore adolescence is an important time period to reduce VAT and SSB intake to minimize metabolic syndrome risk, especially in minorities.

The relationship between SSB and VAT appears to be more nuanced than a simple increase in caloric intake from SSB. Research has shown that intake of SSB is generally not compensated for at later meals[12]. Additionally, evidence indicates that the total intake of calories is greater than those from the added SSB consumption alone, possibly indicating a change in satiety [13]. Recent studies indicate that the stress hormone cortisol, the end product of the hypothalamic-pituitary-adrenal (HPA) axis, may mediate the relationship between sugar intake and fat partitioning. Previous work has found dietary sugar intake and cortisol predicted VAT accumulation in NHB and Hispanic youth (13–18 years of age) [14]. In overweight adolescents, cortisol levels were associated with higher regional fat mass and insulin resistance [15]. In a sample of only overweight Hispanic youth (8–13 years of age), youth with higher morning serum cortisol levels had higher prevalence of metabolic syndrome [16]. In the same adolescent Hispanic sample, morning serum cortisol was associated with a decline in acute insulin response and beta cell function, both of which are pathophysiological underpinnings of metabolic disease[17]. Therefore, the purpose of this study is to examine the interrelationships between of SSB consumption on VAT and cortisol awakening response (CAR) in Hispanic and NHB youth. Our *a priori* hypotheses are: high SSB intake will show a dose response relationship with increasing VAT, increasing SSB will be related to increased CAR, and that the interaction of SSB and CAR will yield differential outcomes on VAT.

2. Materials and Methods

2.1 Participants

Two hundred and forty-one overweight and obese adolescents (8–17 years of age) were recruited from hospitals, clinics, churches, schools, and community centers around the Los Angeles area between 2008 through 2011 into the initial study (Diabetes Risk due to Ectopic Adiposity in Minority Youth)[18]. All subjects were ethnically Hispanic (all four grandparents of Latin-American descent) or self-identified NHB, 8–17 years of age, and either overweight or obese (BMI 85th percentile for age and sex) [19, 20]. For this subset analysis (n=91) the following inclusion criteria were included: Tanner stage 4 or 5 (post-puberty)[21], 13–17 years of age, and having had at least two valid diet recalls within a month of the testing visit. Subjects were excluded if they had a chronic illness (e.g. type 1 or type 2 diabetes), were currently in a weight loss program, or used medications that influenced insulin or body composition. Informed written parental consent and participant assent was obtained before any testing commenced. The Institutional Review Board of the University of Southern California (USC) approved all study procedures.

2.2 Procedures

Subjects attended two visits to the Clinical Trials Unit (CTU) at USC. At the initial visit, a licensed health care professional provided a physical exam for the subjects. Tanner stage was determined via breast stage for females and pubic hair stage for males [22, 23]. The testing visit took place within two months of the initial visit. At the second visit, subjects were

admitted and fed dinner and a snack before 8:00 pm. After 8:00 pm, the subjects began an overnight fast.

To examine salivary cortisol patterns, CTU staff collected three saliva samples at 5:30 am, 5:45 am, and 6:00 am. Samples were collected using the Salivette system (Sarstedt, Newton, NC). Saliva samples were immediately centrifuged at 2500 rpm for 10 min and then frozen at -70 degrees C until assayed. The cortisol awakening response was calculated as the difference between the 5:30 and 6:00 am time points [20]. CAR is a commonly used measure of morning variation and has been associated with disease outcomes [24–28].

Height and weight were measured to the nearest 0.1 kg and 1cm, using a beam medical scale and wall-mounted stadiometer. BMI was calculated as weight (kg) divided by height (m) squared. BMI percentiles were determined using the Center for Disease Control age-and-sex specific values, and were calculated with EpiInfo 2005 (Center for Disease Control, Atlanta, GA).

All subjects underwent abdominal MRI scanning to determine fat deposition on a commercial 3- Tesla MRI system (Excite HD, GE Health-care, Waukesha, WI, USA). Fat quantification for visceral adipose tissue (VAT) abdominal scans was previously described [18] and has been validated in numerous studies [29–32]. Stress was assessed using the Perceived Stress Scale (PSS), a 14-item survey that inquires about perception of stressful experiences in the preceding month [33].

Eighty-one subjects had VAT data. CAR was available for 60 of the 81 with VAT. Student T tests were performed to insure there were no differences between the CAR subset and the larger VAT dataset.

2.3 Dietary Assessment

Dietary intake was assessed over the phone via the multiple pass 24 hour recall method using Nutrition Data System for Research (NDS-R) (University of Minnesota, Minneapolis, MN), which has been validated here [34]. Recalls were performed within one month of the testing visit. To be included in the analysis, subjects needed at least two valid recalls, subjects with less than two recalls or recalls more than a month before the testing visit date were excluded. Beverages considered SSBs were: sweetened soft drinks, sweetened fruit drinks, sweetened tea, sweetened coffee, sweetened coffee substitutes, and sweetened water. A serving was defined as 8 fluid ounces. Subjects were classified into SSB categories based on dietary recall data: low SSB consumers (< 1 servings per day), medium SSB consumers (1 - <2 servings per day), and high SSB consumers (≥ 2 servings per day). These categories were chosen to model an increase in SSB intake by one serving. The American Heart Association (AHA) recommends consuming under 150 kcal/day from added sugar for men and under 100 kcal/day[35]. Since a single serving of SSB can contain anywhere from 100 to 180 kcal, the SSB groups additionally represent an adherence to the AHA's recommendations. SSB was additionally modeled as a categorical variable, rather than a continuous, to minimize reporter bias. Dietary data was also checked for unusual or implausible caloric values via linear regression of caloric intake versus BMI. All subjects

had energy intake residuals less than 2 standard deviations from the mean, and therefore all subjects were included in this analysis.

2.4 Statistical Analysis

R version 3.1.2 (R Foundation for Statistical Computing, Vienna, Austria) was used for all analyses. Perceived stress, VAT, energy intake, and BMI percentile were square root transformed to satisfy normality assumptions. Perceived stress, Tanner stage, ethnicity, sex, BMI percentile, and energy intake were included as covariates. Analysis of covariance (ANCOVA) was used to assess differences in CAR and VAT (dependent variables entered separately) between SSB groups (independent variable), and the following *a priori* covariates were entered: ethnicity, Tanner stage, BMI percentile, sex, and energy intake (calories per day). All covariates were chosen because of previous research indicting possible relationships to either VAT[14] or CAR[36, 37]. SSB by VAT (SSB*VAT) or SSB by CAR (SSB*CAR) interactions were also added to their respective model. In all models, the low SSB group represented the reference category. Log-likelihood ratio tests were used to compare the relative fit of the model after the addition of the SSB*VAT or SSB*CAR interaction term. Tukey post hoc tests were used to evaluate between SSB group differences.

The adjusted means are reported in Figure 1 and Figure 2. Statistical significance was set at $p = 0.05$. The beta value (β) refers to the estimated regression coefficient.

3. Results

Table 1 summarizes the demographics and adiposity variables for subjects with two valid diet recalls by SSB group and outcome variable. Only CAR and daily energy intake differed by SSB category (both $p=0.01$) for both the CAR and VAT analysis.

After adjusting for covariates, the high SSB intake group exhibited a 7% higher VAT compared to the low SSB intake group ($\beta=0.25$, CI: 0.03, 0.33, $p=0.02$) (Fig 1).

VAT was also associated with ethnicity ($p=0.01$), sex ($p=0.01$), and BMI ($p=0.01$). Male subjects exhibited 7% higher VAT compared to female, and Hispanic subjects exhibited 16% higher VAT compared to NHB. Neither sex nor ethnicity interacted with SSB. Post hoc tests revealed the high exhibited greater CAR compared to the low group ($p=0.05$). The addition of CAR improved the fit of the model (0.01). CAR was associated with VAT ($\beta=0.31$, CI=(0.01,0.23), $p=0.02$). The interaction term SSB*CAR did not significantly improve the model fit (Table 2).

The high SSB intake group exhibited 22% higher CAR compared to the low SSB intake group ($\beta=0.30$, CI:(0.02,0.48), $p=0.04$) (Fig 2).

CAR was also associated with sex ($p=0.04$). VAT improved the fit of the model ($p=0.01$). VAT was associated with CAR ($\beta=1.10$, CI:(0.12,1.48), $p=0.02$). The interaction term SSB*VAT did not significantly improve the model (Table 2).

4. Discussion

Contrary to the hypothesis, there was no interactive effect between VAT, CAR, and SSB, rather consuming over two servings of SSB per day intake was independently related to higher VAT and CAR in overweight and obese minority youth compared to drinking less than one serving of SSB per day. CAR was the strongest predictor of VAT (and vice versa), however SSB appears to influence both and therefore is an attractive intervention target to reduce both CAR and VAT.

This is the first paper examining the main effect of SSB on CAR or any biological marker of HPA axis activity. Previous studies have explored the effects of macronutrient intake on the HPA axis with mixed results. In obese adult men, an inverse association between a high fat diet and cortisol metabolism was seen [38], and a high carbohydrate diet was shown to increase cortisol regeneration from cortisone in lean men [39]. However, studies into high protein versus high carbohydrate meals [40, 41], low glycemic index versus high glycemic index [42–44] found no association between the various macronutrients and HPA axis activity. Additionally, much of the previous work between CAR and sugar intake, or sweet perception, has focused on the mediating effect of stress [28, 45–48]. However this study saw no effect of perceived stress on SSB intake nor CAR. The association between elevated CAR and SSB consumption could be a result of chronic elevated cortisol due to metabolic dysregulation increasing the desire to consume SSB. Treatment with exogenous glucocorticoids has been shown to increase food intake [48], and chronic glucocorticoid exposure has been related to increased palatable food intake [49, 50], in particular from sweet foods compared to savory foods [51]. In pathological glucocorticoid exposure, seen in Cushing's disease, patients exhibited increased sweet craving and decreased post meal satiation [52]. Furthermore, patients with Cushing's Disease, VAT was associated with increased post meal hunger [52], also indicating a relationship between dietary consumption cortisol and VAT. The current findings may be modeling a sweet preference due to elevated cortisol.

Additionally, this study showed an increase in VAT with SSB intake. Previous research has implicated SSB consumption, independent of energy intake, in promoting VAT accumulation or increased waist circumference [9, 10, 53–57], although, a majority of this research has taken place in adults [9, 10, 53, 55, 57]. However, adolescence in particular appears to be a critical period in VAT accumulation associated with SSB intake [54], with VAT accrual peaking in young adulthood in minority populations [11]. Zheng and colleagues showed in a longitudinal study that SSB consumption at 15 years of age was associated with higher increases in BMI and waist circumference after 6 years, moreover this relationship was only seen at age 15 and not in younger (pre adolescent) age groups [54]. The current study supports these findings, showing consumption of more than 2 SSB per day was associated with increased VAT after adjusting for daily energy intake and BMI. The mechanism behind the association of SSB intake and VAT has yet to be clarified, however sweetener-type may be related. Stanhope and colleagues have shown fructose beverages, compared to glucose beverages balanced for caloric load, to be positively related to increased VAT [55]. This suggests the possibility that because fructose is directly metabolized in the liver, it increases hepatic triglyceride synthesis and VLDL production and secretion, which may lead to

preferential increase in VAT [55]. A large study of 559 adolescents ages (14–18 years) found that fructose intake was related to VAT, and that VAT mediated fructose related associations with cardiometabolic risk factors[58]. Since popular SSBs have been shown to contain upwards of 60% fructose [59], it is possible that in addition to modeling an increase in SSB intake due to sweet preference, this study is also modeling a snapshot of fat repartitioning due to high fructose intake.

Furthermore, this study showed a moderate positive relationship between CAR and VAT. The VAT itself may provide a mechanism to the elevated CAR. Glucocorticoid receptors in VAT have been linked to heightened local cortisol production and receptor expression [60]. Likewise, high glucocorticoid levels have been linked to increased de novo lipogenesis in coordination with high insulin levels, and this in turn influences fat partitioning [61][60]. Interestingly, this increase in de novo lipogenesis is similar to the fructose mechanism speculated above[55]. The combination of high CAR, VAT, and high fructose intake from SSB may create a metabolic storm, particularly in the liver. Work in similar samples supports this metabolic storm hypothesis, Gyllenhammer and colleagues found that total and added sugar intake increased the relationship between cortisol and VAT in a similar sample of NHB and Hispanic adolescents[14]. Furthermore, cortisol has been shown to be negatively associated with insulin sensitivity in Hispanic children (8–13 years), and total sugar intake were further shown to be related to increased fat mass and lower insulin sensitivity in Hispanic youth (10–17 years)[62]. Finally, in Hispanic youth ages (8–13 years) metabolic syndrome was associated with increased morning cortisol[16]. While the above studies and this study are all cross-sectional, there appears to be a relationship between VAT, CAR, and dietary sugar intake, in particular SSB, on metabolic health in minority youth.

There are several limitations to note. As previously mentioned this is a cross-sectional study and causality cannot be inferred. This study also has a small sample size, especially for the cortisol variables. Additionally, this study did not take into account sleep variables, which could further elucidate the relationship between VAT, CAR, and SSB. Dietary information on the dinner and snack served at the USC CTU the evening before salivary cortisol samples were collected was not available. Having only two recalls limits dietary data quality, however use of a categorical SSB variable is meant to minimize reporter bias. This study should be repeated with an increased sample size and three or more dietary recalls. Future analysis is needed to explore the difference in VAT and CAR between NHB and Hispanic youth. Additionally, nocturnal, urinary, and serum cortisol were available, however were not used due to small sample sizes. Finally, the CAR reading is based on only one day and may not be representative of the average CAR of the individual.

SSB consumption appears to be independently associated with higher VAT and higher CAR in overweight and obese minority youth. This study highlights SSB consumption as a potential moderator of VAT and CAR. Additionally, SSB is an attractive and highly modifiable target for interventions looking to decrease CAR and VAT in a minority youth population. Additionally, further work is needed to elucidate the possibility of CAR increasing desire for palatable foods, as well as the possibility of a positive feed back loop between SSB consumption, VAT accumulation, and CAR elevation.

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DSM and MJW were responsible for research design and oversaw all data collection. JND oversaw the dietary data collection. CMT performed quality control on all clinical and survey data. GES and MJD analyzed the data and performed statistical analysis. GES, JND, DSM, and MJW were involved in the data interpretation. GES, JND, DSM, CMT, MJD, and MJW provided edits for the paper. This study is supported by NCMHD grant P60MD002254 (MI Goran; M.J. Weigensberg for Project 1).

References

1. Bassett R, Chapman GE, Beagan BL. Autonomy and control: the co-construction of adolescent food choice. *Appetite*. 2008; 50:325–332. [PubMed: 17936413]
2. Singh AS, Mulder C, Twisk JWR, van Mechelen W, Chinapaw MJM. Tracking of childhood overweight into adulthood: a systematic review of the literature. *Obes. Rev.* 2008; 9:474–488. [PubMed: 18331423]
3. Ranjit N, Evans MH, Byrd-Williams C, Evans AE, Hoelscher DM. Dietary and activity correlates of sugar-sweetened beverage consumption among adolescents. *Pediatrics*. 2010; 126:e754–e761. [PubMed: 20876172]
4. Bucher Della Torre S, Keller A, Laure Depeyre J, Kruseman M. Sugar-Sweetened Beverages and Obesity Risk in Children and Adolescents: A Systematic Analysis on How Methodological Quality May Influence Conclusions. *J. Acad. Nutr. Diet.* 2015; 116:638–659. [PubMed: 26194333]
5. Dodd AH, Briefel R, Cabili C, Wilson A, Crepinsek MK. Disparities in consumption of sugar-sweetened and other beverages by race/ethnicity and obesity status among United States schoolchildren. *J. Nutr. Educ. Behav.* 45:240–249.
6. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011–2012. *JAMA*. 2014; 311:806–814. [PubMed: 24570244]
7. Malik VS, Popkin BM, Bray GA, Després J-P, Willett WC, Hu FB. Sugarsweetened beverages and risk of metabolic syndrome and type 2 diabetes: a metaanalysis. *Diabetes Care*. 2010; 33:2477–2483. [PubMed: 20693348]
8. Silveira LS, Monteiro PA, de M.M. Antunes B, Seraphim PM, Fernandes RA, Christofaro DGD, Freitas Júnior IF. Intra-abdominal fat is related to metabolic syndrome and non-alcoholic fat liver disease in obese youth. *BMC Pediatr.* 2013; 13:115. [PubMed: 23919592]
9. Odegaard AO, Choh AC, Czerwinski SA, Towne B, Demerath EW. Sugarsweetened and diet beverages in relation to visceral adipose tissue. *Obesity (Silver Spring)*. 2012; 20:689–691. [PubMed: 21901024]
10. Maersk M, Belza A, Stødkilde-Jørgensen H, Ringgaard S, Chabanova E, Thomsen H, Pedersen SB, Astrup A, Richelsen B. Sucrose-sweetened beverages increase fat storage in the liver, muscle, and visceral fat depot: a 6-mo randomized intervention study. *Am. J. Clin. Nutr.* 2012; 95:283–289. [PubMed: 22205311]
11. Hairston KG, Scherzinger A, Foy C, Hanley AJ, McCorkle O, Haffner S, Norris JM, Bryer-Ash M, Wagenknecht LE. Five-year change in visceral adipose tissue quantity in a minority cohort: the Insulin Resistance Atherosclerosis Study (IRAS) family study. *Diabetes Care*. 2009; 32:1553–1555. [PubMed: 19487643]
12. DellaValle DM, Roe LS, Rolls BJ. Does the consumption of caloric and non-caloric beverages with a meal affect energy intake? *Appetite*. 2005; 44:187–193. [PubMed: 15808893]
13. Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. *Am. J. Public Health*. 2007; 97:667–675. [PubMed: 17329656]
14. Gyllenhammer LE, Weigensberg MJ, Spruijt-Metz D, Allayee H, Goran MI, Davis JN. Modifying influence of dietary sugar in the relationship between cortisol and visceral adipose tissue in minority youth. *Obesity (Silver Spring)*. 2014; 22:474–481. [PubMed: 23929660]
15. Misra M, Bredella MA, Tsai P, Mendes N, Miller KK, Klibanski A. Lower growth hormone and higher cortisol are associated with greater visceral adiposity, intramyocellular lipids, and insulin resistance in overweight girls. *Am. J. Physiol. Endocrinol. Metab.* 2008; 295:E385–E392. [PubMed: 18544645]

16. Weigensberg MJ, Toledo-Corral CM, Goran MI. Association between the metabolic syndrome and serum cortisol in overweight Latino youth. *J. Clin. Endocrinol. Metab.* 2008; 93:1372–1378. [PubMed: 18252788]
17. Adam TC, Hasson RE, Ventura EE, Toledo-Corral C, Le K-A, Mahurkar S, Lane CJ, Weigensberg MJ, Goran MI. Cortisol is negatively associated with insulin sensitivity in overweight Latino youth. *J. Clin. Endocrinol. Metab.* 2010; 95:4729–4735. [PubMed: 20660036]
18. Toledo-Corral CM, Alderete TL, Hu HH, Nayak K, Esplana S, Liu T, Goran MI, Weigensberg MJ. Ectopic fat deposition in prediabetic overweight and obese minority adolescents. *J. Clin. Endocrinol. Metab.* 2013; 98:1115–1121. [PubMed: 23386647]
19. Toledo-Corral CM, Alderete TL, Richey J, Sequeira P, Goran MI, Weigensberg MJ. Fasting, post-OGTT challenge, and nocturnal free fatty acids in prediabetic versus normal glucose tolerant overweight and obese Latino adolescents. *Acta Diabetol.* 2015; 52:277–284. [PubMed: 25109287]
20. Toledo-Corral CM, Myers SJ, Li Y, Hodis HN, Goran MI, Weigensberg MJ. Blunted nocturnal cortisol rise is associated with higher carotid artery intima-media thickness (CIMT) in overweight African American and Latino youth. *Psychoneuroendocrinology.* 2013; 38:1658–1667. [PubMed: 23433749]
21. Rasmussen AR, Wohlfahrt-Veje C, Tefre de Renzy-Martin K, Hagen CP, Tinggaard J, Mouritsen A, Mieritz MG, Main KM. Validity of self-assessment of pubertal maturation. *Pediatrics.* 2015; 135:86–93. [PubMed: 25535262]
22. Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. *Arch. Dis. Child.* 1970; 45:13–23. [accessed September 5, 2015] <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2020414&tool=pmcentrez&rendertype=abstract>. [PubMed: 5440182]
23. Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. *Arch. Dis. Child.* 1969; 44:291–303. [accessed April 28, 2015] <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2020314&tool=pmcentrez&rendertype=abstract>. [PubMed: 5785179]
24. Chida Y, Steptoe A. Cortisol awakening response and psychosocial factors: A systematic review and meta-analysis. *Biol. Psychol.* 2009; 80:265–278. [PubMed: 19022335]
25. Hucklebridge F, Stalder T, Evans P, Thorn L. The cortisol awakening response: More than a measure of HPA axis function. *Neurosci. Biobehav. Rev.* 2010; 35:97–103. [PubMed: 20026350]
26. Clow A, Hucklebridge F, Thorn L. The cortisol awakening response in context. *Int. Rev. Neurobiol.* 2010; 93:153–175. [PubMed: 20970005]
27. Therrien F, Drapeau V, Lalonde J, Lupien SJ, Beaulieu S, Tremblay A, Richard D. Awakening Cortisol Response in Lean, Obese, and Reduced Obese Individuals: Effect of Gender and Fat Distribution*. *Obesity.* 2007; 15:377–385. [PubMed: 17299111]
28. Rudenga KJ, Sinha R, Small DM. Acute stress potentiates brain response to milkshake as a function of body weight and chronic stress. *Int. J. Obes.* 2012; 37:309–316.
29. Hu HH, Nayak KS, Goran MI. Assessment of abdominal adipose tissue and organ fat content by magnetic resonance imaging. *Obes. Rev.* 2011; 12:e504–e515. [PubMed: 21348916]
30. Hu HH, Kim H-W, Nayak KS, Goran MI. Comparison of fat-water MRI and singlevoxel MRS in the assessment of hepatic and pancreatic fat fractions in humans. *Obesity (Silver Spring).* 2010; 18:841–847. [PubMed: 19834463]
31. Alabousi A, Al-Attar S, Joy TR, Hegele RA, McKenzie CA. Evaluation of adipose tissue volume quantification with IDEAL fat-water separation. *J. Magn. Reson. Imaging.* 2011; 34:474–479. [PubMed: 21780238]
32. Reeder SB, Cruite I, Hamilton G, Sirlin CB. Quantitative Assessment of Liver Fat with Magnetic Resonance Imaging and Spectroscopy. *J. Magn. Reson. Imaging.* 2011; 34 spcone.
33. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. *J. Health Soc. Behav.* 1983; 24:385–396. [accessed May 24, 2016] <http://www.ncbi.nlm.nih.gov/pubmed/6668417>. [PubMed: 6668417]
34. Jonnalagadda S. Accuracy of Energy Intake Data Estimated by a Multiplepass, 24-hour Dietary Recall Technique. *J. Am. Diet. Assoc.* 2000; 100:303–311. [PubMed: 10719403]
35. Johnson RK, Appel LJ, Brands M, Howard BV, Lefevre M, Lustig RH, Sacks F, Steffen LM, Wylie-Rosett J. American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity, and Metabolism and the Council on Epidemiology and Prevention, Dietary

- sugars intake and cardiovascular health: a scientific statement from the American Heart Association. *Circulation*. 2009; 120:1011–1020. [PubMed: 19704096]
36. Tsai SL, Seiler KJ, Jacobson J. Morning cortisol levels affected by sex and pubertal status in children and young adults. *J. Clin. Res. Pediatr. Endocrinol*. 2013; 5:85–89. [PubMed: 23748059]
 37. DeSantis AS, Adam EK, Doane LD, Mineka S, Zinbarg RE, Craske MG. Racial/Ethnic Differences in Cortisol Diurnal Rhythms in a Community Sample of Adolescents. *J. Adolesc. Heal*. 2007; 41:3–13.
 38. Stimson RH, Johnstone AM, Homer NZM, Wake DJ, Morton NM, Andrew R, Lobley GE, Walker BR. Dietary macronutrient content alters cortisol metabolism independently of body weight changes in obese men. *J. Clin. Endocrinol. Metab*. 2007; 92:4480–4484. [PubMed: 17785367]
 39. Stimson RH, Mohd-Shukri NA, Bolton JL, Andrew R, Reynolds RM, Walker BR. The postprandial rise in plasma cortisol in men is mediated by macronutrient-specific stimulation of adrenal and extra-adrenal cortisol production. *J. Clin. Endocrinol. Metab*. 2014; 99:160–168. [PubMed: 24092834]
 40. Lemmens SG, Born JM, Martens EA, Martens MJ, Westerterp-Plantenga MS. Influence of consumption of a high-protein vs. high-carbohydrate meal on the physiological cortisol and psychological mood response in men and women. *PLoS One*. 2011; 6:e16826. [PubMed: 21304815]
 41. Ormsbee MJ, Kinsey AW, Eddy WR, Madzima TA, Arciero PJ, Figueroa A, Panton LB. The influence of nighttime feeding of carbohydrate or protein combined with exercise training on appetite and cardiometabolic risk in young obese women. *Appl. Physiol. Nutr. Metab. = Physiol. Appl. Nutr. Métabolisme*. 2015; 40:37–45.
 42. Micha R, Nelson M. Glycemic index and glycemic load used in combination to characterize metabolic responses of mixed meals in healthy lean young adults. *J. Am. Coll. Nutr*. 2011; 30:113–125. [accessed July 13, 2016] <http://www.ncbi.nlm.nih.gov/pubmed/21730219>. [PubMed: 21730219]
 43. Micha R, Rogers PJ, Nelson M. Glycaemic index and glycaemic load of breakfast predict cognitive function and mood in school children: a randomised controlled trial. *Br. J. Nutr*. 2011; 106:1552–1561. [PubMed: 21736777]
 44. Satchek JM, Rasmussen HM, Hall MM, Kafka T, Blumberg JB, Economos CD. The association between pregame snacks and exercise intensity, stress, and fatigue in children. *Pediatr. Exerc. Sci*. 2014; 26:159–167. [PubMed: 24091353]
 45. Tomiyama AJ, Dallman MF, Epel ES. Comfort food is comforting to those most stressed: evidence of the chronic stress response network in high stress women. *Psychoneuroendocrinology*. 2011; 36:1513–1519. [PubMed: 21906885]
 46. Born JM, Lemmens SGT, Rutters F, Nieuwenhuizen AG, Formisano E, Goebel R, Westerterp-Plantenga MS. Acute stress and food-related reward activation in the brain during food choice during eating in the absence of hunger. *Int. J. Obes. (Lond)*. 2010; 34:172–181. [PubMed: 19844211]
 47. Tryon MS, DeCant R, Laugero KD. Having your cake and eating it too: a habit of comfort food may link chronic social stress exposure and acute stress-induced cortisol hyporesponsiveness. *Physiol. Behav*. 2013; 114–115:32–37.
 48. Adam TC, Epel ES. Stress, eating and the reward system. *Physiol. Behav*. 2007; 91:449–458. [PubMed: 17543357]
 49. Warne JP. Shaping the stress response: Interplay of palatable food choices, glucocorticoids, insulin and abdominal obesity. *Mol. Cell. Endocrinol*. 2009; 300:137–146. [PubMed: 18984030]
 50. Zellner DA, Loaiza S, Gonzalez Z, Pita J, Morales J, Pecora D, Wolf A. Food selection changes under stress. *Physiol. Behav*. 2006; 87:789–793. [PubMed: 16519909]
 51. Rutters F, Nieuwenhuizen AG, Lemmens SGT, Born JM, Westerterp-Plantenga MS. Acute stress-related changes in eating in the absence of hunger. *Obesity (Silver Spring)*. 2009; 17:72–77. [PubMed: 18997672]
 52. Geer EB, Lalazar Y, Couto LM, Cohen V, Lipton LR, Shi W, Bagiella E, Conwell I, Bederson J, Kostadinov J, Post KD, Freda PU. A prospective study of appetite and food craving in 30 patients with Cushing's disease. *Pituitary*. 2016; 19:117–126. [PubMed: 26496766]

53. Ma J, Sloan M, Fox CS, Hoffmann U, Smith CE, Saltzman E, Rogers GT, Jacques PF, McKeown NM. Sugar-sweetened beverage consumption is associated with abdominal fat partitioning in healthy adults. *J. Nutr.* 2014; 144:1283–1290. [PubMed: 24944282]
54. Zheng M, Rangan A, Olsen NJ, Bo Andersen L, Wedderkopp N, Kristensen P, Grøntved A, Ried-Larsen M, Lempert SM, Allman-Farinelli M, Heitmann BL. Sugarsweetened beverages consumption in relation to changes in body fatness over 6 and 12 years among 9-year-old children: the European Youth Heart Study. *Eur. J. Clin. Nutr.* 2014; 68:77–83. [PubMed: 24281311]
55. Stanhope KL, Schwarz JM, Keim NL, Griffen SC, Bremer AA, Graham JL, Hatcher B, Cox CL, Dyachenko A, Zhang W, McGahan JP, Seibert A, Krauss RM, Chiu S, Schaefer EJ, Ai M, Otokozawa S, Nakajima K, Nakano T, Beysen C, Hellerstein MK, Berglund L, Havel PJ. Consuming fructose-sweetened, not glucosesweetened, beverages increases visceral adiposity and lipids and decreases insulin sensitivity in overweight/obese humans. *J. Clin. Invest.* 2009; 119:1322–1334. [PubMed: 19381015]
56. Mollard RC, Snchal M, MacIntosh AC, Hay J, Wicklow BA, Wittmeier KDM, Sellers EAC, Dean HJ, Ryner L, Berard L, McGavock JM. Dietary determinants of hepatic steatosis and visceral adiposity in overweight and obese youth at risk of type 2 diabetes. *Am. J. Clin. Nutr.* 2014; 99:804–812. [PubMed: 24522441]
57. Funtikova AN, Subirana I, Gomez SF, Fit M, Elosua R, Bentez-Arciniega AA, Schrder H. Soft drink consumption is positively associated with increased waist circumference and 10-year incidence of abdominal obesity in Spanish adults. *J. Nutr.* 2015; 145:328–334. [PubMed: 25644355]
58. Pollock NK, Bundy V, Kanto W, Davis CL, Bernard PJ, Zhu H, Gutin B, Dong Y. Greater fructose consumption is associated with cardiometabolic risk markers and visceral adiposity in adolescents. *J. Nutr.* 2012; 142:251–257. [PubMed: 22190023]
59. Walker RW, Dumke KA, Goran MI. Fructose content in popular beverages made with and without high-fructose corn syrup. *Nutrition.* 2014; 30:928–935. [PubMed: 24985013]
60. Lee M-J, Pramyothin P, Karastergiou K, Fried SK. Deconstructing the roles of glucocorticoids in adipose tissue biology and the development of central obesity. *Biochim. Biophys. Acta.* 2014; 1842:473–481. [PubMed: 23735216]
61. Fried SK, Russell CD, Grauso NL, Brolin RE. Lipoprotein lipase regulation by insulin and glucocorticoid in subcutaneous and omental adipose tissues of obese women and men. *J. Clin. Invest.* 1993; 92:2191–2198. [PubMed: 8227334]
62. Davis JN, Alexander KE, Ventura EE, Kelly LA, Lane CJ, Byrd-Williams CE, Toledo-Corral CM, Roberts CK, Spruijt-Metz D, Weigensberg MJ, Goran MI. Associations of dietary sugar and glycemic index with adiposity and insulin dynamics in overweight Latino youth. *Am J Clin Nutr.* 2007; 86:1331–1338. <http://www.ncbi.nlm.nih.gov/pubmed/17991643>. [PubMed: 17991643]

Highlights

- Sugar sweetened beverage intake is associated with higher visceral adipose tissue and cortisol awakening response.
- Cortisol awakening response is related to visceral adipose tissue in adolescents.
- Sugar sweetened beverage intake does not appear to influence the relationship between visceral adipose tissue and cortisol awakening response.

Fig. 1.

Mean cortisol awakening response adjusted for sex, BMI, tanner stage, ethnicity, and energy intake by sugar sweetened beverage consumption categories. Error bars represent plus and minus one standard error. * $p < 0.05$.

Fig. 2. Mean visceral adipose tissue adjusted for sex, BMI, tanner stage, ethnicity, and energy intake by sugar sweetened beverage consumption categories. Error bars represent plus and minus one standard error. * $p < 0.05$.

Table 1

Demographics, adiposity, cortisol, and dietary intake variables of study sample

	Total	Low SSB (<1serv/day)	Medium SSB (1 serv<2/day)	High SSB (2serv/day)
N	60	31	9	20
Sex (Female/Male)	28/32	14/17	4/5	10/10
Tanner (stage 4/stage 5)	14/46	10/21	2/7	2/18
Ethnicity (NHB/Hispanic)	18/42	11/20	1/8	6/14
BMI z-score	2.1±0.4	2.2±0.4	2.1±0.5	2.0±0.5
Visceral Adiposity (L)	1.9±1.0	1.8±1.0	1.9±1.1	2.1±1.1
Subcutaneous Adiposity (L)	8.4±3.5	8.5±3.3	8.3±4.5	8.1±3.5
CAR (µg/dl)	0.9±0.6	0.6±0.6	0.8±0.7	1.2±0.6
Energy Intake (kcal/day)	1710±608	1502±618	1753±506	2061±544

Data are presented as n or mean ± SD

Table 2
Analysis of covariance (ANCOVA) models for Cortisol Awakening Response (CAR) and Visceral Adipose Tissue (VAT).

	CAR			VAT		
	Beta	95% CI	P	Beta	95% CI	P
Model 1						
SSB Low		Reference category			Reference category	
SSB Medium	0.07	-0.48, 0.48	0.99	0.03	-0.17, 0.20	0.88
SSB High	0.30	0.02, 0.48	0.04	0.25	0.03, 0.33	0.02
Sex	-0.28	-0.70, -0.01	0.04	0.29	0.09, 0.36	<0.01
BMI	-0.08	-0.47, 0.22	0.48	0.53	0.30, 0.58	<0.01
Tanner	-0.05	-0.41, 0.42	0.96	0.03	-0.14, 0.18	0.76
Ethnicity	0.02	-0.29, 0.41	0.74	0.47	0.27, 0.55	<0.01
Energy Intake (kcal/day)	0.23	-0.14, 0.71	0.19	-0.22	-0.31, 0.02	0.08
Perceived stress	0.27	-0.11, 0.51	0.20	-0.07	-0.15, 0.09	0.65
Tukey adjusted means						
SSB High-Low		-0.50, 0.61	0.09		-0.01, 0.36	0.05
SSB High-Medium		-0.19, 0.94	0.23		-0.06, 0.40	0.21
SSB Medium-Low		-0.50, 0.61	1.00		-0.21, 0.24	0.99
Model 2						
SSB Low		Reference category				
SSB Medium	-0.01	-0.47, 0.45	0.96	0.03	-0.16, 0.19	0.88
SSB High	0.21	-0.13, 0.66	0.18	0.19	-0.02, 0.28	0.09
Sex	-0.37	-0.91, -0.16	<0.01	0.35	0.13, 0.41	<0.01
BMI	-0.33	-0.93, -0.02	0.03	0.59	0.32, 0.59	<0.01
Tanner	-0.01	-0.41, 0.39	0.95	0.02	-0.13, 0.18	0.76
Ethnicity	-0.17	-0.71, 0.16	0.22	0.48	0.27, 0.53	<0.01
Energy Intake (kcal/day)	0.21	-0.02, 0.82	0.06	-0.34	-0.34, -0.02	0.02
Perceived stress	0.17	-0.07, 0.52	0.14	-0.07	-0.17, 0.06	0.37
VAT	1.10	0.12, 1.48	0.02			
CAR				0.31	0.01, 0.23	0.02
Interaction terms						
SSB*VAT	-0.30	-1.39, 0.54	0.66			

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SSB*CAR	CAR			VAT		
	Beta	95% CI	P	Beta	95% CI	P
				0.09	-0.11, 0.24	0.12

Significant value p 0.05;

^a Sample size=60