Published in final edited form as:

Physiol Behav. 2012 July 16; 106(5): 651–656. doi:10.1016/j.physbeh.2012.04.027.

Associations between weight-related eating behaviors and adiposity in postmenopausal Japanese American and white women

Susan M. Schembre, PhD, RD^a, Cheryl L. Albright, MPH, PhD^{b,c}, Unhee Lim, PhD^d, Lynne R. Wilkens, DrPH^d, Suzanne P. Murphy, PhD, RD^d, Rachel Novotny, PhD, RD^{d,e}, Thomas Ernst, PhD^f, Linda Chang, MD^f, Laurence N. Kolonel, MD, PhD^d, and Loïc Le Marchand, MD, PhD^d

^aDepartment of Preventive Medicine, Keck Medical Center of University of Southern California, Honolulu, Hawaii, USA

^bPrevention and Control, University of Hawaii Cancer Center, Honolulu, Hawaii, USA

^cSchool of Nursing and Dental Hygiene, University of Hawaii, Honolulu, Hawaii, USA

^dEpidemiology Programs, University of Hawaii Cancer Center, Honolulu, Hawaii, USA

^eDepartment of Human Nutrition, Food, and Animal Sciences, University of Hawaii, Honolulu, Hawaii, USA

^fDepartment of Medicine, John A. Burns School of Medicine, University of Hawaii, Honolulu, Hawaii, USA

Abstract

The purpose of this study was to test the associations between cognitive and psychological eating behavior traits and detailed measures of adiposity and body fat distribution using imaging-based methods in a cross-sectional study. Eating behavior traits (compensatory and routine restraint, external eating, and emotional eating) were assessed using the validated Weight-Related Eating Questionnaire, and measures of adiposity using anthropometry, dual energy X-ray absorptiometry (DXA), and magnetic resonance imaging (MRI). Each adiposity outcome of interest (total fat, ratio of trunk fat to periphery fat, visceral and subcutaneous fat as % of abdominal area, and % liver fat) was regressed on the four eating behaviors while adjusting for age and race/ethnicity. This study included a total of 60 postmenopausal Japanese American (n=30) and white (n=30) women (age: 60–65y, BMI: 18.8–39.6 kg/m²). Weight-related eating behavior traits did not differ by ethnicity. Higher external eating scores were associated with measures of total adiposity, including higher BMI ($\beta = 0.36$, $p = 0.02$) and DXA total fat mass ($\beta = 0.41$, $p = 0.001$), and with MRI abdominal subcutaneous fat ($\beta = 0.55$, $p = 0.001$). Higher routine restraint scores were associated with visceral adiposity ($\beta = 0.42$, $p = 0.04$). Our findings suggest that different weight-related eating behavior traits might increase not only total adiposity but also abdominal and visceral fat deposition associated with higher metabolic risks. Future research, preferably in a

© 2012 Elsevier Inc. All rights reserved.

Corresponding author: Susan M. Schembre, Department of Preventive Medicine, Keck Medical Center of University of Southern California, 2001 North Soto Street, Suite 312, Los Angeles, California, 90033, phone: 1-323-442-7247, fax: 1-323-442-7254, schembre@usc.edu.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

prospective study of men and women and including biomarkers related to psychological stress, will be needed to explore potential underlying biological mechanisms.

Keywords

Eating Behaviors; Body Fat Distribution; Central Obesity; Liver Fat; Subcutaneous Adipose Tissue; Visceral Fat

1.0 Introduction

Body fat deposition in the abdominal region is a known predictor of cardiovascular disease and diabetes [1], as well as some obesity-related cancers [2, 3]. Compared to peripheral or subcutaneous abdominal adiposity, intra-abdominal visceral adiposity has a greater negative impact on health outcomes, with recent evidence suggesting several biological differences between subcutaneous and visceral abdominal adiposity [4]. While the tendency for some individuals to disproportionately store body fat in the abdomen might be influenced by race/ethnicity or genetic traits [5, 6], there is a need to explore other, potentially modifiable, behavioral characteristics that contribute to elevated chronic disease and cancer risk related to increases in abdominal adiposity [4].

Much of the existing eating behavior research related to obesity has focused on identifying specific components of the diet (e.g., sugar sweetened beverages, dietary fat) and/or dietary patterns (e.g., breakfast skipping, fast food consumption) that might promote excessive weight gain [7-9]. Fewer studies have explored the role in obesity or central adiposity of cognitive and psychological aspects of eating behavior traits, which have been demonstrated to develop in childhood and persist through adulthood [10-14]. There are three theory-based constructs that describe these aspects of eating behavior. Dietary restraint [15], which reflects a rigid approach to weight control (routine restraint) as in chronic dieting, and a flexible approach to weight control (compensatory restraint), such as consciously eating more or less around times of overeating [16-18]. External eating [19] refers to the susceptibility to eat in response to the hedonic properties of food or other social/environmental influences. Emotional eating [20] reflects eating that occurs in response to negative affect. These eating behaviors, particularly external and emotional eating, have been associated with high-fat, high-sugar patterns of dietary intake [21-23] related to higher levels of adiposity, and might contribute to explaining differences in body fat distribution [24].

To date, studies on eating behavior traits and obesity have been almost exclusively based on crude measures of adiposity (e.g., body mass index (BMI) and/or waist circumference) and, therefore, have limitations of potentially misclassifying total or abdominal adiposity. Furthermore, eating behaviors could influence body fat accumulation in the abdominal versus peripheral region or in the visceral versus subcutaneous compartment of the abdomen. For example, chronic dietary restraint, characterized by dieting and food avoidance has been associated with higher perceived stress, measured by higher levels of cortisol in saliva [25] or urine [26, 27]. Also, increases in cortisol levels have also been positively associated with subsequent consumption of energy-dense foods [28-30] and fat deposition in the visceral compartment [31]. To our knowledge, only one prior study has examined the associations between eating behavior traits and image-based direct measures of abdominal and intra-abdominal fat distribution [24]. Provencher et al. [24], using the Three Factor Eating Questionnaire, demonstrated that flexible restraint had a negative correlation, and, rigid restraint and disinhibition, which encompasses external and emotional eating, had a positive correlation with greater total, abdominal, and visceral and

subcutaneous adiposity in the abdomen. These findings provided support that theory-based eating behaviors might contribute to explaining the distribution of total and abdominal adiposity.

The purpose of our study was to test the association of eating behaviors not only with total body fat but also with regional body fat distribution adjusted for total adiposity, using imaging-based methods of dual energy X-ray absorptiometry (DXA) and magnetic resonance imaging (MRI) scans. The analyses utilize data collected for a pilot study exploring the differences in adiposity and abdominal fat distribution between Asian American and white women. The study was conducted to investigate prior observations that Japanese American women showed higher risks for breast cancer [32] and diabetes [33] compared to whites at any given BMI level even after accounting for other risk factors. It was hypothesized that differences in body fat distribution might explain the heterogeneity in disease risk. Findings from the pilot study confirmed that Asian women have greater abdominal and visceral adiposity than white women with similar body mass indexes [34]. The sample size of 30 per group was selected as the minimum required to detect the large differences in fat distribution between Japanese and white women expected based on preliminary data.

Based on previous literature, various eating behaviors are associated with both increased adiposity and biomarkers of acute and chronic psychological stress. It was, therefore, hypothesized that routine restraint, external eating and emotional eating would be associated with greater total adiposity, whereas compensatory restraint would be associated with lower total adiposity. We also hypothesized that routine restraint and emotional eating behaviors may be related to greater visceral as opposed to subcutaneous adiposity.

2.0 Participants and methods

2.1 Participants

White (n=30) and Japanese American (n=30) postmenopausal women living in Hawaii, age 60-65 years were recruited from the ongoing Multiethnic Cohort Study. Details of recruitment and data collection methods are fully described elsewhere [34]. Briefly, recruitment was stratified by race/ethnicity and BMI categories based on self-reported weight and height. Exclusion criteria included: current or recent smoking (<2 years); use of medications that may interfere with metabolism and body composition (chemotherapy, insulin or weight-loss drugs); substantial weight change (loss or gain of 20 pounds or more) in the past 6 months; pre- or perimenopausal status (had menses in the past 12 months); a measured BMI outside the target range (18.5–40 kg/m²); and a report of any soft or metal implants/objects in the body that could bias the body composition estimates or put subjects at risk in the MRI magnetic field. The study was approved by the Institutional Review Boards of the University of Hawaii and the Queen's Medical Center, Honolulu, and all participants signed an informed consent.

2.2 Body composition measures

As described in detail in the original publication [34], subjects fasted overnight for an early-morning examination that included anthropometry measurements, DXA/MRI scans and fasting blood collection. All the participants wore a hospital gown for the anthropometry and DXA examinations, and comfortable shirts and pants without any metal parts for the MRI scan.

Anthropometric measures including height, weight, and waist circumference taken at the navel were collected using standardized protocols. Each measurement was taken twice, and

a third time if the two measures differed by >0.1 kg for weight or by >0.5 cm for height or waist, after which the average of the two closest values was used in the analysis [34].

DXA was used to measure total and regional fat mass in the trunk, arms and legs. DXA measurements were completed by a certified radiographic technician using a whole-body DXA scan (GE Lunar Prodigy, Madison, WI, USA) [35]. Calibration using a manufacturer's phantom was performed daily. Measures of total and regional body fat mass in the trunk, arms, and legs were used to calculate trunk-to-periphery fat mass ratio (fat mass in the trunk divided by the sum of fat mass in the arms and legs).

Abdominal MRI scans were additionally conducted to determine the trunk fat distribution in visceral, subcutaneous and hepatic compartments, which cannot be attained directly using DXA. MRI scans were obtained at a separate appointment from 20 Japanese American and 28 white women among the 60 participants. Due to scheduling limitations, the time between DXA and MRI measurements were within one to thirteen weeks (median 7.6 weeks). However, the body fat distributions based on DXA were found to be similar for women whose visits were close in time and those with visits spaced further apart. MRI was performed in a single session on a research-dedicated 3 Tesla TIM Trio scanner (Siemens Medical Systems, Erlangen, Germany; software version VB13). Details of the MRI protocol have been reported [34]. The abdominal and peritoneal walls were manually traced to measure the total fat area (mm^2) and the visceral fat area (intraperitoneal, including fat inside internal organs), respectively, at the L4-L5 lumbar spine position [34]. The visceral fat area was then subtracted from total fat area to obtain the subcutaneous fat area (extraperitoneal). Percent liver fat was measured in a circular region of interest ($15\text{--}25\text{ cm}^2$) in the lateral portion of the right lobe of the liver [36].

2.3 Eating behaviors

Four theory-based, cognitive/psychological aspects of eating behavior labeled compensatory restraint, routine restraint, susceptibility to external cues, and emotional eating were assessed with the 16-item Weight-Related Eating Questionnaire (WREQ) [17], which has been validated as an online survey in a diverse sample of males and females confirming its unbiased generalizability across gender, age (young adults, 18-24 years and adults, 25-81 years), race (Japanese Americans and whites), and BMI subgroups (under/normal weight and overweight/obese) [37]. Routine restraint (3 items) and compensatory restraint (3 items) reflect two subscales of cognitive dietary restraint that represent rigid and flexible approaches to weight control, respectively. Routine restraint is defined as the perceived routine restriction of energy intake to control weight by means of calorie counting, food avoidance/refusal, and purposefully intending to eat less than normal, consistent with dieting. Alternatively, compensatory restraint refers to an intentional restriction of energy intake that predominantly occurs before or following an episode of overeating. External eating (5 items) refers to eating that occurs in response to external oro-sensory cues (e.g., social and environmental cues or hedonic properties of food). Emotional eating (5 items) is defined as eating in response to negative emotions (e.g., anxiety, stress, and disappointment). Subscale scores are the average of responses on a five-point scale: 1 to 5 assigned to responses ("not at all, slightly, more or less, pretty well, completely") to how each item "describes" the participant's eating behavior. In a previously published validation study conducted in a diverse population [37], the subscales of the WREQ have shown good internal consistency (Cronbach's $\alpha = 0.67$ to 0.91 ; four-week test-retest reliability coefficients $r = 0.74$ to 0.86). In the current study, Cronbach's α ranged from 0.72 (routine restraint) to 0.90 (emotional eating). Each participant completed the WREQ following their body composition testing in paper and pen format while at the clinic. The study coordinator was available to aid with the questionnaires; however, none of the participants reported having difficulty completing the WREQ.

2.4 Statistical analyses

All analyses were conducted using the SAS Statistical Software, version 9.2 (SAS Institute, Inc., Cary, NC, USA). Demographics, body composition, and eating behavior characteristics were compared between Japanese American and white women using analysis of covariance. To account for correlations among the WREQ subscales ($r = 0.17$ to 0.69) [17, 37], each body composition measure was regressed on the four continuous eating behavior variables. All models were adjusted for age and race/ethnicity. The addition of height had little influence on the observed associations, and this variable was not included in the final regression models. In separate models, interaction terms (race/ethnicity-by-eating behaviors) were included to determine whether the associations with body composition variables varied between Japanese American women and white women. Regression diagnostics were performed to confirm that model assumptions were met. Only % liver fat required log transformation and is presented as geometric means in tables. Participants missing MRI-specific measures ($n=12$) were excluded from analyses with these outcomes. Significance was defined as $p < 0.05$.

3.0 Results

The women in this study (50% white, 50% Japanese American) had, on average, an age of 63.4 ± 1.4 years, a weight of 66.3 ± 5.3 kg, a height of 157.5 ± 5.3 cm, and a BMI of 26.7 ± 4.9 kg/m². By study design, there were no race/ethnicity differences on measures of total adiposity as measured by DXA. However, after adjusting for age, height, and total body fat, Japanese American women had significantly greater measures of abdominal adiposity (waist circumference, trunk fat mass, and trunk-to-periphery fat ratio), and of visceral fat (percent of abdominal area), but no statistically significant differences were found in subcutaneous fat or percent liver fat (Table 1), as reported earlier in detail [34]. There were no race/ethnic differences in mean eating behavior scores, and scores for each subscale were similar to our previous findings in these populations (Table 2) [37].

Multiple regression analyses (Table 3) demonstrated that after adjusting for age and race/ethnicity, higher external eating scores were associated with significantly greater BMI, waist circumference, total body fat, and trunk fat, as well as greater abdominal subcutaneous fat. Also, higher routine restraint scores were positively associated with greater visceral fat. There was the suggestion of an inverse association between compensatory restraint scores and trunk-to-periphery fat ratio ($p=0.08$) and % visceral fat ($p=0.07$); however, there was no association between emotional eating scores and body fat distribution. All associations were consistent between Japanese American women and white women (data not shown, p 's for interaction > 0.05).

4.0 Discussion

The purpose of this study was to test the associations of the four weight-related eating behavior traits, assessed by the validated WREQ, to detailed measures of total, abdominal and intra-abdominal adiposity. Higher external eating scores were associated with higher measures of total adiposity (greater BMI and total fat) and abdominal adiposity (waist circumference and trunk fat), particularly subcutaneous abdominal adiposity, after adjusting for total adiposity. Higher routine restraint scores were positively associated with greater visceral adiposity. Additionally, there were non-significant suggestions that compensatory restraint scores were negatively associated with trunk-to-periphery fat and visceral adiposity. Emotional eating scores were not significantly associated with adiposity or abdominal adiposity distribution. Although further research is needed to replicate these findings, preferably using a prospective study design, they suggest that different weight-related eating

behavior traits influence the deposition of adipose tissue in a manner that could negatively impact disease risk in some populations.

The current study is similar to one conducted by Provencher et al. [24], which used the Three Factor Eating Questionnaire (TFEQ) [38, 39] for the associations of eating behaviors with direct measures of visceral and subcutaneous adiposity. Among 352 middle-aged women (age 42.0 ± 14.6 years; BMI 28.8 ± 8.2 kg/m²), they observed significant and positive bivariate correlations of “rigid restraint” (equivalent to our “routine restraint”) or disinhibition (combining our “external eating” and “emotional eating”) with both visceral and subcutaneous fat areas [39] (r 's = 0.12 to 0.49, $p < 0.05$). Differences between the Provencher study and ours is likely due to different categorizations in the instruments and also different statistical modeling. The WREQ separately assesses two, moderately correlated constructs of external and emotional eating, whereas the TFEQ assessed disinhibition that combined situational and emotional eating subscales [17, 18, 37]. Also, we assessed multivariable-adjusted independent associations for each eating behavior, whereas Provencher et al. examined bivariate Spearman correlations. As a result, final categorization we were able to show an independent association of routine restraint with visceral adiposity, and of external eating with subcutaneous adiposity. Also, we observed a stronger association for the relative compared to the absolute visceral or subcutaneous fat area, which supports independent associations for either adipose compartment. As such, this study makes a unique contribution by providing more detailed information for the associations between eating behaviors and measures of adiposity.

As discussed above, we found that external eating was positively associated with measures of adiposity, including greater BMI, total body fat, waist circumference, and trunk fat, but not visceral adiposity. These associations were hypothesized based on Schachter's theory of externality [40], which states that some individuals are at risk of obesity as a result of the susceptibility to eat in response to cues in the external environment (e.g., food-related cues) more so than internal cues (e.g., physiological states of hunger and fullness). However, empirical support for the associations between external eating and measures of total adiposity, particularly BMI, has not been consistent [41, 42]. In two validation studies of the WREQ, one with 721 university students (70% female, age 18.9 ± 1.1 years; BMI 23.1 ± 3.5 kg/m²) and another with 621 adults (61% female; age 34.8 ± 16.6 years; BMI 25.7 ± 6.2 kg/m²), the relationships between external eating and current BMI as well as weight change rate were not significant [17, 37]. These findings have not been limited to the WREQ as similarly null associations have been observed between external or uncontrolled eating and BMI using the Dutch Eating Behavior Questionnaire and the revised 18-item Three Factor Eating Questionnaire, respectively [17, 43, 44]. The reasons for observing significant associations between external eating and measures of adiposity in this study and not others are unclear. Future research will be needed to explore possible moderators and mediators of this relationship.

Our observations are consistent with the hypothesis that restrained eating (i.e., routine restraint) may stimulate, or coincide with, cortisol secretion and the sympathetic nervous system, and thus may be conducive to visceral adipogenesis [45], whereas external eating may occur without stress responses and thus, increase overall, mostly subcutaneous, adiposity. Routine restraint reflects a more rigid approach to weight control, and the chronic aspects of dieting have been associated with depression, stress, and issues of self-esteem [46]. Measures of dietary restraint are consistently demonstrated to be positively associated with higher urinary cortisol excretion in pre- and post-menopausal women [25-27, 47], as well as higher BMI and weight gain [48]. On the other hand, rigid eating or routine restraint may simply be a confounding trait among people who are susceptible to stress and cortisol responses, and who, thus, develop more visceral adiposity in response to the higher

concentration of vagus nerve ending stimuli in the visceral than subcutaneous adipose tissue [49]. However, even in such a case, modifying routine restraint eating may be beneficial to mitigate the cycle of overeating and stress. We also observed inverse associations between compensatory restraint and measures of adiposity, though the associations for trunk-to-peripheral fat and visceral adiposity were only of borderline significance. These findings are consistent with the literature on dietary restraint that suggests that using a flexible approach towards weight control protects against weight gain [24, 50]. Published findings have also shown that compensatory restraint is associated with greater fruit and vegetable consumption in young adults [17] and is consistent with trying to prevent weight gain, as well as trying to lose weight [37]. Although these findings may be confounded by a personality trait that affects both the eating behaviors and adipogenicity, they suggest that future interventions may be targeted to curb the detrimental effects of rigid and chronic dieting, as well as to encourage more flexible approaches to eating and weight control.

There was little evidence that emotional eating was associated with abdominal adiposity: although our study's power was suboptimal for detection of modest associations, the slopes for this relationship in the regression models are close to null. To our knowledge, there are no other studies that have explored the relationship between emotional eating scores with image-based direct measures of abdominal adiposity. However, associations between emotional eating and measures of BMI, waist circumference, body fat, and/or weight change are generally positive in adult populations [14, 17, 37, 51-53]. Reasons for this attenuated association in our sample are unknown, but it may be related to the specificity of the sampled population of postmenopausal women. The mean emotional eating scores were low, suggesting that few women in this sample would describe themselves as susceptible to eating in response to negative affect. However, the mean emotional eating score among the mature women in the current study (1.96) were only slightly lower than that observed among younger women (2.13) [37]. Replication of the findings will be important in clarifying this association. On the other hand, episodes of stress, which could initiate an episode of emotional eating, are known to induce acute increases in cortisol [28, 29], which might have only a small and transient influence on visceral adiposity [29].

This study adds to the very limited research exploring the association between the cognitive/psychological aspects of eating behavior with detailed body composition measures. One of the strengths of our study was the stratified recruitment that ensured equal distribution of BMI within race/ethnic groups. With the balanced distribution for total adiposity between whites and Japanese Americans, we were able to show that the associations between eating behavior and adiposity were similar, although the latter group has proportionately greater abdominal and visceral fat. Also, the use of the WREQ and multivariable-adjusted modeling allowed us to distinguish the effects of two different weight control strategies (compensatory and routine restraint) as well as of external and emotional disinhibition. To our knowledge, the current study is among the first to demonstrate that the eating style can account for differences in the deposition of abdominal adipose tissue.

Despite the strengths of the study, generalizability of our findings is limited to postmenopausal women of ages 60 to 65 and will need to be replicated in other populations. Like the previous study by Provencher et al. [24], we used a cross-sectional design and, thus, lack evidence for the temporal sequence between eating behaviors and adiposity development. However, considering the cumulated research that demonstrated the early development and stability of eating behavior traits from childhood [10, 13], as evidenced by an association of early eating behaviors with long-term weight gain [14], our results add to the supportive evidence that certain eating behaviors may be modified to prevent total, abdominal or visceral adiposity. Our sample size was small, which may have limited our ability to detect modest associations as statistically significant (e.g., emotional eating and

adiposity) and our ability to explore any moderation effects of race on the association between eating behaviors and measures of adiposity, though no modification by race was hypothesized. Yet, we were able to observe several significant and potentially important associations in the current and the previously published original study analyses [34]. In addition, the WREQ is a self-reported, subjective assessment of the cognitive/psychological aspects of eating behavior. Some individuals may be unaware of eating episodes that are motivated by external (social/environmental cues or hedonic properties of palatable foods) or emotional cues [37]; however, this random misclassification would most likely have attenuated rather than strengthened the observed associations. Lastly, other factors could have influenced both adiposity and eating behaviors, and thus could have confounded the association, such as the independent effect of stress. Although other factors, such as physical activity and dietary composition, might be correlated with both eating behaviors and adiposity distribution, we purposefully did not control for them as it is believed that they would be intermediaries in the pathway from eating behavior to adiposity and adjustment would unduly attenuate the relationships of interest.

5.0 Conclusions

Our findings suggest that several body weight-related eating behavior traits were associated with measures of body fat amount and distribution. Future research will be needed to replicate these findings prospectively in a larger and more diverse sample of people, including both sexes and across broader age and race/ethnicity groups. In addition, the assessment of biomarkers related to stress (e.g., cortisol) and inflammation would help to understand the underlying physiological mechanisms linking eating behavior traits to the distribution of abdominal adipose tissue.

Acknowledgments

Author SMS was supported by National Cancer Institute postdoctoral fellowship grants (R25 CA90956 and T32 CA009492) during the preparation of this manuscript. Funding for this study was provided in part by the University of Hawaii Cancer Center (SPM, RN, LLM), as well as by the National Cancer Institute for the Multiethnic Cohort (R37 CA54281: LNK), the National Institutes of Health for the UH-QMC MR Research Center (5P20-RR11091 and G12-RR003061: TE and LC) and the National Center for Research Resources at National Institute of Health for the University of Hawaii Clinical Research Center (P20 RR11091). We thank the study participants and the dedicated staff at the University of Hawaii Cancer Center (Karin Koga, Eugene Okiyama, Naomi Hee, Janice Nako-Piburn, Janine Abe, Wileen Mau, Maj Earle), Clinical Research Center (Sara Murakami, Jane Yakuma, Patty Iwamoto) and the MR Research Center (Carol Kosaki).

References

- [1]. Hajer GR, van Haften TW, Visseren FL. Adipose tissue dysfunction in obesity, diabetes, and vascular diseases. *European heart journal*. 2008; 29:2959–71. [PubMed: 18775919]
- [2]. Lahmann PH, Cust AE, Friedenreich CM, Schulz M, Lukanova A, Kaaks R, et al. Anthropometric measures and epithelial ovarian cancer risk in the European Prospective Investigation into Cancer and Nutrition. *Int J Cancer*. 2010; 126:2404–15. [PubMed: 19821492]
- [3]. Harris HR, Willett WC, Terry KL, Michels KB. Body fat distribution and risk of premenopausal breast cancer in the Nurses' Health Study II. *J Natl Cancer Inst*. 2011; 103:273–8. [PubMed: 21163903]
- [4]. Hamdy O, Porramatikul S, Al-Ozairi E. Metabolic obesity: the paradox between visceral and subcutaneous fat. *Curr Diabetes Rev*. 2006; 2:367–73. [PubMed: 18220642]
- [5]. Wang K, Li WD, Zhang CK, Wang Z, Glessner JT, Grant SF, et al. A genome-wide association study on obesity and obesity-related traits. *PLoS One*. 2011; 6:e18939. [PubMed: 21552555]
- [6]. Ledoux T, Watson K, Baranowski J, Tepper BJ, Baranowski T. Overeating styles and adiposity among multiethnic youth. *Appetite*. 2010; 56:71–7. [PubMed: 21115080]

- [7]. Du H, Feskens E. Dietary determinants of obesity. *Acta Cardiol.* 2010; 65:377–86. [PubMed: 20821929]
- [8]. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr.* 2006; 84:274–88. [PubMed: 16895873]
- [9]. Mesas AE, Munoz-Pareja M, Lopez-Garcia E, Rodriguez-Artalejo F. Selected eating behaviours and excess body weight: a systematic review. *Obes Rev.* 2012; 13:106–35. [PubMed: 21955734]
- [10]. Webber L, Hill C, Saxton J, Van Jaarsveld CH, Wardle J. Eating behaviour and weight in children. *Int J Obes (Lond).* 2009; 33:21–8. [PubMed: 19002146]
- [11]. Gallant AR, Tremblay A, Perusse L, Bouchard C, Despres JP, Drapeau V. The Three-Factor Eating Questionnaire and BMI in adolescents: results from the Quebec family study. *Br J Nutr.* 2010; 104:1074–9. [PubMed: 20447324]
- [12]. Hays NP, Bathalon GP, McCrory MA, Roubenoff R, Lipman R, Roberts SB. Eating behavior correlates of adult weight gain and obesity in healthy women aged 55–65 y. *Am J Clin Nutr.* 2002; 75:476–83. [PubMed: 11864852]
- [13]. Wardle J, Marsland L, Sheikh Y, Quinn M, Fedoroff I, Ogden J. Eating style and eating behaviour in adolescents. *Appetite.* 1992; 18:167–83. [PubMed: 1510461]
- [14]. Hays NP, Roberts SB. Aspects of eating behaviors “disinhibition” and “restraint” are related to weight gain and BMI in women. *Obesity.* 2008; 16:52–8. [PubMed: 18223612]
- [15]. Herman CP, Mack D. Restrained and unrestrained eating. *J Pers.* 1975; 43:647–60. [PubMed: 1206453]
- [16]. Westenhofer J, Stunkard AJ, Pudel V. Validation of the flexible and rigid control dimensions of dietary restraint. *Int J Eat Disord.* 1999; 26:53–64. [PubMed: 10349584]
- [17]. Schembre S, Greene G, Melanson K. Development and validation of a weight-related eating questionnaire. *Eating behaviors.* 2009; 10:119–24. [PubMed: 19447354]
- [18]. Schembre, SM. Weight-Related Eating Behavior Questionnaires: Applying Theory to Measurement. In: Preedy, VR.; Watson, RR.; Martin, CR., editors. *Handbook of behavior, food and nutrition.* Springer; New York: 2011. p. 3487–506.
- [19]. Schachter S, Goldman R, Gordon A. Effects of fear, food deprivation, and obesity on eating. *J Pers Soc Psychol.* 1968; 10:91–7. [PubMed: 5725907]
- [20]. Bruch H. Psychological Aspects of Overeating and Obesity. *Psychosomatics.* 1964; 5:269–74. [PubMed: 14235740]
- [21]. Lindroos AK, Lissner L, Mathiassen ME, Karlsson J, Sullivan M, Bengtsson C, et al. Dietary intake in relation to restrained eating, disinhibition, and hunger in obese and nonobese Swedish women. *Obes Res.* 1997; 5:175–82. [PubMed: 9192390]
- [22]. de Lauzon B, Romon M, Deschamps V, Lafay L, Borys JM, Karlsson J, et al. The Three-Factor Eating Questionnaire-R18 is able to distinguish among different eating patterns in a general population. *J Nutr.* 2004; 134:2372–80. [PubMed: 15333731]
- [23]. Keskitalo K, Tuorila H, Spector TD, Cherkas LF, Knaapila A, Kaprio J, et al. The Three-Factor Eating Questionnaire, body mass index, and responses to sweet and salty fatty foods: a twin study of genetic and environmental associations. *Am J Clin Nutr.* 2008; 88:263–71. [PubMed: 18689360]
- [24]. Provencher V, Drapeau V, Tremblay A, Despres JP, Lemieux S. Eating behaviors and indexes of body composition in men and women from the Quebec family study. *Obes Res.* 2003; 11:783–92. [PubMed: 12805400]
- [25]. Putterman E, Linden W. Cognitive dietary restraint and cortisol: importance of pervasive concerns with appearance. *Appetite.* 2006; 47:64–76. [PubMed: 16624445]
- [26]. Rideout CA, Linden W, Barr SI. High cognitive dietary restraint is associated with increased cortisol excretion in postmenopausal women. *J Gerontol A Biol Sci Med Sci.* 2006; 61:628–33. [PubMed: 16799147]
- [27]. McLean JA, Barr SI, Prior JC. Cognitive dietary restraint is associated with higher urinary cortisol excretion in healthy premenopausal women. *Am J Clin Nutr.* 2001; 73:7–12. [PubMed: 11124742]
- [28]. George SA, Khan S, Briggs H, Abelson JL. CRH-stimulated cortisol release and food intake in healthy, non-obese adults. *Psychoneuroendocrinology.* 2010; 35:607–12. [PubMed: 19828258]

- [29]. Raspopow K, Abizaid A, Matheson K, Anisman H. Psychosocial stressor effects on cortisol and ghrelin in emotional and non-emotional eaters: influence of anger and shame. *Horm Behav.* 2010; 58:677–84. [PubMed: 20540943]
- [30]. Arce M, Michopoulos V, Shepard KN, Ha QC, Wilson ME. Diet choice, cortisol reactivity, and emotional feeding in socially housed rhesus monkeys. *Physiol Behav.* 2010; 101:446–55. [PubMed: 20670639]
- [31]. Anagnostis P, Athyros VG, Tziomalos K, Karagiannis A, Mikhailidis DP. Clinical review: The pathogenetic role of cortisol in the metabolic syndrome: a hypothesis. *J Clin Endocrinol Metab.* 2009; 94:2692–701. [PubMed: 19470627]
- [32]. White KK, Park SY, Kolonel LN, Henderson BE, Wilkens LR. Body size and breast cancer risk: The multiethnic cohort. *Int J Cancer.* 2011
- [33]. Maskarinec G, Erber E, Grandinetti A, Verheus M, Oum R, Hopping BN, et al. Diabetes incidence based on linkages with health plans: the multiethnic cohort. *Diabetes.* 2009; 58:1732–8. [PubMed: 19258435]
- [34]. Lim U, Ernst T, Buchthal SD, Latch M, Albright CL, Wilkens LR, et al. Asian women have greater abdominal and visceral adiposity than Caucasian women with similar body mass index. *Nutrition and Diabetes.* In press.
- [35]. Glickman SG, Marn CS, Supiano MA, Dengel DR. Validity and reliability of dual-energy X-ray absorptiometry for the assessment of abdominal adiposity. *J Appl Physiol.* 2004; 97:509–14. [PubMed: 15075304]
- [36]. Guiu B, Loffroy R, Petit JM, Aho S, Ben Salem D, Masson D, et al. Mapping of liver fat with triple-echo gradient echo imaging: validation against 3.0-T proton MR spectroscopy. *Eur. Radiol.* 2009; 19:1786–93. [PubMed: 19247667]
- [37]. Schembre SM, Geller KS. Psychometric properties and construct validity of the Weight-Related Eating Questionnaire in a diverse population. *Obesity.* 2011; 19:2336–44. [PubMed: 21546931]
- [38]. Stunkard AJ, Messick S. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *J Psychosom Res.* 1985; 29:71–83. [PubMed: 3981480]
- [39]. Bond MJ, McDowell AJ, Wilkinson JY. The measurement of dietary restraint, disinhibition and hunger: an examination of the factor structure of the Three Factor Eating Questionnaire (TFEQ). *Int J Obes Relat Metab Disord.* 2001; 25:900–6. [PubMed: 11439306]
- [40]. Schachter S. Obesity and eating. Internal and external cues differentially affect the eating behavior of obese and normal subjects. *Science.* 1968; 161:751–6. [PubMed: 5663800]
- [41]. Herman CP, Polivy J. External cues in the control of food intake in humans: the sensory-normative distinction. *Physiol Behav.* 2008; 94:722–8. [PubMed: 18499202]
- [42]. Herman CP, Polivy J. Normative influences on food intake. *Physiol Behav.* 2005; 86:762–72. [PubMed: 16243366]
- [43]. Lluich A, Herbeth B, Mejean L, Siest G. Dietary intakes, eating style and overweight in the Stanislas Family Study. *Int J Obes Relat Metab Disord.* 2000; 24:1493–9. [PubMed: 11126347]
- [44]. Snoek HM, van Strien T, Janssens JM, Engels RC. Emotional, external, restrained eating and overweight in Dutch adolescents. *Scand. J. Psychol.* 2007; 48:23–32. [PubMed: 17257366]
- [45]. Mussig K, Remer T, Maser-Gluth C. Brief review: Glucocorticoid excretion in obesity. *J. Steroid Biochem. Mol. Biol.* 2010; 121:589–93. [PubMed: 20109546]
- [46]. Chaput JP, Drapeau V, Hetherington M, Lemieux S, Provencher V, Tremblay A. Psychobiological impact of a progressive weight loss program in obese men. *Physiol Behav.* 2005; 86:224–32. [PubMed: 16112692]
- [47]. Anderson DA, Shapiro JR, Lundgren JD, Spataro LE, Frye CA. Self-reported dietary restraint is associated with elevated levels of salivary cortisol. *Appetite.* 2002; 38:13–7. [PubMed: 11883913]
- [48]. McLean JA, Barr SI. Cognitive dietary restraint is associated with eating behaviors, lifestyle practices, personality characteristics and menstrual irregularity in college women. *Appetite.* 2003; 40:185–92. [PubMed: 12781168]
- [49]. Purnell JQ, Kahn SE, Samuels MH, Brandon D, Loriaux DL, Brunzell JD. Enhanced cortisol production rates, free cortisol, and 11 beta-HSD-1 expression correlate with visceral fat and insulin resistance in men: effect of weight loss. *Am J Physiol-Endoc M.* 2009; 296:E351–E7.

- [50]. Timko CA, Perone J. Rigid and flexible control of eating behavior in a college population. *Eat Behav.* 2005; 6:119–25. [PubMed: 15598598]
- [51]. van Strien T, Herman CP, Verheijden MW. Eating style, overeating, and overweight in a representative Dutch sample. Does external eating play a role? *Appetite.* 2009; 52:380–7.
- [52]. Koenders PG, van Strien T. Emotional Eating, Rather Than Lifestyle Behavior, Drives Weight Gain in a Prospective Study in 1562 Employees. *J. Occup. Environ. Med.* 2011; 53:1287–93. [PubMed: 22027541]
- [53]. Kontinen H, Silventoinen K, Lahteenkorva SS, Mannisto S, Haukkala A. Emotional eating and physical activity self-efficacy as pathways in the association between depressive symptoms and adiposity indicators. *Am. J. Clin. Nutr.* 2010; 92:1031–9. [PubMed: 20861176]

Highlights

We test associations between weight-related eating behaviors and objective measures of total adiposity and abdominal adiposity. > Visceral and subcutaneous abdominal adipose was measured by DXA and MRI. > External eating is associated with subcutaneous abdominal adiposity. > Routine dietary restraint is associated with visceral abdominal adiposity. > Different weight-related eating behaviors are associated with depot-specific adiposity.

Table 1

Comparison of adjusted means* of body composition measures between white and Japanese American women

<i>Body composition measures</i>	<i>White</i>	<i>Japanese American</i>	<i>P-value</i>	<i>ω^2</i>
<i>Total adiposity</i>				
Body mass index (kg/m ²)	26.9 (25.2,28.7)	26.5 (24.7,28.3)	0.718	--
DXA total fat (kg)	28.8 (25.0,32.5)	25.5 (21.8,29.3)	0.280	--
<i>Abdominal adiposity</i>				
Waist circumference at navel (cm)	92.8 (90.9,94.7)	97.4 (95.5,99.3)	0.002	0.02
DXA trunk fat (kg)	13.4 (13.4,14.6)	15.3 (14.7,15.9)	0.008	0.10
DXA trunk-to-periphery fat ratio	1.1 (1.0,1.2)	1.4 (1.3,1.5)	0.008	0.10
<i>Abdominal adiposity distribution</i>				
MRI subcutaneous fat area (cm ²)	190.1 (169.5,210.7)	200.8 (176.4,225.2)	0.520	--
MRI % subcutaneous fat (% abdominal area)	30.3(27.1,33.4)	33.4 (29.7,37.2)	0.207	--
MRI visceral fat area (cm ²)	127.5 (98.6,156.4)	153.0 (118.7,187.3)	0.275	--
MRI % visceral fat (% abdominal area)	18.5 (16.0,21.0)	23.9 (20.9,26.9)	0.011	0.09
MRI % liver fat (geometric mean)	3.8(3.5,4.1)	5.9 (5.6,6.3)	0.100	--

DXA – dual energy x-ray absorptiometry; MRI – magnetic resonance imaging

P-values and omega-squared (ω^2) represent the effect of group designation on each outcome variable.

Sample included n=30 White women and n=30 Japanese American women for anthropometry and DXA variables and n=28 White women and n=20 Japanese American women for MRI variables.

* All measures of adiposity were adjusted for age. DXA total fat, trunk fat, trunk-to-periphery fat ratio, and % liver fat were further adjusted for height and total fat (where appropriate). Measures of adiposity distribution were adjusted for age and total fat only.

Table 2

Comparison of adjusted means ^{*} in weight-related eating questionnaire scores between White and Japanese American women

<i>Weight-related eating behaviors</i>	<i>White</i>	<i>Japanese American</i>	<i>P-value</i>
Compensatory restraint	2.98 (2.61,3.35)	2.89 (2.52,3.26)	0.743
Routine Restraint	1.96 (1.68,2.25)	1.83 (1.55,2.12)	0.512
External Eating	2.39 (2.11,2.66)	2.43 (2.16,2.71)	0.813
Emotional Eating	1.96 (1.63,2.29)	1.96 (1.63,2.30)	0.996

Weight-related eating questionnaire subscale scores range from 1 (does not describe me at all) – 5 (describes me completely).

Sample included n=30 White women and n=30 Japanese American women.

^{*} All means (95% CI) were adjusted for age.

Table 3
Multivariate regression models of Weight-Related Eating Questionnaire scores and body composition measures (n=60)

<i>Body composition measures</i>	<i>R² eating behaviors only</i>	<i>R² eating behaviors, age, race</i>	<i>Compensatory restraint</i> β (SE)	<i>p-value</i>	<i>Routine restraint</i> β (SE)	<i>p-value</i>	<i>External eating</i> β (SE)	<i>p-value</i>	<i>Emotional eating</i> β (SE)	<i>p-value</i>
<i>Total adiposity</i>										
Body mass index	0.154 [†]	0.161	-0.123 (0.177)	0.490	-0.048 (0.180)	0.790	0.364 (0.155)	0.023	0.055 (0.169)	0.743
DXA total fat	0.142 [†]	0.193 [†]	-0.192 (0.173)	0.272	0.027 (0.176)	0.877	0.411 (0.153)	0.001	-0.053 (0.166)	0.750
<i>Abdominal adiposity</i>										
Waist circumference at navel	0.141 [†]	0.148	-0.131 (0.178)	0.462	-0.050 (0.181)	0.781	0.378 (0.157)	0.019	-0.004 (0.170)	0.981
DXA trunk fat	0.112	0.131	-0.243 (0.180)	0.183	0.068 (0.183)	0.710	0.336 (0.159)	0.039	-0.036 (0.173)	0.836
DXA trunk-to-periphery fat	0.050	0.192 [†]	-0.313 (0.173)	0.077	0.170 (0.176)	0.340	-0.043 (0.155)	0.783	0.038 (0.169)	0.821
<i>Abdominal adiposity distribution</i>										
MRI subcutaneous fat area	0.238 [*]	0.241 [†]	-0.083 (0.189)	0.563	0.199 (0.199)	0.325	0.484 (0.158)	0.004	-0.088 (0.183)	0.633
MRI % subcutaneous fat	0.208 [*]	0.238 [†]	-0.046 (0.189)	0.811	0.057 (0.200)	0.778	0.545 (0.161)	0.001	-0.242 (0.190)	0.208
MRI visceral fat area	0.128	0.140	-0.305 (0.201)	0.136	0.429 (0.212)	0.050	-0.042 (0.175)	0.810	0.263 (0.202)	0.201
MRI % visceral fat	0.131	0.210	-0.359 (0.192)	0.069	0.423 (0.203)	0.044	-0.008 (0.164)	0.963	0.163 (0.193)	0.403
MRI % liver fat	0.076	0.171	-0.278 (0.197)	0.167	0.184 (0.208)	0.382	-0.186 (0.167)	0.270	0.160 (0.193)	0.411

WREQ = Weight-Related Eating Questionnaire; β – standardized parameter estimate in the models including age, ethnicity and all WREQ variables; *SE* – standard error; *R²* = percent of variance accounted for by variables in the models; DXA – dual energy X-ray absorptiometry; MRI – magnetic resonance imaging

Sample included n=30 white women and n=30 Japanese American women for anthropometry and DXA variables and n=28 white women and n=20 Japanese American women for MRI variables.

^{*} P<0.05 for the model F test

[†] P<0.10 for the model F test