Disordered-Eating Attitudes in Relation to Bone Mineral Density and Markers of Bone Turnover in Overweight Adolescents

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Abstract

Purpose—To examine the relationships between cognitive eating restraint and both bone mineral density (BMD) and markers of bone turnover in overweight adolescents.

Methods—137 overweight (BMI 39.1±6.8 kg/m^2) African American and Caucasian adolescent (age=14.4 ± 1.4y) girls (66.4%) and boys were administered the Eating Disorder Examination (EDE) interview and Eating Inventory (EI) questionnaire and underwent dual energy x-ray absorptiometry (DXA) to measure total lumbar spine BMD. Markers of bone formation (serum bone specific alkaline phosphatase and osteocalcin), bone resorption (24-hour urine N-telopeptides), and stress (urine free cortisol) were measured.

Results—After accounting for the contribution of demographics, height, weight, serum 25-hydroxyvitamin D, and depressive symptoms, adolescents' weight concern, as assessed by interview, was a significant contributor to a model of urine free cortisol (β =.30, p <.05). Shape concern, as also assessed by interview, was significantly associated with lumbar spine bone mineral density (β =.−.15, p < 05). Dietary restraint was not a significant predictor in any of these models.

Conclusions—These findings suggest that among severely overweight adolescents, dissatisfaction with shape and weight may be salient stressors. Future research is required to illuminate the relationship between bone health and disordered-eating attitudes in overweight adolescents.

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Introduction

Preliminary data in adults suggest that cognitive eating restraint, even in the absence of actual dietary restriction, may be related to decreased bone mineral density (BMD) and altered biomarkers of bone turnover [1–4]. Cognitive eating restraint, as originally defined by the Eating Inventory (EI), is the intent to restrict or limit food intake in order to either prevent weight gain, or to induce weight loss [5], and is reported by both overweight and non-overweight individuals [6]. The effect of cognitive eating restraint on bone health is speculated to be the result of increased endogenous cortisol production, due to the stress of maintaining disturbed-eating cognitions [1]. Cortisol has been implicated as an inhibitor of osteoblast replication, with the subsequent disturbance of bone metabolism leading to bone mineral loss [1].

Most of the literature to date has focused upon samples of non-overweight women. One study found that women endorsing greater cognitive eating restraint, as measured by questionnaire [7], had significantly higher urine free cortisol excretion than those with lower restraint scores [2]. Another study of non-overweight women, 18–25 years, reported an inverse relationship between cognitive eating restraint, as assessed by questionnaire [7], and serum osteocalcin, a marker of bone formation [1]. The relationship with osteocalcin suggests that cognitive eating restraint might be associated with decreased osteoblastic activity [8]. Another study of women 18–45 years found that cognitive eating restraint, also assessed by questionnaire [7], was negatively correlated with bone mineral content (BMC), but not BMD [3]. Findings were especially pronounced in women weighing less than 71 kg. The authors of this study posited that high cognitive restraint and disordered eating attitudes, particularly during young adulthood, may compromise long term bone health.

To our knowledge, no studies have examined the association between cognitive eating restraint and bone health in overweight youth. This gap in the literature is notable, given the high prevalence of pediatric overweight [9], which has tripled in recent decades [9], and a wealth of data indicating a robust association between overweight and disordered-eating attitudes among youth [10]. Overweight during childhood puts individuals at high risk for a host of complications including a greater risk of fractures, orthopedic discomfort and impairment in mobility [11]. The paucity of data exploring bone health in overweight samples may be, in part, due to the presumption that overweight individuals are at a relatively low risk for osteoporosis, a view that has recently come into question [12]. Only one study [4] has examined this relationship in a sample of obese pre-menopausal women with histories of chronic dieting. Thirty-one percent of this sample had either osteopenia or osteoporosis. Furthermore, a significant negative association was found between BMC and both cognitive restraint and reported number of dieting attempts [4]. Irrespective of current intake or dietary patterns, women with histories of greater cognitive dietary restraint presented with significantly lower BMC compared to women without such histories. However, depressive symptoms were not controlled for, despite the existence of data suggesting that individuals with depression present with lower BMD [13].

Adolescence is the critical period for the acquisition of peak bone mass. Nearly 90% of adult bone mass is believed to be acquired before the age of 20, [14] making adolescence a crucial time during which to study bone health, and those variables that might have a deleterious effect on the markers of bone turnover. Included in such variables is cognitive eating restraint, which

has been shown to emerge during adolescence [15]. Since adolescence is also the peak time for the development of disordered-eating [16], the relationship between eating pathology and bone health in overweight youth warrants investigation.

We studied markers of bone turnover and bone density along with constructs of cognitive eating restraint and other disordered-eating attitudes in overweight adolescents. To expand upon the extant adult literature, we specifically examined cognitive eating restraint, as assessed by the Eating Inventory. However, since cognitive eating restraint has been shown to be associated with other measures of disturbed-eating attitudes, namely, eating, shape and weight concerns [15] that are salient in overweight samples, we expected that constructs of disordered-eating, other than restraint, might also be related to BMD and bone turnover. To this effect, we also examined the four subscales of the Eating Disorder Examination: restraint, eating concern, shape concern, and weight concern. Markers of bone formation (serum bone specific alkaline phosphatase and osteocalcin), bone resorption (24-hour urine N-telopeptides), and stress (urine free cortisol) were measured in addition to BMD. We specifically chose to examine BMD in our analyses, rather than BMC. This is due to the fact that BMD corrects, to some extent, for bone area, thereby reducing dependency on bone size [17]. We also included height in our models as an additional adjustment for bone size. Areal BMD is believed to be superior to BMC in the prediction of hip fracture in adults [18].

Since it remains unclear whether questionnaire or interview is a more valid assessment of disordered-eating in youth [10], we included both methodologies to capture several related constructs. Based upon the available studies in adult samples, we hypothesized that a negative association would be found between measures of disordered-eating attitudes and both BMD and markers of bone turnover, accounting for nutritional and demographic variables in overweight adolescents.

Methods

Participants

Overweight adolescents were recruited for a weight-loss treatment study involving medication as previously described [19]. Inclusion criteria for the treatment study were ages 12–17 years at study entry, body mass index (BMI, kg/m^2) $\geq$ than the NHANES II 95th percentile for age, race, and sex and the presence of at least one quantifiable obesity-related comorbidity, including hypertension, type 2 diabetes or impaired glucose tolerance, hyperinsulinemia (insulin$\geq$15 µU/L), hyperlipidemia (total triglyceride$\geq$200 mg/dL, total cholesterol>200 mg/dL, or LDL-cholesterol$\geq$130 mg/dL), hepatic steatosis, or sleep apnea documented by a formal sleep study. Individuals were excluded if they had a major pulmonary, hepatic, cardiac, or musculoskeletal disorder unrelated to obesity, a history of substance abuse or other psychiatric disorder that would impair compliance with the study protocol, used an anorexiant in the past 6 months, or recently lost significant weight. For a complete description of study requirements and recruitment strategies, see McDuffie et al. [19]. This study was approved by the National Institute of Child Health and Human Development Institutional Review Board. Each adolescent gave written assent, and a parent or guardian gave written consent, for protocol participation.

Procedures

All data were collected at baseline, before subjects initiated treatment.

Psychological Measures—The *Eating Disorder Examination version 12OD/C.2* (EDE) [20], is a semi-structured interview that contains 21 items that assess disordered-attitudes and behaviors related to eating, body shape, and weight, and 13 items designed to diagnose specific

Diagnostic and Statistical Manual of Mental Disorders IV-TR eating disorders. The EDE was administered to all participants by trained interviewers. Responses are coded via four subscales: restraint, eating concern, shape concern, and weight concern. The restraint subscale of the EDE is derived from questions probing food restriction and specific food avoidance, desire for an empty stomach, avoidance of eating, and self-imposed dietary rules. Unlike many other measures of dietary restraint, the EDE restraint scale assesses both behavioral and cognitive restraint. Therefore, items are endorsed if the individual tried to engage in the behavior, whether or not he/she actually succeeded in doing so. The shape concern subscale is a composite score from questions assessing dissatisfaction and preoccupation with one's shape, discomfort in seeing one's own body, and reports of feeling 'fat'. The weight concern subscale assesses dissatisfaction and preoccupation with one's weight and the desire to lose weight. The eating concern subscale is derived from questions pertaining to preoccupation with food and calories, fear of losing control over eating, and eating in secret. The global score represents the mean of the 4 subscale scores. All subscale scores range from 0 to 6. The EDE has demonstrated excellent internal consistency in adult and adolescent samples (with correlation coefficients ranging from .67 to .90) [21], test–retest reliability [22], and discriminant as well as concurrent validity [20].

The *Eating Inventory* (EI) is a 51-item self-report questionnaire that was completed by all participants. Thirty-six items are true-false, and the remaining 15 are on a 7-point likert scale. The EI is comprised of three subscales which measure cognitive restraint, disinhibition, and perceived hunger. The cognitive restraint subscale is calculated from 21 items questions and also assesses both cognitive and behavioral dietary restriction. The disinhibition subscale is derived from 16 items and measures the tendency to overeat, and the hunger subscale from 14. Cognitive restraint subscale scores of 14, disinhibition scores of 12, and hunger subscale scores of 11 indicate clinical significance [5]. We included two out of the three EI subscales in our present analyses (cognitive restraint and disinhibition), and omitted the hunger subscale, since we did not hypothesize that self-reported hunger would have an impact on bone health. The EI has been shown to be a valid and reliable instrument in some [23], but not all [24] studies, with internal consistency coefficients ranging from .70 to .90 [24].

The *Children's Depression Inventory* (CDI) [25] is a self-report questionnaire used to assess cognitive, affective, and behavioral signs of depression. The CDI has demonstrated adequate internal consistency (Cronbach's alphas = .71 to .89), test–retest reliability, and discriminant validity [25,26] The CDI consists of 27 items, each of which has three possible answer choices. For example, item # 10 gives the following options: "I feel like crying every day," "I feel like crying many days," or "I feel like crying once in a while." The child is instructed to check the answer that best describes how he or she feels. CDI scores range from 0–54, with a score of 19 serving as the cutoff for clinical depression [25]. The total score of depression (which is the sum total of all of the items on the measure) was used as a covariate for all analyses. The total composite score is most frequently used when including CDI as a covariate since previous factor analyses of the CDI have not supported the presence of subscales [26]. The total score represents the sum of depressive symptoms related to negative mood, interpersonal problems, ineffectiveness, anhedonia, and negative self-esteem.

Physical Measures—Weight and height were obtained and BMI (kg/m^2) and BMI Z-scores were calculated according to the Center for Disease Control as previously described [19]. Participants underwent a medical history and a physical examination performed by a physician or nurse practitioner. Breast and pubertal hair development were assigned according to the stages of Tanner [27] and testicular volumes were measured (in cc) according to Prader [28].

Body composition was assessed by whole-body dual energy x-ray absorptiometry (DXA) using the 4500A fan-beam densitometer (Hologic QDR-4500A, Bedford, MA) administered by

trained DXA technicians and analyzed using software package 12.4. Results include measurements of areal lumbar spine BMD, as well as lean and fat mass totals. Lumbar spine BMD was examined since it is has been shown to be a primary site of osteoporotic fracture [29]. Range of BMD scores are reported in Table 1. BMD Z-scores ranged from −1.3 to 3.4, with an average Z-score of .58. A Z-score of ≤ -2.0 indicates clinically low bone mass accounting for age and gender, while Z-scores between −1.0 and −2.0 indicate that an individual is at-risk for low bone mass [30].

Participants supplied venous blood samples after fasting (10 to 12h) for measurement of serum markers of bone turnover. The concentration of the bone turnover marker bone specific alkaline phosphatase was measured by the ALP (IFCC-DGKCh) reagent in conjunction with the SYNCHRON Enzyme Validator Set (Beckman Coulter, Inc, Fullerton, CA) to determine assay values. Serum osteocalcin was measured by ECLIA (electrochemiluminescence immunoassay) and assessed by ROCHE 1010/2010 and Modular Analytics E170 immunoassay analyzers (Roche Diagnostics, Indianapolis, IN). Urine N-telopeptide was measured by Vitros ECi Competitive Assay (Ortho-Clinical Diagnostics, Rochester, NY) using standard Rate Jaffe methodology. Urine was collected over a designated 24 hour period, and aliquots were analyzed for urine free cortisol, creatinine and calcium.

Statistical Analysis—All analyses were conducted using SPSS for Windows, 12.0 (SPSS, Inc., Chicago, IL). Pearson correlation coefficients were computed to examine the bivariate relationships between the variables of interest. A series of linear regressions were conducted to determine whether cognitive eating restraint and other disordered-eating attitudes, as measured by the EDE and EI, significantly contributed to the prediction of cortisol, BMD and markers of bone turnover, specifically: a) serum bone specific alkaline phosphatase, b) osteocalcin and c) N-telopeptides. For each model, the following covariates were considered: Age, pubertal status, race, sex, height, weight, serum 25-hydroxyvitamin D, and depressive symptoms. Depression, measured by CDI total score, was used as a covariate since data have shown that depressed individuals often present with lower BMD than their non-depressed counterparts [13]. Height and weight were used, rather than BMI, since height serves as an adjustment for bone size. Five participants met criteria for binge eating disorder, which does not appear to be associated with abnormalities in cortisol metabolism [31]. Findings did not change in direction or significance when these individuals were removed from the sample, and therefore, these adolescents were included in the presented analyses.

We ran separate regressions for each dependent variable (cortisol, BMD, serum bone specific alkaline phosphatase, osteocalcin, and N-telopeptides). A two-step modeling process was used wherein the covariates were entered into the first step, and the relevant eating pathology scales were entered into the second step. We ran all models with the EDE global score (which is a composite score of the 4 subscales) and the EI cognitive restraint and disinhibition scores for all of the dependent variables. EDE global score trended toward significance (p =.07) in the model predicting BMD, but was not a trend nor significant in the model predicting cortisol (p > .05).

In order to elucidate which of the EDE subscales might be most salient, we then ran the analyses using each individual EDE subscale. Since the subscales of the EDE are highly correlated, the 4 subscales were entered into separate models in order to avoid problems of multicollinearity. Thus, for the prediction of each dependent variable, we ran a total of 5 different models (one for each of the 4 EDE subscales separately and one for the 2 EI subscales: cognitive restraint and disinhibition). We ran the EI subscales together in one model, because disinhibition and cognitive restraint measure two distinct constructs, and accordingly had a very low correlation (r = .08). Thus, we did not have concern for the multicollinearity of these items.

Although we ran a number of regression models, we did not conduct post-hoc adjustments, and instead, opted for the standard p < .05 convention. Statisticians have argued that adjustments do not, in fact, correctly identify the findings that do and do not occur by chance [32]. Furthermore, these adjustments can yield inconsistent conclusions from results of different studies finding the same effect size [33].

In the prior adult literature, the associations of cognitive eating restraint with BMD have been monotonic, and thus we did not establish a cut-off point to examine BMD, restraint, or disordered eating scores. Additionally, to further replicate the adult literature, we did not create a composite score for markers of bone turnover, but rather examined each variable (serum bone specific alkaline phosphatase, osteocalcin, and N-telopeptides) separately.

Beta coefficients are presented and contributions of variables to models were considered significant when p values were $\leq .05$.

Results

One-hundred thirty-seven participants (14.39 ± 1.41 years) with a mean BMI of 39.1 ± 6.8 kg/m^2 were studied. The sample was comprised of 76 African American (55.5%) and 61 Caucasian adolescents. Ninety-one (66.4%) participants were female. Bivariate correlations between independent and dependent variables of interest are presented in Table 1.

In the model for urine free cortisol, CDI total score ($\beta = .30$, p = .027) and EDE weight concern ($\beta = .30$, p = .03) were the only significant contributors to the model (change in model $R^2 = .11$; see Table 2).

In the regression model of lumbar spine BMD, age ($\beta = .288$, p < .001), sex (= .582, p <.001), weight ($\beta = .461$, p <.001), race ($\beta = .156$, p < .05), and EDE shape concern ($\beta = -.154$, p < .05) all served as significant contributors to the prediction of lumbar spine BMD (change in model $R^2 = .021$). None of the other EDE subscales or any of the EI scales contributed significantly to the model (see Table 3).

None of the EDE subscales served as significant contributors in models of osteocalcin, N-telopeptides, or bone specific alkaline phosphatase. In order to examine whether sex potentially moderated the observed effects in all of the aforementioned variables, tests were conducted for any sex by independent variable interactions. No significant interactions were found for either BMD ($\eta_p^2 = .477$, p = .078) or urine free cortisol ($\eta_p^2 = .630$, p = .160). However, the pattern of these interactions suggest that significance was not reached due to a lack of statistical power.

At the time of assessment, 13 participants were taking medications that have been demonstrated to impact BMD (ie. Advair, Albuterol, hydrocortisone, and oral contraceptives), thus all analyses were re-run excluding these individuals. However, findings did not differ when these individuals were removed from the sample. Since both being very underweight *and* very overweight may lead to amenorrhea, we also controlled for menstrual history in our analyses. Although no females in our sample had amenorrhea (defined as not starting menstruation after the age of 15, or ≥ 6 months since last menstrual period), 12 girls had oligo- amenorrhea (infrequent periods: ≥ 35 days between cycles)[34]. We re-ran all analyses excluding these individuals and findings did not differ from those found using the entire sample.

Discussion

In this examination of the relationships of disordered-eating attitudes and behaviors with BMD and markers of bone turnover, we found a positive relationship between interview assessment

of weight concern and urine free cortisol, a biological marker of stress. Additionally, we found that shape concern, but not cognitive restraint, was inversely associated with lumbar spine BMD. Since the field is still uncertain as to whether questionnaire or interview is a more valid assessment of disordered-eating in adolescents [10], we included both measures in order to assess for any methodological differences in findings. Furthermore, since the extant adult literature examining bone health in relation to restraint utilized solely questionnaire methods, we believe it is important to include both questionnaire as well as interview measures. Our findings revealed that only weight and shape concerns, as assessed by interview, but not by questionnaire, were predictive of markers related to bone health.

In contrast to studies of healthy pre-menopausal women and overweight women, [1–4] we found no relationship between adolescents' cognitive dietary restraint and BMD, serum osteocalcin, or urine free cortisol. We did, however, find a positive association between weight concern and urine free cortisol. As suggested in the adult literature [1,2], we speculate that the stress of maintaining undue concern with one's weight may be a cause of increased endogenous cortisol excretion. Cortisol is a physiological marker of stress and has been shown to impede osteoblast replication [1].

Studies in adults have found that depression is negatively associated with BMD [14, 41]. One such study reported elevated levels of urine free cortisol in pre-menopausal women with major depressive disorder or depressive symptoms [35]. However, we found no significant relationship between depressive symptoms and cortisol or markers of bone turnover. This finding may be, in part, due to the relatively healthy psychological profile of participants. The cutoff score for clinical depression on the CDI is 19 out of 54 [25], while the mean CDI score for our sample was 7.4 and only 8 subjects had values above 19. Since the mean depression score for our sample was relatively low, it seems unlikely that depression served as an important factor in the levels of cortisol or BMD found in our analyses.

Another potential physiologic mediator of the effect of restraint and disordered eating on bone health is Peptide YY (PYY) which has been shown to be elevated in anorexia nervosa, and inversely correlated with BMD [36]. Although this relationship may be due to the inverse relationship between PYY and BMI, the role that PYY plays in the impact of restraint and eating pathology on BMD warrants further exploration.

Since our sample consisted entirely of treatment-seeking, overweight youth, it is possible that cognitive eating restraint was somewhat less pathological among this sample. Since restraint may actually be beneficial to individuals attempting to lose weight, concern with weight may be a more salient stressor than restraint to our studied group, thereby accounting for the positive association with cortisol. Bolstering this possibility are data suggesting that dietary restraint is common among overweight individuals [6]. The finding that shape concern, but not cognitive restraint, was a significant contributor to the model predicting lumbar spine BMD further suggests that among severely overweight adolescents, dissatisfaction with shape and importance of shape in self-evaluation, may be a more relevant stressor than cognitive dietary restraint.

Exclusive of eating patterns, data regarding the relationship between overweight status and bone health are conflicting. De Schepper et al. found that overweight status during adolescence had no negative effect on BMD [37], while other studies have found that overweight children present with increased total body BMC [38], and BMD [39] compared to non-overweight peers. By contrast, other data suggest that overweight is associated with lower BMD Z-scores among children ages 5–19y who have experienced at least two fractures of the forearm [40]. Our data indicating that psychological distress from disordered-eating patterns may influence bone

density in overweight adolescents, independent of body weight, may help explain these contradictory findings.

Strengths of this study include the use of both interview and questionnaire methods to assess eating-disordered psychopathology and the measurement of BMD by DXA, considered to be an excellent measure of BMD. Moreover, the studied sample was relatively large, racially diverse and included both males and females. A study limitation includes the usage of a questionnaire-measure of depressive symptoms as opposed to a diagnostic interview. Our findings are also limited in that they may only be generalizable to samples of weight-loss treatment-seeking overweight adolescents. Another limitation is that a number of models were tested (in order to avoid issues of multicollinearity), which increases the likelihood of Type 1 error. However, analyses were planned a priori and were similar to findings from previous data. Finally, a notable limitation is the cross-sectional nature of our study. Future longitudinal studies investigating the effects of eating pathology on bone health and markers of bone turnover are required.

In conclusion, our findings corroborate previous studies demonstrating that disordered-eating attitudes may be closely linked with an increase in endogenous cortisol production and a decrease in BMD. Our data suggest that underlying psychological distress, rather than merely dietary restraint, may be the primary contributor to these associations. However, the exact mechanism by which this distress may predispose individuals to decreased BMD remains unclear.

Adolescence is a crucial time during which to study bone health; the literature has shown that 90% of adult bone mass is acquired before the age of 20 [14]. Furthermore, disordered-eating attitudes and restraint have been shown to emerge primarily during adolescence [15,16]. Thus the relationship between eating pathology and bone health in adolescent samples seems to warrant extensive investigation.

Further study is required to determine the most relevant aspects of disordered-eating for bone health in adolescents, and whether such constructs are prospectively predictive of the physical outcome of overweight youth.

Acknowledgments

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Table 1

Bivariate correlations between markers of bone turnover and disordered eating attitudes.

	Range, Mean	1	2	3	4	5	6	7	8	9	10	11
1. Lumbar Spine BMD	.55 – 1.50 g/cm², 1.00	--										
2. Osteocalcin (n = 111)	.5 – 157.3 ng/mL, 28.13	.25**	--									
3. Bone Specific Alkaline Phosphatase (n = 32)	11 – 93 µg/L, 37.06	.51**	.39	--								
4. Urine Free Cortisol (n = 106)	1.4 – 164 ug/dL, 38.03	–.14	–.27*	–.01	--							
5. N-Telopeptide 24 hr (n =88)	8.3 – 506 nmol/L, 160.48	.64**	.60**	.72**	.13	--						
6. EDE Restraint	0 – 4.95	–.07	.05	–.28	–.10	.03	--					
7. EDE Eating	0 – 4.41	.23**	–.05	–.23	–.07	–.11	.52**	--				
8. EDE Shape	0 – 5.75, 1.65	–.05	–.15	–.17	.08	–.01	.49**	.63**	--			
9. EDE Weight	0 – 4.8, 1.82	–.09	–.12	–.13	.12	–.04	.51**	.55**	.82**	--		
10. EI Cognitive Eating Restraint	2-15, 8.43	.31**	.05	.17	.12	.02	.26**	–.01	.17	.17	--	
11. EI Disinhibition	0-15, 6.08	–.01	–.19	–.21	–.06	.04	.38**	.56**	.58**	.53**	.08	--
12. EI Hunger	1-14, 5.75	.04	–.03	.03	–.21	.06	.31**	.44**	.36**	.32**	–.06	.67**

Note. N = 137 except where otherwise noted

*
p < 0.05

**
p < 0.01 EDE = Eating Disorder Examination; EI = Eating Inventory.

Table 2

EDE Weight Concern is a Significant Predictor of Urine Free Cortisol

Variable	Beta	Significance
Race	−.118	.375
Sex	−.240	.201
Age	.074	.574
CDI Total Score	.302	**.027**
Tanner Stage	.168	.337
Vitamin D	−.047	.727
Weight	−.127	.373
Height	.019	.886
EDE Weight Concern	.300	**.033**

Table 3

EDE Shape Concern is a Significant Predictor of Lumbar Spine Bone Mineral Density

Variable	Beta	Significance
Race	.156	**.044**
Sex	.582	**.000**
Age	.288	**.000**
CDI Total Score	.075	.387
Tanner Stage	.146	.170
Vitamin D	−.107	.211
Weight	.461	**.000**
Height	.154	.063
EDE Shape Concern	−.154	**.048**