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Pressure to be Thin and Insulin Sensitivity among Adolescents

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Abstract

Purpose—Extant research indicates that some of the comorbidities associated with adult obesity may be adversely affected by the stress resulting from negative body image and weight-related teasing. This study examined the association between weight-related pressure and insulin sensitivity in adolescents, who are vulnerable to both weight-based teasing and the onset of metabolic dysregulation.

Methods—Participants were 215 adolescent healthy volunteers (55% female; 59% White; 35% overweight/obese; $M \pm SD$ age = 15.4 ± 1.4 y), who completed a self-report measure of pressure to be thin from parents, friends, and romantic partners. Fasting blood samples were obtained to assess serum insulin and glucose, which were used to calculate insulin sensitivity; fat mass (kg) and fat-free mass (%) were measured with air displacement plethysmography. Pubertal stage was determined by physical examination.

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Results—Pressure to be thin was positively associated with fasting insulin ($p = .01$) and negatively associated with insulin sensitivity ($p = .02$), after controlling for pubertal stage, sex, race, height, fat-free mass, and adiposity. Pressure to be thin was associated with a greater odds of having hyperinsulinemia (fasting insulin ≥ 15 $\mu\text{IU/mL}$; Odds Ratio (95% CI): 1.65 (1.08–2.50), $p = .02$), adjusting for the same covariates.

Conclusions—Results indicate that adolescents perceiving more pressure to be thin have greater elevations of fasting insulin and poorer insulin sensitivity above and beyond the effect of fat mass. Future research is warranted to elucidate the mechanisms responsible for this relationship.

Keywords

Adolescents; pressure to be thin; insulin; insulin sensitivity

Susceptibility to type 2 diabetes and the metabolic syndrome is only partially explained by excess adiposity and obesity [1]. Extant research has indicated that, in addition to excess body fat, psychological factors may be associated with, and predictive of elevated insulin concentration and impaired insulin sensitivity, which themselves predict the onset of type II diabetes [2], and confer additional risk for hypertension, above and beyond the contribution of obesity [3]. For instance, weight-based stigmatization, a considerable source of psychological stress, is associated with biochemical stress [4] and has been shown to amplify the link between central adiposity and nondiabetic glycemic control among overweight adults [5]. Body dissatisfaction and weight-related concerns have also been implicated in the pathophysiology of obesity, above and beyond the effects of excess adiposity [6]. Additionally, both chronic and acute psychological stressors have been hypothesized to contribute to elevated fasting insulin and have been linked in multiple studies to metabolic changes, such as increased cortisol and insulin, greater abdominal adiposity, and worsening inflammation [7]. For instance, prenatal exposure to stressful life events predicts insulin elevation in adolescence [8], and in a study of healthy adult women, psychosocial stress increases the risk of developing metabolic syndrome 15 years later [9].

To date, most research on stress and metabolic indices has focused on adult populations. However, adolescence marks a critical time for the study of metabolic function, as insulin sensitivity naturally decreases during puberty [10]. While for most teens, insulin sensitivity normalizes after puberty, for those at risk for type 2 diabetes, adolescence may be a sensitive period for worsening insulin resistance. Additionally, myriad psychosocial stressors emerge during adolescence, such as pubertal maturation, academic challenges, social pressures, role transitions, and increased focus on romantic relationships. Notably, existing research investigating the associations of mood with fasting insulin and insulin sensitivity among adolescents has revealed a link between depression and greater fasting insulin and decreased insulin sensitivity both cross-sectionally [11] and prospectively [12]. It is less clear, however, whether other psychological risk factors are related to indices of insulin function among adolescents.

One particularly salient psychological stressor among adolescents is pressure to be thin, which may be promoted by the media, peers, friends, and family members. Longitudinal research has implicated pressure to be thin in the development of body dissatisfaction [13,

14], depression [15], and recurrent dieting [16] among both males and females. As adolescence is a vulnerable period for the onset of body dissatisfaction [17] and youth of all weight strata report frequent instances of weight-related pressures and teasing [18], adolescents may be particularly sensitive to weight-related comments. Given that pressure to be thin is a significant psychosocial stressor for adolescents irrespective of weight status, in addition to the developmental and metabolic changes inherent in adolescence [10], it is important to ascertain the association between pressure to be thin and indices of insulin sensitivity in adolescents.

We therefore investigated whether perceived pressure to be thin from parents, friends, and romantic partners was associated with fasting insulin and insulin sensitivity among adolescents. Based upon data demonstrating links between psychosocial stressors and impaired metabolic function [7], we hypothesized that greater pressure to be thin would be associated with greater fasting insulin and insulin resistance among adolescents of all weight strata.

Materials and Methods

Participants and procedures

Participants were a convenience sample of healthy adolescent volunteers participating in a non-treatment study investigating eating behaviors ([ClinicalTrials.gov](https://clinicaltrials.gov/ct2/show/study/NCT00631644) ID: NCT00631644). Families were recruited through flyers posted on bulletin boards at the National Institutes of Health (NIH) Mark O. Hatfield Clinical Research Center as well as at local area supermarkets, libraries, and listservs in the Washington, DC greater metropolitan area. Flyers and advertisements specified that studies were investigating eating behaviors in adolescent volunteers and that no treatment would be provided. Individuals were eligible to participate if they were between 13 and 17 years of age and in good general health, as indicated by medical history and physical examination, negative urine glucose, and normal electrolytes, hepatic, and thyroid function. Individuals were excluded if they reported a significant chronic illness, use of medications likely to affect body weight or appetite, pregnancy, ongoing weight-loss treatment, or a psychiatric condition that would impede adherence to study procedures, such as a clinically significant anxiety, mood, or eating disorder, or a cognitive delay that would preclude the completion of procedures. Adolescents provided written assent and parents/guardians gave written consent for participation. The study was approved by the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development Institutional Review Board.

Participants were seen at the NIH Clinical Research Center following an overnight fast initiated at 10:00 pm the night before. Study coordinators contacted all participants and their parents the evening prior to remind them to fast in preparation for the appointment. Participants' fasting state was confirmed by the research team member prior to the beginning of study procedures and individuals who were not fasting were excused. All study visits were scheduled for weekday mornings at 8:00 am. The following measures were collected.

Anthropometrics

Height and fasting weight were obtained. Participants remained clothed, but did not wear shoes. Height was measured in triplicate to the nearest millimeter by a stadiometer (Holtain, Crymmych, Wales) calibrated before each measurement. Weight was measured to the nearest 0.1 kg with a calibrated digital scale (Scale-Tronix, Wheaton, IL). Body mass index (BMI; kg/m^2), BMI standard deviation scores (BMI z-score), and BMI percentiles for age and sex, according to the Centers for Disease Control and Prevention 2000 growth charts [19] were calculated. Body composition was measured using air-displacement plethysmography (Life Measurement Inc., Concord, CA) to determine fat-free mass and fat mass. All participants underwent a medical history and a physical examination performed by an endocrinologist or a nurse practitioner. Testicular volume (mL) was measured using a set of orchidometer beads as standards according to Prader [20], and breast development (using inspection and palpation) was assigned according to the five stages of Tanner [21]. Testicular volume for males and Tanner breast staging for females were used to categorize adolescents into three groups: pre-puberty (for boys: testes ≤ 3 mL; for girls: Tanner stage 1), early/mid-puberty (for boys: testes 4–15 mL; for girls: Tanner stages 2–3) or late puberty (for boys: testes ≥ 15 mL; for girls: Tanner stages 4–5). In a prior study of lean and obese youth conducted by our group, interrater reliability between examiners for Tanner breast stage was 100% [22].

Insulin Resistance and Sensitivity

Fasting blood samples were obtained for plasma glucose (mg/dL) and serum insulin ($\mu\text{IU}/\text{mL}$). Plasma for glucose was collected in tubes containing powdered sodium fluoride and measured by the NIH Clinical Center clinical laboratory using a Hitachi 917 analyzer (Roche Diagnostics Indianapolis, IN). Insulin concentrations were determined using a commercially-available immunochemiluminometric assay purchased from Diagnostic Product Corp., Los Angeles, CA and calibrated against insulin reference preparation 66/304. The insulin assay uses a monoclonal anti-insulin antibody and was run on an Immulite2000 machine (Diagnostic Product Corp., Los Angeles, CA). Insulin sensitivity was estimated via two methods. The homeostatic model assessment of insulin resistance (HOMA-IR) was calculated as the product of fasting plasma glucose concentration (mg/dL) and fasting serum insulin concentration ($\mu\text{IU}/\text{mL}$) divided by 405. The quantitative insulin sensitivity check index (QUICKI) was estimated as 1 divided by the sum of the $\log_{10}$ of fasting glucose (mg/dL) and the $\log_{10}$ of fasting insulin ($\mu\text{IU}/\text{mL}$). Both HOMA-IR and QUICKI are well correlated with clamp-derived measures of insulin sensitivity in children [23].

Questionnaires

Pressure to be Physically Attractive Questionnaire (PPAQ) [24] was completed by participants. This measure assesses pressure to be thin and criticism about physical appearance within various interpersonal relationships. The pressure to be thin subscale assesses adolescents' perceptions of how often their mother, father, a same-sex friend, an opposite-sex friend and romantic partner provide positive reinforcement of thinness, for instance, "This person gives me compliments when I look like I've lost weight." The subscale is comprised of 3 items per relationship. Items are rated along a 5-point Likert scale

and averaged. The PPAQ was originally piloted in a sample of 99 undergraduates and the measure demonstrated good internal reliability [25]. In a separate study of adolescents, the pressure to be thin subscale demonstrated high convergent validity with the Perceived Sociocultural Pressure Scale [13] (mean $r = .47$) [26]. The pressure to be thin subscale demonstrated good reliability in the present sample ($\alpha = .90$). The composite score from the pressure to be thin subscale (the average of pressure to be thin from mother, father, same-sex friend, opposite-sex friend, and romantic partner) was used for the present study.

Beck Depression Inventory (BDI-II) [27] is a 21-item scale that assesses symptoms of depression over the past two weeks. It is widely used in diverse populations, and has demonstrated excellent reliability and validity [27]. Scores range from 0 – 63; higher scores indicate greater levels of depression. The BDI-II has well-established psychometric properties in adolescent samples, and showed good internal consistency in the present sample ($\alpha = .84$).

The Eating Disorder Examination V.14 OD/C.2, EDE, [28] yields restraint, eating-concern, shape-concern, weight-concern subscales, and a global score. Subscale scores range from 0–6, wherein higher numbers indicate greater eating-related psychopathology. The EDE has excellent psychometric properties, and demonstrated good reliability ($\alpha = .94$) in the present sample.

The Hollingshead Four-Factor Index of Social Status was used to assess socioeconomic status based on parental occupation and education [29]. Scores range from 1 to 5, wherein lower scores indicate higher socioeconomic status.

Data Analytic Plan

All analyses were conducted using SPSS for Windows version 22 (SPSS, Inc., Chicago, IL). Data were examined for outliers, skewness and kurtosis. Outliers (<6% of all data points) were adjusted to fall 1.5 times the interquartile range below the 25th percentile or above the 75th percentile. This strategy minimizes outliers' influence on the characteristics of the distribution, minimally changes the distribution overall and avoids potential bias associated with eliminating outliers altogether. To satisfy assumptions of normality, insulin and glucose were log (base 10) transformed. Unless otherwise specified, all subsequent analyses utilize transformed data.

Multiple regressions were performed to determine the main effects of pressure to be thin on indices related to glucose homeostasis after controlling for variables selected *a priori* that have been shown to be associated with insulin function in adolescents (pubertal stage, sex, race, body fat mass, fat-free mass, height) [30]. All variables were entered into the model in a single step. Logistic regression was used to assess whether pressure to be thin predicted hyperinsulinemia (fasting insulin ≥ 15 uIU/mL) [31]. Given the links between depressive symptoms and insulin resistance [11, 12], all models were re-analyzed with depressive symptoms as a covariate. However, depressive symptoms neither contributed significantly to any model, nor changed the direction or significance of any result. Therefore, depressive symptoms were removed as a covariate. Additionally, as dietary restraint could impact insulin function through a number of different mechanisms (e.g., weight loss, dietary

changes, or stress resulting from prolonged cognitive or behavioral restraint), we re-analyzed all models including the restraint subscale of the Eating Disorder Examination. The addition of the restraint subscale did not change the direction or significance of any model, nor was it a significant contributor to any model ($ps: .6-.9$), thus it was also removed as a covariate. This may have been due to the low mean restraint score observed in the current sample ($.48 \pm .74$).

Differences were considered significant when p values were $\leq .05$. All tests were two-tailed.

Results

Participant characteristics

Two-hundred fifteen non-overweight (BMI < 85th percentile; 65%), overweight (BMI 85th percentile; 15%), and obese (BMI $\geq$ 95th percentile; 20%) boys (45%) and girls (55%) participated. Average age was 15.3 ± 1.4 y, and mean BMI z-score was $.64 \pm .99$. The racial distribution of the sample was: 59% White, 30% Black, 5% Asian, 3% Multiracial, 3% “Other” or Unknown; 93% of the participants were non-Hispanic. The mean socioeconomic status of the sample was $2.18 \pm .97$. See Table 1 for additional demographic information.

Pressure to be thin and insulin sensitivity

Perceived pressure to be thin was positively associated with BMI z-score, such that the heavier the adolescent, the more he or she perceived pressure to be thin from parents, friends, and romantic partners ($r = .34, p < .001$; Table 1). Accounting for sex, pubertal stage, race, height (cm), fat-free mass (%), and fat mass (kg), pressure to be thin was significantly associated with fasting insulin ($\beta = .18, t(209) = 2.59, p = .01$; Figure 1A). Pressure to be thin was also positively associated with HOMA-IR ($\beta = .16, t(207) = 2.33, p = .02$; Figure 1B) and negatively associated with QUICKI ($\beta = -.17, t(207) = -2.46, p = .02$; Figure 1C). Using the same covariates, pressure to be thin was not significantly associated with fasting glucose ($\beta = .10, t(207) = 1.39, p = .16$). Controlling for covariates, for every one unit increase in perceived pressure to be thin, the odds of being classified as hyperinsulinemic (fasting insulin ≥ 15 μ IU/mL) increased by a factor of over 1.5 (Odds Ratio (95% CI): 1.65 (1.08–2.50), $p = .019$; Table 2).

Findings were not moderated by BMIz, fat mass, or fat-free mass. However, sex moderated the relationship between pressure to be thin and all insulin indices, such that the associations between pressure to be thin and indices of insulin function were especially pronounced among male respondents.

Although separately controlling for fat and lean mass should allow for a more precise analysis for obesity and health than BMI [32, 33], secondary analyses were also conducted controlling for BMIz, rather than fat and fat-free mass. The association of pressure to be thin with insulin was unchanged and remained significant ($p = .05$). The associations of pressure to be thin with QUICKI and hyperinsulinemia became marginally significant ($ps = .055$), and the association of pressure to be thin with HOMAIR became nonsignificant ($p = .11$) when accounting for BMIz, rather than body composition.

Discussion

In this study of healthy male and female adolescents, we found that pressure to be thin from parents, friends, and romantic partners was positively associated with fasting insulin and negatively associated with insulin sensitivity after controlling for potential confounds, including body composition and pubertal status. Additionally, pressure to be thin was significantly associated with greater odds of hyperinsulinemia. The association between pressure to be thin and fasting glucose was not significant. This lack of significance should be considered in light of data indicating that fasting measures of insulin resistance and sensitivity (HOMA-IR and QUICKI, respectively) are considered better proxies for the more subtle dysregulation in glucose homeostasis observable in healthy (non-diabetic) samples as compared to fasting glucose, since glucose concentrations are maintained in the normal range through alterations of insulin concentrations.

These data suggest that weight-related pressures may be associated with impaired insulin action among boys and girls of all weight strata. Although the present sample consisted of healthy volunteers, 16% ($n = 35$) met criteria for clinically significant hyperinsulinemia (fasting insulin ≥ 15 $\mu\text{IU/mL}$), which was significantly associated with perceived pressure to be thin. Taken together, our findings suggest that weight-related pressures may be associated with elevated insulin even among healthy weight youth. Importantly, insulin resistance predicts the onset of type II diabetes [2], and confers additional risk for hypertension, above and beyond the contribution of obesity [3].

As results are cross-sectional, the exact nature of these relationships cannot be gleaned from the present data; however, there are possible mechanisms that warrant further exploration. For instance, it may be that the experience of pressure to be thin from family and friends results in a chronic physiological stress response which has been shown to increase vulnerability for abdominal adiposity, oxidative stress, insulin resistance, and type 2 diabetes in adult samples [34, 35]. Additional mediators for the relationship between weight-related pressures and metabolic function should be explored. For instance, free fatty acids, which have been proposed as a mediator between the experience of weight stigma and glycemic control [5], might partially account for the observed relationships. Various psychosocial factors might also mediate this relationship; for instance, pressure to be thin is associated with low self-esteem, anxiety, and depression [26], which may promote chronic stress and subsequent metabolic dysfunction [11, 12, 36]. Perceived pressure to be thin might also trigger disordered eating behaviors [26], such as unhealthy caloric restriction and/or binge eating, both of which may affect insulin function [37]. Additionally, it will be important to identify whether pressure to be thin has an additive effect on other risk factors for insulin elevation, such as adiposity, binge eating, and chronic stress.

Our findings support previous research in adults linking weight-related dissatisfaction and stigma to indices of health, such as nondiabetic glycemic control [5], increased endogenous cortisol secretion [4], and blood pressure [38]. Accordingly, it has been proposed that body dissatisfaction and weight-related stress may partially account for the pathophysiology of obesity [6]. Thus, the experience of pressure to be thin and subsequent physiological stress may contribute to the development of impaired insulin action above and beyond the effect of

excess adiposity. Prospective data are required to explore whether pressure to be thin from parents, peers, and romantic partners is associated with the development of insulin resistance and other metabolic abnormalities over time or if treatments that diminish such pressures improve these parameters.

Findings from the present study may signal a need for greater attention to the metabolic function of youth who are subjected to weight-related pressures, irrespective of sex and body weight. Should these data be confirmed, it would be beneficial to raise awareness that pressure to be thin from parents and peers may be associated with impaired metabolic function among adolescent boys and girls. Though weight-related comments made by family members or friends may be intended as constructive or even complimentary, the current data suggest that these remarks may, in fact, be associated with biochemical stress. It is possible that comments alluding to the desirability of a thin body may be perceived as stigmatizing by the adolescent. As the deleterious effects of weight stigma [5, 39] and pressure to be thin [13, 14] are well-documented, comments that promote the thin body ideal, even if intended as praise, should be discouraged. It may instead be beneficial to direct praise and encouragement at healthy behavioral changes, rather than body shape/weight.

As adolescence marks an important time where insulin sensitivity is rapidly changing [10], it may be advisable to screen adolescents for weight-related pressures from family and friends, which we found to be associated with insulin resistance and which others have found to be associated with weight gain over time [39]. Additionally, in the current sample, the association between pressure to be thin and insulin elevation was especially pronounced among boys, thus it may be important to focus research efforts on male adolescents, who may be particularly vulnerable to the physiological correlates of pressures to be thin.

Study strengths include the use of measured height, weight, and body adiposity as well as systematic collection of metabolic indices. In addition, the sample consisted of non-treatment-seeking healthy volunteers and was relatively racially/ethnically diverse. Limitations include the cross-sectional nature of the data, which precludes any causal interpretations, as well as the use of fasting measures of glucose and insulin, rather than criterion measures like the euglycemic hyperinsulinemic clamp, to estimate whole body insulin sensitivity. Additionally, our primary variable of interest, pressure to be thin, was obtained via self-report. However, it may be that the *perception* of pressure to be thin is a more salient psychological stressor than actual pressure to be thin as reported by parents, friends, and romantic partners. Future research is required to elucidate the predictive utility of pressure to be thin as reported by the source (e.g., mother, father) versus perceived pressure to be thin as reported by the target.

In conclusion, perceived pressure to be thin may place adolescents at-risk for impaired insulin sensitivity. Longitudinal, prospective studies of the impact of weight-related pressures on metabolic function and health are required to ascertain the effects of pressure to be thin over time. As adolescence marks a critical period for the study of metabolic function [10] and weight gain[40], it will be important to further elucidate the effects of weight-related pressure from family, friends, and romantic partners, on metabolic health and body weight.

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Abbreviations

BDI-II	Beck Depression Inventory-II
BMI	Body mass index
BMI z-score	BMI standard deviation score
HOMA-IR	homeostatic model assessment of insulin resistance
QUICKI	quantitative insulin sensitivity check index

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Implications and Contribution

Among healthy adolescent boys and girls of all weight strata, pressure to be thin from parents, friends, and romantic partners was positively associated with fasting insulin and negatively associated with insulin sensitivity after controlling for relevant covariates. Weight-related pressures may place adolescents at risk for metabolic dysregulation.

Figure 1.

Association of pressure to be thin scale with indices of glucose homeostasis. Pressure to be thin was significantly associated with: fasting insulin (A; $p = .01$), insulin resistance as measured by HOMA-IR (B; $p = .02$), and insulin sensitivity, as measured by the QUICKI index (C; $p = .02$). Unstandardized residuals are shown, controlling for covariates as specified in Methods.

Table 1

Participant Demographics

Measure	Overweight %		Lean %		F		Total Sample %	
	N		N		N		N	
Sex						5.15*		
Male	35	26	51	110			45	97
Female	65	49	49	105			55	118
Race						2.9		
White	51	38	63	136			59	127
Black	37	28	26	56			30	65
Asian	5	4	5	11			5	11
Multiracial	3	2	3	6			3	6
Other/Unknown	4	3	3	6			3	6
Ethnicity						.51		
Hispanic	4	3	6	13			6	14
Non-Hispanic	96	72	94	202			93	201
Pubertal Status (Girls) ^a								
Pre-Pubertal	4	3	1	2	2.1		3	6
Early-Mid Pubertal	33	25	54	116			45	97
Late Pubertal	63	47	45	97			52	112
Pubertal Status (Boys) ^b								
Pre-pubertal	4	3	6	13	.09		5	11
Early-Mid Pubertal	63	47	63	135			63	135
Late Pubertal	33	25	31	67			32	69
	M ^c	SD ^d	N	M ^c	SD ^d	N	M ^c	SD ^d
Socioeconomic Status ^e	2.30	.92	71	2.11	.99	133	1.67	2.18
Age	15.4	1.4	75	15.3	1.3	140	.006	15.4
BMI Percentile	94.1	4.6	75	52.7	22.0	140	257.16***	67.1
Fat Mass (%)	34.1	9.8	74	19.3	7.9	138	143.71***	24.5
Pressure to be Thin	2.6	1.0	75	1.9	.9	140	23.99***	2.1
							1.0	215

Measure	Overweight %		Lean %		F	Total Sample %	N
Depressive Symptoms	6.8	5.9	75	5.1	4.17 *	5.7	215
Fasting Insulin (μIU/mL)	12.9	8.2	75	7.9	27.16 ***	9.7	215
Fasting Glucose (mg/dL) (mg/dL)	87.5	7.4	75	87.6	.02	87.6	215
HOMA-IR ^g	2.79	1.82	75	1.73	23.55 ***	2.10	215
QUICKI ^h	.35	.06	75	.37	12.59 ***	.36	215

Note:

^a Assigned according to the stages of Tanner; assessed by nurse practitioner or endocrinologist

^b Measured using orchidometer beads as standards according to Prader; assessed by nurse practitioner or endocrinologist.

^c Mean

^d Standard deviation

^e As assessed by the parent-reported Hollingshead Four Factor Index of Socioeconomic Status

^f Body Mass Index

^g Homeostatic model assessment of insulin resistance (Calculated as: fasting glucose (mg/dL) * fasting insulin (μIU/mL) /405)

^h Quantitative insulin sensitivity check index (Calculated as: 1 / (log(fasting insulin μIU/mL) + log(fasting glucose mg/dL))) Statistical tests conducted: one-way analysis of variance, median test

* p < .05,

** p < .01,

*** p < .001

Table 2
Association of Variables with Hyperinsulinemia^a - Controlling for Body Composition or for BMIz

A. Controlling for Body Composition			B. Controlling for BMIz		
Independent Variable	Hyperinsulinemia OR (95% CI)	P	Independent Variable	Hyperinsulinemia OR (95% CI)	P
Pubertal Stage	1.44 (.65–3.19)	.36	Pubertal Stage	1.28(.59–2.72)	.53
Sex	.53 (.16–1.68)	.29	Sex	.97 (.39–2.41)	.95
Race	.88 (.70–1.11)	.26	Race	.96(.76–1.20)	.72
Height (cm)	1.03 (.96–1.10)	.45	BMIz	2.40 (1.48–3.90)	<.001
Lean Mass (%)	.92 (.86–.99)	.03	Depression (BDI)	1.04 (.97–1.12)	.27
Fat Mass (kg)	1.00 (.96–1.05)	.99	Pressure to be Thin	1.50 (.99–2.28)	.055
Depression (BDI)	1.00 (.93–1.08)	.92			
Pressure to be Thin	1.66 (1.08–2.50)	.02			

^ahyperinsulinemia = fasting insulin 15 µU/mL