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Original article

Pressure To Be Thin and Insulin Sensitivity Among Adolescents

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A B S T R A C T

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 stigma. 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; mean ± standard deviation age = 15.4 ± 1.4 year), 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.

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 μIU/mL; odds ratio (95% confidence interval): 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.

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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.

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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

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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]. Although 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 on 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 nontreatment study investigating eating behaviors (ClinicalTrials.gov 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 after an overnight fast initiated at 10:00 P.M. 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 before the beginning of study procedures and individuals who were not fasting were excused. All study visits were scheduled for weekday mornings at 8:00 A.M. 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 .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: prepuberty (for boys: testes $\leq$ 3 mL; for girls: Tanner stage 1), early/midpuberty (for boys: testes 4 mL–15 mL; for girls: Tanner stages 2–3) or late puberty (for boys: testes $\geq$15 mL; for girls: Tanner stages 4–5). In a prior study of lean and obese youth conducted by our group, inter-rater 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 (μIU/mL). Plasma for glucose was