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The Significant Effects of Puberty on the Genetic Diathesis of Binge Eating in Girls

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Abstract

Objective—Recent data show significant phenotypic and genetic associations between ovarian hormones and binge eating in adulthood. Theories of hormonal risk focus on puberty and the possibility that hormone activation induces changes in genetic effects that then lead to differential risk for binge eating in post-puberty and adulthood. Although this theory is difficult to test in humans, an indirect test is to examine whether genetic influences on binge eating increase during the pubertal period in girls. Prior work has shown pubertal increases in genetic influences on overall disordered eating symptoms, but no study to date has examined binge eating. The present study was the first to examine these increases for binge eating.

Methods—Participants included 1,568 female twins (ages 8–25 years) from the Michigan State University Twin Registry. Binge eating and pubertal development were assessed with self-report questionnaires.

Results—Twin moderation models showed significant linear increases in genetic effects from pre-puberty (5%) to post-puberty (42%), even after controlling for the effects of age and body mass index.

Discussion—Results provide critical support for increased genetic influences on binge eating during puberty. Additional studies are needed to identify hormonal mechanisms and fully test contemporary models of ovarian hormone risk.

Recent etiologic theories focus on the role of ovarian hormones in risk for binge eating in females (Klump, Culbert, & Sisk, 2017). These theories rest on animal and human data showing significant associations between binge eating and estrogen and progesterone in adulthood (Klump et al., 2017). Proposed mechanisms center on the genomic effects of hormones and their role in altering gene transcription in neurobiological systems known to be disrupted in binge eating (e.g., dopamine; Klump et al., 2017).

A key aspect of these theories is the belief that ovarian hormone activation during puberty induces changes in genetic effects that then lead to increased rates of binge eating in adulthood (Klump, 2013; Klump et al., 2017). To date, no study has examined these

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developmental pathways, likely due to difficulties in directly examining developmental changes in neural gene expression in humans. However, an indirect method is to examine differences in the degree of genetic influence on binge eating across pubertal development. Given that ovarian hormones drive pubertal development in girls, increasing genetic effects across puberty would provide indirect support for an effect of ovarian hormones on genetic risk. Importantly, increasing genetic effects across puberty have been observed for overall disordered eating symptoms (i.e., omnibus measures that include binge eating and body dissatisfaction, weight preoccupation, compensatory behaviors; see Culbert, Burt, McGue, Iacono, & Klump, 2009), but have never been examined for binge eating. The aim of this study was to examine differences in genetic influences on binge eating across puberty in a large and developmentally diverse twin sample that has never been investigated in twin studies of puberty.

Methods

Participants

The sample consisted of 1,568 (MZ = 814 (52%); DZ = 754 (48%)) female twins ages 8 to 25 (*M(SD)* =14.19 (3.57); see Table 1 in Supplemental Material for more sample descriptives) from the Michigan State University Twin Registry (MSUTR; Burt & Klump, 2012; Klump & Burt, 2006). The MSUTR is a population-based twin registry that recruits twins (ages 3–50 years) using birth records in collaboration with the Michigan Department of Health and Human Services (see Klump & Burt, 2006 and Burt & Klump, 2012 for recruitment details). The current study included archival data from two studies within the MSUTR: (1) the *Twin Study of Mood, Behavior, and Hormones during Puberty* (see O'Connor, Burt, VanHuysse, & Klump, 2016), and (2) the *Twin Study of Hormones and Behavior across the Menstrual Cycle* (Klump et al., 2013, 2014, 2016). Twins had to meet several inclusion/exclusion criteria for these studies (e.g., no current medication use) but were nevertheless representative of MSUTR twins who did not meet these criteria in terms of disordered eating symptoms (d's = .07–.11) and racial/ethnic composition (White (81.3%), Black (10.7%), Asian (0.7%), American Indian/Alaskan Native (0.3%), multiracial (7.2%), Hispanic (6.6%)). The Michigan State University Institutional Review Board approved these studies, and all participants provided informed consent/assent and (for minors) parental consent.

Measures

Zygosity was determined using a well-validated physical similarity questionnaire that has been shown to be over 95% accurate when compared to genotyping (Lykken, Bouchard, McGue, & Tellegen, 1990; Peeters, Van Gestel, Vlietinck, Derom, & Derom, 1998). Trained research assistants independently completed the questionnaire for all twins. All twins over the age of 15 also completed a self-report version, while twins under the age of 18 had a parent (i.e., the mother in over 95% of cases) complete the questionnaire as well. Reports were compared amongst raters, and discrepancies were resolved using questionnaire responses, review of photographs of the twins by one of the principal investigators (KLK), and/or twin concordance across (Burt & Klump, 2012).

Binge eating was assessed using the binge eating scale (assessing binge episodes, thinking about binge eating) from the Minnesota Eating Behavior Survey (von Ranson, Klump, Iacono, & McGue, 2005).[1] The MEBS is appropriate for use in pre-pubertal children (Luo, Donnellan, Burt, & Klump, 2016) and shows adequate internal consistency in past (alpha = .65–.69) (von Ranson et al., 2005) and current (alpha = .68) samples. Women with bulimia nervosa (BN) also score significantly higher on the this scale than controls (von Ranson et al., 2005).

Pubertal development was measured with the self-report Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988) that assesses secondary sex characteristics (e.g., breast development) using a 4-point scale (i.e., development (1) has not yet begun, (2) has barely started, (3) is definitely underway, or (4) seems complete) that is summed across items and averaged. Maternal reports on the PDS were used for a subset of twins (n = 16; 1% of sample) who were missing PDS scores. PDS scores correlate with physician ratings (r = .61–.67) and exhibit good internal consistency in past (alphas = .76–.83) (Petersen et al., 1988) and current (alpha = .82) samples.

Body mass index (BMI) was calculated (weight in kg/height in m2) using height and weight measured with a wall-mounted ruler and digital scale, respectively. Similar to previous developmental twin studies (Culbert et al., 2013; Culbert et al., 2009; Culbert, Burt, Sisk, Nigg, & Klump, 2014; Klump et al., 2012, 2003; Klump, Burt, McGue, & Iacono, 2007; Klump, Burt, McGue, Iacono, & Wade, 2010; Klump, Holly, Iacono, McGue, & Willson, 2000; Klump, Keel, Sisk, & Burt, 2010), we used raw (instead of age- and sex-adjusted) BMI scores in analyses, although the two types of BMIs were very highly correlated in our sample (r > .99) with minimal mean differences (Mean difference = .038, SD = .03).

Statistical Analyses

Binge eating and PDS scores were log transformed prior to analysis to account for positive skew. Twin age and BMI were regressed out of each twin's binge eating score to ensure that differences across puberty were not due to these potentially confounding variables.

Twin correlations provided initial indications of pubertal differences in genetic effects. Although continuous PDS scores were used in model-fitting (see below), categorical groups were developed for the correlations based on previous twin studies (pre-pubertal: PDS < 2.5, N = 593; pubertal: PDS = 2.5–3.9, N = 317, post-pubertal: PDS = 4.0; N = 658; see Culbert et al., 2009). Only pairs concordant on pubertal status (e.g., both co-twins pre-pubertal) were included in correlations, although concordant *and* discordant co-twins were included in model-fitting. The number of discordant pairs was small (n = 57; 7% of sample), and twins from concordant/discordant pairs did not differ significantly on binge eating (d = .12; p = .18).

[1]The Minnesota Eating Behavior Survey (MEBS; previously known as the Minnesota Eating Disorder Inventory [M-EDI]) was adapted and reproduced by special permission of Psychological Assessment Resources, Inc., 16204 North Florida Avenue, Lutz, FL 33549, from the Eating Disorder Inventory (collectively, EDI and EDI-2) by Garner, Olmstead, and Polivy (1983) by the Psychological Assessment Resources, Inc. Further reproduction of the MEBS is prohibited without prior permission from Psychological Assessment Resources, Inc.

We used extended univariate twin moderation models (see Supplemental Figure 1) (van der Sluis, Posthuma, & Dolan, 2012) to examine differences in additive genetic (A; effects that add across genes), shared environmental (C; factors that are common to siblings and contribute to similarity), and nonshared environmental (E; factors that are unique to siblings and contribute to differences, including measurement error) influences on binge eating across pubertal status. These models estimate: 1) path coefficients assessing genetic/environmental influences at the lowest level of pubertal development; 2) linear moderators assessing linear increases/decreases in etiologic influences across puberty; and 3) quadratic moderators assessing non-linear increases/decreases.[2]

We first fit the "full" model that included all parameters. We then directly tested our hypotheses by comparing the fit of this model to one that constrained all genetic moderators (linear and non-linear) to 0. This model provided a poor fit to the data (see below), and thus we fit other nested models that differentially constrained the moderators. Because of the large number of submodels that could be fit, we used parameter estimates from the full model to identify appropriate submodels. This approach allowed us to test relevant submodels without unduly increasing the number of tests.

Models were fit to the raw data using the maximum likelihood option in Mx (Neale, Boker, Xie, & Maes, 2003). Fit comparisons were made by taking the difference in minus twice the log-likelihood (−2lnL) between the full and nested models, which is chi-squared distributed under the null hypothesis implied by the reduced model. Large (statistically significant) differences led to a rejection of the nested model. Akaike's information criterion (AIC), Bayesian information criterion (BIC), sample-size adjusted BIC, and deviance information criterion (DIC) were also used to select the best fitting model, where models that minimized these scores were preferred.

Prior to model-fitting, binge eating scores were standardized and minimum PDS scores were "floored" to 0. Unstandardized parameter estimates are reported in figures, as they more accurately depict absolute differences in etiologic effects than standardized estimates that represent differences as proportions of the total variance. However, we report standardized estimates in the text, as these estimates allow for a direct comparison of our findings to previous studies.

Results

Twin correlations provided initial support for study hypotheses. Genetic influences were significant during puberty/post-puberty, as the MZ twin correlations (r_{MZ} = .44/.39) were ~2x higher than the DZ twin correlations (r_{DZ} = .27/.15) (z tests of equality = 1.72 and 3.00, *p's* = .08 and .001; effect size (q) = .20–.26). By contrast, in pre-puberty, the MZ/DZ twin correlations were very similar (r_{MZ} = .37; r_{DZ} = .35) and not significantly different (z test of equality = .28, *p* = .78, q = .02), suggesting a lack of genetic effects.

[2]Twin moderation models rest on the assumption that the moderator (puberty) is genetically independent of the dependent variable (binge eating). Without independence, genetic mediation (i.e., gene-environment correlations (rGE)) could "masquerade" as genetic moderation. We ruled out mediation effects by fitting "GxE in the presence of rGE" models (Purcell, 2002) and finding that the parameter testing rGE (i.e., the moderation of the covariance path) was non-significant and could be constrained to 0 (comparison with full model: $\chi^2\Delta(3) = 5.38$, $p = .15$).

Unstandardized estimates from the full model (see Figure 1) showed continuous increases in genetic effects across pubertal development. Model fit comparisons indicated that the model constraining all genetic moderators to 0 provided a poor fit to the data (i.e., significant chi-square change test; see Table 1). Submodels were subsequently fit based on full model estimates that seemed to show: 1) linear increases in genetic effects, 2) non-linear increases in shared environmental effects; and 3) minimal changes in nonshared environmental influences. The best-fitting submodel constrained the quadratic genetic moderators and all of the nonshared environmental moderators to 0 (see Table 1). Unstandardized (see Figure 2) and standardized (see parentheses) estimates from this model showed significant linear increases in genetic effects from pre- (5%) to post- (42%) puberty, increases (from 1%–42%) and then decreases (to 1%) in shared environmental effects, and no differences in nonshared environmental influences (51–82%)[3] across pubertal development.[4]

Discussion

This was the first study to examine pubertal differences in genetic effects on binge eating. Results showed substantial increases in genetic influences from pre-puberty to post-puberty that were linear in nature and became noticeably pronounced around mid-puberty (i.e., PDS score ≥ 2.5).

Although findings are similar to those for overall disordered eating (see Culbert et al., 2009; Klump et al., 2007), findings conflict somewhat with studies showing no significant age differences in genetic influences on binge eating in twins aged 11 versus aged 17 (Klump, McGue, & Iacono, 2000). This past study did not examine puberty, and given the diverse ages in our pre-pubertal (range = 8–15 years) and pubertal (range = 9–16 years) groups, differing results are likely due to the imprecise nature of age as a proxy for pubertal development. To confirm this, we conducted post hoc age models and found no significant differences in genetic effects across age in our study (i.e., the “no age difference model” fit the data best – comparisons with the full model: $\chi^2\Delta(6) = 11.29$, $p = .08$). These data suggest that developmental differences for binge eating may be more closely tied to pubertal development than age and provide additional, albeit indirect, support for significant hormone/binge eating associations. Findings from pilot work showing increased genetic effects on binge eating at high (versus low) estradiol levels during puberty (Klump, Keel, et al., 2010) further support these associations and the need for studies examining hormonal mechanisms underlying puberty’s effects.

Although not a specific focus of this brief report, there were significant differences in shared environmental influences on binge eating across puberty as well. The quadratic shared environmental moderator was significant (see bolded estimates in the Table 1 Note), and unstandardized parameter estimates (see Figure 1) indicated an initial increase during pre-early puberty (from 0–42% standardized estimates) and then a pretty dramatic decrease (from 42-1% standardized estimates) from mid-puberty into post-puberty. Decreasing shared environmental influences across puberty are similar to past work for overall levels of

[3]Percentages are not identical because they are standardized estimates that are proportional to the total variance.
[4]We were concerned that inclusion of twins ages 16–25 may have unduly influenced our results. We re-fit all models in twins ages 8–15 only and found identical results (see Supplemental Table 2 and Figure 2).

disordered eating (Klump, Perkins, et al., 2007), although the nature of the effects differ – past work showed linear decreases (Klump, Perkins, et al., 2007), while the current study showed non-linear increases then decreases. Discrepant results may reflect true differences in the nature of shared environmental influences on overall eating pathology versus binge eating, or they could reflect sample differences. Because we included much younger twins (i.e., ages 8 and up; Klump, Perkins, et al. (2007) included ages 13 and up) and a substantially larger sample of pre-early puberty twins in the current study, we likely had much more power to detect quadratic/non-linear effects during pre-early puberty than previous work. Regardless of similarities/differences, however, our current results suggest that shared environmental factors that are specific to the early pubertal period (e.g., changing overall pressures for thinness) rather than common across development (e.g., general family factors, e.g., divorce) might be the most important types of shared environmental risk factors for binge eating.

Before ending, we should note two key limitations. We relied on a self-report rather than interview measure of binge eating. Although self-reports might over-estimate binge eating (Berg, Peterson, Frazier, & Crow, 2011), the percentage of our twins (3.4%; see Supplemental Table 1) scoring above the binge eating scale cut-off for women with BN (von Ranson et al., 2005) is on par with what would be expected. Moreover, our measure performs well across the broad ages examined (Luo et al., 2016) and it reliably distinguishes between individuals with BN versus controls (von Ranson et al., 2005). We focused on binge eating rather than binge-related disorders. Samples sizes for disorders were too small for analysis, but binge eating appears to be dimensional rather than categorical in nature (Luo et al., 2016), and heritabilities of binge eating and binge-related disorders are similar (e.g., Bulik, Sullivan, & Kendler, 1998). Clearly, more studies of clinical disorders with interview-based measures are needed, but initial data suggest that results might extend to these disorders and measures as well.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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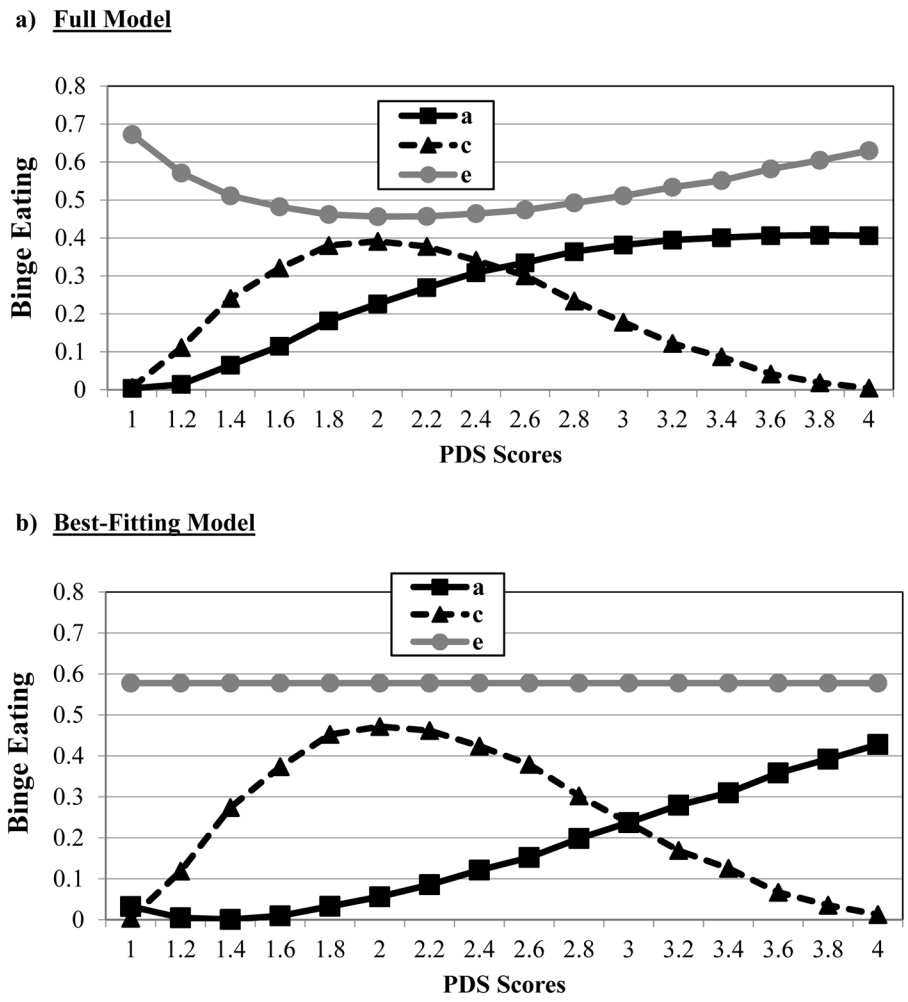

Figure 1. Unstandardized Estimates of Additive Genetic (a), Shared Environmental (c), and Nonshared Environmental (e) Influences from the a) Full Model and b) Best-Fitting Model across Pubertal Development Scale (PDS) Scores

Although log-transformed PDS scores that were floored to 0 were used in all models (see Methods), raw PDS scores are depicted here for ease of interpretation. The best-fitting model constrained the quadratic genetic moderator and all of the nonshared environmental moderators to 0.

Table 1

Model Fit Comparisons.

Model	-2lnL (df)	χ^2_Δ (df)	*p*	AIC	BIC	Sample-Size Adjusted BIC	DIC
Full Model	4274.59 (1538)	--	--	1198.59	-2981.73	-539.78	-1568.40
Nested Submodels:							
Constrain all A mods to 0	4282.25 (1540)	6.66 (2)	.02	1202.25	-2984.56	-539.43	-1569.39
Constrain A quadratic mod to 0	4275.24 (1539)	0.65 (1)	.42	1197.24	-2984.73	-541.20	-1570.49
Constrain C quadratic mod to 0	4279.16 (1539)	4.57 (1)	.03	1201.16	-2982.77	-539.23	-1568.52
Constrain all E mods to 0	4278.71 (1540)	4.12 (2)	.13	1198.71	-2986.32	-541.20	-1571.16
Constrain A quadratic and all E mods to 0	**4278.83 (1541)**	**4.24 (3)**	**.24**	**1196.83**	**-2989.59**	**-542.88**	**-1573.51**
Drop all mods	4304.00 (1544)	29.41 (6)	<.0001	1216.00	-2986.99	-535.52	-1568.15

Note. −2lnL = minus twice the log-likelihood; χ^2_Δ = chi-square change; AIC = Akaike information criterion; BIC = Bayesian information criterion; DIC = deviance information criterion; Full Model = model with paths, linear, and quadratic moderators; A = additive genetic effects; C = shared environmental effects; E = nonshared environmental effects; mod(s) = moderator(s); quadratic = non-linear increases/decreases in effects; Constrain all A mods to 0 = model in which both the linear and quadratic (i.e., nonlinear) additive genetic moderators were constrained to 0; Constrain A quadratic mod to 0 = model in which just the quadratic (i.e., non-linear) additive genetic moderator was constrained to 0; Constrain all C mods to 0 = model in which both the linear and quadratic (i.e., non-linear) shared environmental moderators were constrained to 0; Constrain all E mods to 0 = model in which both the linear and quadratic (i.e., non-linear) nonshared environmental moderators were constrained to 0; Constrain A quadratic and all E mods to 0 = model in which the quadratic (i.e., non-linear) additive genetic moderator and all of the nonshared environmental moderators (i.e., linear and quadratic) were constrained to 0; Drop all mods = model in which the linear and quadratic moderators for additive genetic, shared environmental, and nonshared environmental influences were constrained to 0. Each nested submodel is compared to the full model when calculating the χ^2_Δ and degrees of freedom. The best-fitting model, as determined by a non-significant chi-square change test and the lowest AIC, BIC, sample-adjusted BIC, and DIC values, is noted by cell borders and bolded text. Unstandardized estimates (95% confidence intervals) from the best-fitting model is included here, with significant effects noted in bolded text: additive genetic path (a) = −.17 (−.49, .36), shared environment path (c) = .06 (−.28, .31), nonshared environment path (e) = −.76 (−.81, −.72), linear genetic moderator (β_X) = 1.39 (.47, 1.98), linear shared environment moderator (β_Y) = 4.09 (2.06, 5.92), linear nonshared environment moderator (β_Z) = 0, quadratic genetic moderator (β_{X^2}) = 0, quadratic shared environment moderator (β_{Y^2}) = −6.67 (−9.84, −3.45), quadratic nonshared environment moderator (β_{Z^2}) = 0.