

Environmental and genetic pathways between early pubertal timing and dieting in adolescence: distinguishing between objective and subjective timing

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Background. Early pubertal timing in girls is associated with elevated risk for dieting and eating pathology. The relative importance of biological *versus* socio-environmental mechanisms in explaining this association remains unclear. Moreover, these mechanisms may differ between objective measures of pubertal development and girls' subjective perceptions of their own maturation.

Method. The sample comprised 924 sister pairs from the National Longitudinal Study of Adolescent Health. Objective pubertal timing (menarcheal age), girls' perceptions of pubertal status and timing relative to peers, dieting and disordered eating behaviors were assessed during a series of confidential in-home interviews.

Results. Behavioral genetic models indicated that common genetic influences accounted for the association between early menarcheal age and increased risk for dieting in adolescence. In contrast, girls' subjective perceptions of their timing relative to peers were associated with dieting through an environmental pathway. Overall, subjective and objective measures of pubertal timing accounted for 12% of the variance in dieting.

Conclusions. Genetic differences in menarcheal age increase risk for dieting among adolescent girls, while girls' perceptions of their maturation represent an environmentally mediated risk.

Received 19 November 2010; Revised 2 May 2011; Accepted 10 May 2011; First published online 16 June 2011

Key words: Behavioral genetics, dieting, eating disorders, menarche, pubertal timing.

Introduction

Adolescence is a period of vulnerability for the onset of eating disorders, and clinical diagnoses are commonly preceded by subclinical symptoms. In particular, dieting has been implicated as a key feature of prodromal eating pathology (Stice *et al.* 2010). Dieting predicts risk for developing diagnostic levels of anorexic and bulimic symptomatology (Mussell *et al.* 1997; Stice *et al.* 1998, 2008; Patton *et al.* 1999; Stice, 2001; Keel *et al.* 2007) and is also concurrently associated with depressed mood and psychological distress (Casper & Offer, 1990; French *et al.* 1995; Neumark-Sztainer *et al.* 1997). Interest in understanding the precursors of eating pathology has been bolstered by recent evidence that identifying girls at high risk for the onset of future eating disorder diagnoses improves the efficacy of intervention and prevention programs (Stice *et al.* 2010).

One well-established risk factor for restrictive dieting in girls is early pubertal timing (e.g. Blyth *et al.* 1985; Graber *et al.* 1994; Keel *et al.* 1997; McCabe & Ricciardelli, 2004), which also predicts increased risk for eating disorder diagnoses (Fairburn *et al.* 1997; Ruuska *et al.* 2003). A major conceptual challenge for understanding the adverse impact of early pubertal timing is that puberty involves a complex, interconnected set of transitions across biological (e.g. hormonal, somatic and neural changes), psychological (e.g. cognition, affect and self-perception) and social (e.g. peer, parent and romantic relationships) domains. Moreover, early pubertal timing is itself influenced by 'upstream' biological and environment inputs. Thus, it is often difficult to discriminate which specific aspects of the pubertal transition are most important for the emergence of eating pathology.

Explanations for the relationship between pubertal timing and eating pathology most commonly emphasize socio-environmental mechanisms. Specifically, the maturation disparity hypothesis posits that puberty precipitates a cascade of new social challenges.

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Early maturing girls, because of their relative youth, have fewer cognitive and emotional resources with which to navigate these challenges (Ge & Natsuaki, 2009). Certainly, the relatively early development of secondary sex characteristics (and feelings of sexual attraction; McClintock & Herdt, 1996) promotes not only early initiation of romantic or sexual relationships, but also thinking of oneself and one's body as a potential object of romantic or sexual desire. This shift in behavior and thinking is hypothesized to result in greater risk for eating disorder symptoms. The physical changes of puberty, moreover, involve increasing adiposity and breast development, which create discrepancies between a post-pubertal girl's body shape and the 'thin ideal'. This may provoke body dissatisfaction and lower self-esteem (Graber *et al.* 1994; Stice, 2001), heightening disordered attempts at weight control to stop or reverse unwanted bodily changes. The discrepancy between the thin body ideal and pubertal maturation may be particularly accentuated for early maturers, as later developing peers are likely to display thinner, pre-pubertal shapes.

An alternative perspective is that the relationship between pubertal timing and eating disorder symptomatology is due to common underlying genetic risks. Some twin studies have estimated moderate to large heritabilities for eating disorder diagnoses (50–83%; Bulik *et al.* 1998, 2000), while others have estimated more modest genetic variance for anorexia symptoms (22%, Mazzeo *et al.* 2009). With regard to specific eating behaviors, dieting and weight loss have moderate (31–42%) heritability (Rutherford *et al.* 1993; Mazzeo *et al.* 2009). Pubertal timing is also moderately to strongly heritable (43–88%; Rowe, 2002; Mustanski *et al.* 2004; Ge *et al.* 2007). Initial molecular genetic research has identified genes related to ovarian hormone biosynthesis and metabolism (Gorai *et al.* 2003; Kadlubar *et al.* 2003; Guo *et al.* 2006; Mitchell *et al.* 2008) and ovarian hormone receptors (Stavrou *et al.* 2002, 2006; Long *et al.* 2005) as important for individual differences in age at menarche. Interestingly, ovarian hormone genes are also promising candidates for genetic influence on eating disorders (Klump & Culbert, 2007). Ovarian hormones predict changes in food intake (Asarian & Geary, 2006; Edler *et al.* 2007; Klump *et al.* 2008) and regulate the expression of genes in the serotonin system, which influences appetite and food intake (Rubinow *et al.* 1998; Bethea *et al.* 2002). In addition, Elks *et al.* (2010), in a meta-analysis of genome-wide association studies of age at menarche, found evidence for association with four genetic loci, which had been previously associated with adult body mass index, and three genetic loci, which were located in or near genes involved in energy homeostasis and body weight. This overlap in the specific

genes involved in the etiology of both eating-related outcomes and age at menarche suggests that the elevated rates of eating-related pathology in early maturing girls may be due, at least in part, to common genetic influences.

It is important to note that the question of how girls' risk for disordered eating is associated with pubertal timing is a different research question than how disordered eating is associated with pubertal status. This latter question focuses on puberty as a universal transition: What mechanisms make post-pubertal girls generally more vulnerable to eating disorder symptoms than pre-pubertal girls? In particular, behavioral genetic research by Klump and colleagues has shown that pubertal status moderates the genetic influences on disordered eating behaviors and attitudes, with negligible genetic influence in pre-pubertal females and strong genetic influences post-puberty (Klump *et al.* 2003, 2007; Culbert *et al.* 2009) – a pattern that may be due to rising levels of estradiol (Klump *et al.* 2010). While this line of research highlights the importance of biological mechanisms for understanding the relationship between within-person change in pubertal status and eating disorder symptoms, whether biological mechanisms underlie the impact of between-person differences in pubertal change remains unknown.

Understanding the relationship between early pubertal development and eating pathology is further complicated by ambiguity regarding how best to conceptualize and measure pubertal timing. Dorn and colleagues (2006) noted that many measures of pubertal timing employed in research show only modest levels of agreement and, in fact, tap distinct developmental constructs. Building off this work, we believe that it is important for research on the sequelae of early pubertal timing to distinguish between 'objective' pubertal timing, defined as a girl's actual age at pubertal maturation relative to the population as measured by a discrete and accurately assessed indicator (such as age at menarche), and 'subjective' pubertal timing, defined as a girl's perceptions of her pubertal status relative to her peers. Although girls' perceptions of pubertal development may not be biologically accurate, they may nevertheless be psychologically meaningful. A girl who perceives her body to be more mature than other girls her age may be at elevated risk for eating-related pathology, even if her development is not objectively early.

The current study used a longitudinal, family-based research design to investigate two research questions. First, to what extent is objectively early pubertal timing, as measured by age at menarche, associated with elevated risk for dieting in adolescence? Second, are girls' subjective perceptions of their pubertal

Table 1. Sibling pair correlations for age at menarche, self-rated pubertal development and peer comparison of pubertal development

	Age at menarche	Self-rated pubertal development	Peer comparison of pubertal development
MZ twins	0.61 (n=139)	0.38 (n=137)	0.55 (n=139)
DZ twins	0.31 (n=111)	0.29 (n=109)	0.27 (n=111)
Full siblings	0.29 (n=362)	0.17 (n=355)	0.09 (n=362)
Half siblings	0.18 (n=109)	0.08 (n=109)	0.08 (n=108)
Cousins	0.22 (n=65)	0.04 (n=64)	0.16 (n=64)
Non-biological siblings	0.08 (n=110)	0.04 (n=107)	0.00 (n=110)

MZ, Monozygotic; DZ, dizygotic.

Correlations significantly different than zero at $p < 0.05$ are shown in bold.

Numbers of complete pairs are shown in parentheses.

timing associated with dieting via different mechanisms than objective measures of pubertal timing?

Methods

Participants

Data were drawn from the National Longitudinal Study of Adolescent Health (AddHealth; Harris *et al.* 2006). Participants were identified using a stratified, school-based sampling design and school rosters were used to select a sample of adolescents ($n = 10\,480$ females; $10\,264$ males) who completed an in-home interview in 1994–1995 (Wave I, mean age = 16.12 years, $s.d. = 1.67$). Sensitive topics were assessed by having participants listen through earphones to audio-recorded questions and entering their answers directly into a laptop. Follow-up home interviews were completed in 1995–1996 (Wave II interview; age 11–23 years), 2001–2002 (Wave III; age 18–26 years) and in 2007–2009 (Wave IV; age 24–32 years).

The current sample comprises 1848 female participants from 924 sister pairs: 145 monozygotic twin pairs; 116 dizygotic twin pairs; 369 full sibling pairs; 117 half-sibling pairs; 65 cousin pairs raised as siblings; 112 non-biologically related pairs (e.g. step-siblings, adopted siblings). Twin pair zygosity was diagnosed through 11 molecular genetic markers and responses to four questionnaire items concerning physical appearance and frequency of being mistaken for one's twin (Harris *et al.* 2006). The socio-demographic composition of sibling pairs in AddHealth is comparable to the full sample (Jacobson & Rowe, 1999). Race/ethnicity were classified as white ($n = 984$, 53.2%), African-American ($n = 493$, 26.7%), Hispanic ($n = 245$, 13.3%) or other ($n = 126$, 6.8%).

Measures

Age at menarche

At Waves I and II, participants were asked whether they had experienced menarche ('have you ever had a menstrual period?') and, if so, during which month and year. At Wave III, participants were asked 'how old were you when you got your period for the first time?' We used participants' first reported age at menarche. Mean age of menarche in the sample was 12.17 years ($s.d. = 1.43$, range = 7.0–25.0 years). In total, 96% of the sample ($n = 1774$) reported an age at menarche that preceded the Wave I assessment; for these individuals, the mean duration between menarche and the Wave I assessment was 3.92 ($s.d. = 1.89$) years. For girls who had not yet experienced menarche at Wave I, the mean delay between Wave I and menarche was 1.19 ($s.d. = 1.03$) years.[†] Sibling pair correlations for all measures of pubertal timing are summarized in Table 1.

Subjective perceptions of pubertal development

Two subjective measures of pubertal development from the Wave I in-home interview were: (1) girls' peer comparisons; (2) girls' self-ratings. Peer comparisons were assessed with the item, 'How advanced is your physical development compared to other girls your age?' using a 5-point scale (1 = I look younger than most; 5 = I look older than most; mean = 3.31, $s.d. = 1.15$). Peer comparisons were significantly and negatively correlated with age at menarche ($r = -0.26$). Participants' self-ratings of pubertal development were assessed using two Likert scale items regarding breast size (1 = my breasts are about

[†] The notes appear after the main text.

Table 2. Predictive validity of dieting in adolescence for disordered eating outcomes in early adulthood

Eating outcome	Items	Sample statistics	Predicted by dieting in adolescence?
Binge eating	Eaten so much in a short period that you would have been embarrassed if others had seen you do it Afraid to start eating because you thought you wouldn't be able to stop or control your eating	8.9%	OR 1.5* (1.26–1.79)
Purging	Made yourself throw up Took laxatives Used diuretics (water pills)	1.2%	OR 3.18* (2.12–4.77)
Fasting	Fasted or skipped meals Took food supplements that are intended to reduce appetite or replace meals	16.7%	OR 1.84* (1.61–2.10)
History of ED diagnosis	Ever been told by a doctor that you have an eating disorder, such as anorexia or bulimia	3.6%	OR 1.62* (1.25–2.10)
BMI (kg/m ²)	Calculated from weight and height measured by interviewer	26.6 (6.8) ^a	$\beta = 3.73^*$ (3.41–4.17)

ED, Eating disorder; BMI, body mass index.

Eating outcomes measured at AddHealth Wave III (conducted in 2001–2002, age 18–26, mean age = 22 years).

^a Sample mean and s.d.

* Parameter significant at $p < 0.05$.

the same size as when I was in grade school to 5 = my breasts are a whole lot bigger than when I was in grade school; they are as developed as a grown woman's breasts; mean = 3.33, s.d. = 1.11) and body curviness (1 = My body is about as curvy as when I was in grade school to 5 = my body is a whole lot more curvy than when I was in grade school; mean = 3.36, s.d. = 1.09). To calculate a measure of subjective pubertal timing, we calculated the deviation of each participant's score from the mean level of development reported by adolescents of the same age and standardized this deviation score (mean = 0, s.d. = 1). Thus, higher scores reflect whether a girl perceives her current pubertal development as more or less advanced than is typically reported by other girls her age. The correlation between self-rated development and age at menarche was small, but in the expected direction ($r = -0.20$), with girls who reported earlier menarche also reporting greater perceived development. There was a significant and positive correlation between self-rated development and peer comparisons ($r = 0.39$).

Dieting

At Wave I, adolescents reported whether they were trying to lose weight and whether they had 'restricted food intake' in the past 7 days in order to lose weight or keep from gaining weight. Dieting was coded as a dichotomous variable, with adolescents who were trying to lose weight or stay the same weight and who had restricted food intake coded as 1 ($n = 344$, 19% of

the sample). It was found that < 1% of the sample had missing data on dieting ($n = 18$). To test the predictive validity of this brief measure of dieting for the development of disordered eating, we used the full sample of AddHealth women ($n = 10\,480$) to examine the phenotypic association between adolescent dieting and five eating-related outcomes measured at Wave III, when participants were in early adulthood (age 18–26 years, mean age = 22 years). As summarized in Table 2, participants who reported dieting in adolescence showed significantly higher odds of binge eating [odds ratio (OR) 1.51], purging (OR 3.18), fasting (OR 1.84) and having been diagnosed with an eating disorder by a physician (OR 1.62) in early adulthood, and had significantly higher adult body mass index. These analyses support dieting as an important index of vulnerability for future disordered eating.

Data analysis

Between- and within-family means comparisons

To estimate between- and within-family effects for menarche, we divided participants into two groups: early maturers (age at menarche ≤ 12 ; range = 7–12 years; mean = 11.3, s.d. = 0.88); late maturers (age at menarche > 12 ; range = 13–19 years; mean = 13.6, s.d. = 0.88).² Similarly, between-family and within-family effects for self-rated pubertal development and peer comparisons were estimated by dividing

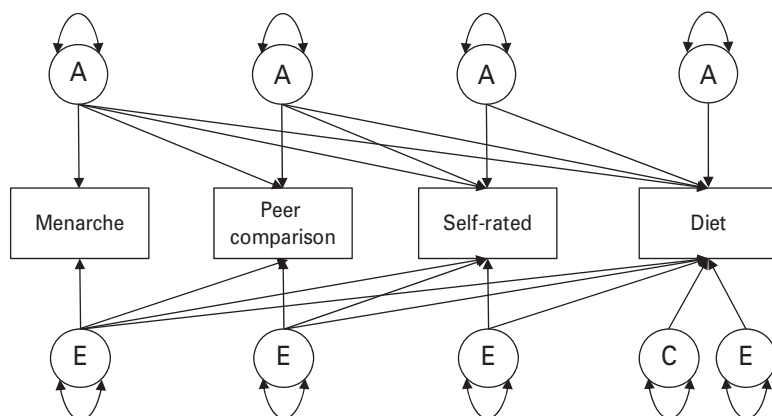


Fig. 1. Behavioral genetic model for age at menarche, perceived pubertal development and disordered weight control behaviors. A, Additive genetic; C, shared environment; E, non-shared environment. All A, C and E components are standardized (mean = 0, s.d. = 1). Only one sister per pair is illustrated. Correlations between A components in first and second sister per pair are fixed according to genetic theory (1.0 in monozygotic twin pairs, 0.5 in dizygotic twin and full sibling pairs, 0.25 in half-sibling pairs, 0.125 in cousins and 0 in non-biological related pairs). Correlation between C components is fixed to 1.0; correlations between E components are fixed to 0. Age and race/ethnicity used as statistical covariates in all models (not shown).

participants based on scores above or below the sample mean. The between-family effect was estimated by comparing the rate of dieting in girls from families where both sisters were concordant for early maturation with families where both sisters were concordant for late pubertal maturation. Thus, the between-family effect compares unrelated individuals and is comparable to epidemiological associations that do not control for genetic and environmental differences between families. In contrast, the within-family effect was estimated by comparing sisters who were discordant for early *versus* late maturation. [Sisters who were classified as discordant for early *versus* late menarche differed in their age at menarche, on average, by 2.13 (s.d. = 1.16, range = 1–7) years.] This tests whether a girl who experiences puberty earlier than her sister exhibits a correspondingly higher risk for dieting than her sister. To the extent that sisters are genetically similar, this within-family comparison controls for genetic differences between families (Dick *et al.* 2000), plus all environmental factors shared by sisters raised in the same home (e.g. race/ethnicity, socio-economic status, family structure, etc., commonly referred to as the shared environment). If the magnitude of the within-family effect is attenuated relative to the between-family effect, it suggests that genetic and/or environmental factors shared by siblings in the same family account for the association between pubertal timing and dieting. In contrast, a significant within-family effect indicates that the association between pubertal timing and dieting persists even after a rigorous control for genetic and shared environmental background factors, as would be predicted by the maturation-disparity hypothesis.

Behavioral genetic model

Behavioral genetic models decompose variance in a given phenotype into three components: additive genetic effects (A); shared environmental effects (C); non-shared environmental effects (E) (Neale & Cardon, 1992). The full behavioral genetic model is illustrated for one twin per pair in Fig. 1. Previous analyses of this dataset (Ge *et al.* 2007; Harden & Mendle, in press) found that shared environmental influences on both measures of pubertal timing were minimal and could be fixed to zero without significant decrement in model fit. This minimal contribution of the shared environment is evident in the sibling pair correlations for menarche and perceived development (Table 1). Thus, only additive genetic and non-shared environmental influences on pubertal timing were estimated. The key parameters in this model are the regressions of dieting on the A and E components of the three measures of pubertal timing. The regressions of dieting on the A components test whether genes influencing the timing of pubertal development also influence girls’ propensity for dieting. For example, if genes related to ovarian hormone receptors influenced both age at menarche and risk for dieting, this common genetic influence would be reflected in the regressions of dieting on the A component of age at menarche. In contrast, the regressions on the E components of each measure of pubertal timing test whether sisters who differ in their pubertal timing also differ in their dieting. If, as predicted by the maturation-disparity hypothesis, the association between early pubertal timing and elevated risk for eating-related pathology is due to

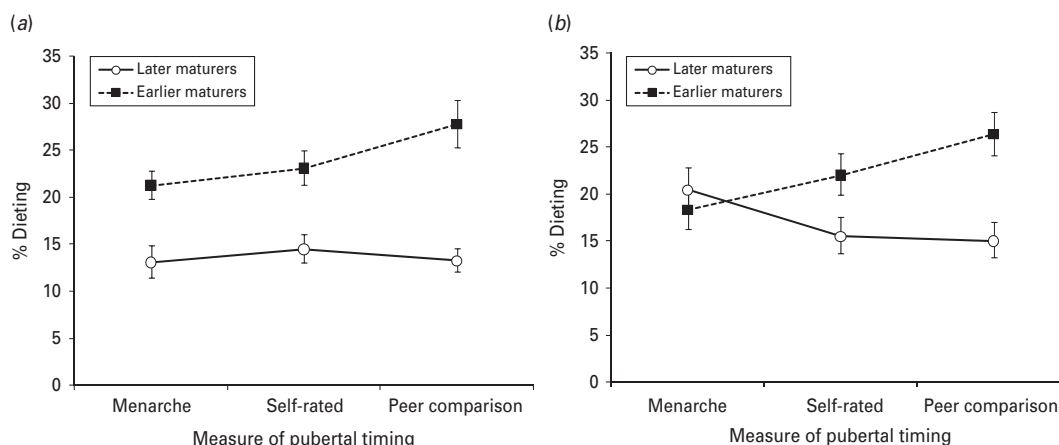


Fig. 2. (a) Between-family and (b) within-family effects of pubertal timing on dieting, by measure of pubertal development. Girls classified as earlier maturers based on age at menarche <12 years; self-rated pubertal development greater than the mean for chronological age; peer comparison >3 (look older than some/most other same-aged girls). Bars represent ± 1 s.e. All comparisons between earlier maturers and later maturers are statistically significant at $p < 0.05$, except for the within-family effect of menarche.

socio-environmental experiences, this would be reflected in the E path.

All models included race/ethnicity and chronological age as statistical covariates and were fit using the statistical program MPlus (Muthén and Muthén, 1998–2010). Model fit was evaluated using root mean square error of approximation (RMSEA) (Steiger, 1990; Browne & Cudeck, 1993). RMSEA values up to 0.08 represent reasonable errors of approximation.

Results

Between- and within-family means comparisons

The between-family effects are shown in Fig. 2a and the within-family effects are shown in Fig. 2b. Consistent with previous epidemiological research, girls from families where both sisters were early maturers were significantly more likely to report dieting than girls from families where both sisters were later maturers. This between-family effect, which does not control for genetic and environmental background factors, was consistent across all measures of pubertal timing; regardless of whether a girl was classified as early maturing according to menarcheal age, self-reported pubertal development, or peer comparisons, earlier pubertal timing was associated with greater propensity for dieting.

A different pattern of results was evident for within-family effects, which compare sisters discordant for earlier *versus* later pubertal maturation. Most notably, sisters discordant for early menarche did not significantly differ from each other with regard to dieting (18.3% for earlier menarche *versus* 20.4% for later

menarche; likelihood ratio $\chi^2 = 0.48$, $p = 0.49$). Thus, after controlling for background risks among sisters raised in the same home, the association between menarcheal age and dieting was no longer evident. In contrast, there were significant within-family effects for both girls' self-reported pubertal development (likelihood ratio $\chi^2 = 13.16$, $p < 0.01$) and girls' peer comparisons of pubertal development (likelihood ratio $\chi^2 = 14.37$, $p < 0.01$). Taken together, these results suggest that different measures of pubertal timing may be associated with elevated risk for dieting via different mechanisms, with girls' subjective perceptions of puberty associated with dieting via a non-shared environmental pathway and menarche associated with dieting via common genetic influences.

Behavioral genetic model

Standardized parameter estimates from the behavioral genetic model are summarized in Table 3 (comparative fit index = 0.88, RMSEA = 0.04). Individual differences in age at menarche were influenced by both genetic and non-shared environmental factors, with a heritability of 60% ($h = 0.78$). The commonality between age at menarche and the subjective measures of pubertal timing was entirely due to genetic influences ($A_{\text{menarche}} \rightarrow \text{peer comparison} = -0.36$; $A_{\text{menarche}} \rightarrow \text{self-reported puberty} = -0.20$); the non-shared environmental paths between objective and subjective measures of pubertal timing were not significant. In other words, the environmental influences on girls' subjective ratings of their own pubertal timing were independent of environmental influences on

Table 3. Standardized parameter estimates from behavioral genetic model of pubertal timing and dieting

	Age at menarche		Peer comparison		Self-rated puberty		Dieting	
	A	E	A	E	A	E	A	E
Age at Menarche	0.78* (0.03)	0.63* (0.04)						
Peer Comparison	-0.36* (0.05)	0.03 (0.06)	0.51* (.05)	0.78* (0.04)				
Self-Rated Puberty	-0.20* (0.06)	-0.09 (0.06)	0.30* (0.08)	0.17* (0.05)	0.49* (0.06)	0.77* (0.03)		
Dieting	-0.21* (0.08)	0.13 (0.10)	-0.04 (0.12)	0.19* (0.07)	0.07 (0.13)	0.12 (0.08)	0.56* (0.10)	0.71* (0.14)

Parameters on the diagonal represent the paths from the genetic and environmental components of each phenotype. Each off-diagonal parameter represents the regression of the row variable on the genetic or environmental component of the column variable. Parameters in bold font (also noted with *) were significant at $p < 0.05$.

menarcheal age. The genetic path between menarche and dieting was significant ($A_{menarche} \rightarrow dieting = -0.21, p = 0.01$), whereas the non-shared environmental path was not ($E_{menarche} \rightarrow dieting = 0.13, p = 0.22$). This pattern of results indicates that the elevated risk for dieting seen among ‘objectively’ early maturing girls can be entirely attributed to common underlying genetic risks that influence both phenotypes. Overall, 4.4% of the variance in dieting could be attributed to genetic variation in menarcheal age.

The residual variance (unique of menarcheal age) in peer comparisons of pubertal timing was due to both genetic (30%) and non-shared environmental (70%) influences (Table 3, column 2). Peer comparisons of pubertal timing were associated with dieting only through a non-shared environmental path ($E_{menarche} \rightarrow dieting = 0.13, p < 0.05$), while common underlying genetic influences did not contribute to the association between peer comparisons of pubertal timing and dieting. Finally, as summarized in the third column of Table 3, unique variation in self-rated pubertal development was predominantly due to non-shared environmental differences between siblings (71%), plus some genetic variance (29%). Notably, self-rated pubertal development, when controlling for age at menarche and girls’ peer comparisons, did not uniquely predict dieting. Overall, subjective and objective measures of pubertal timing accounted for 12% of the total variance in dieting in adolescence (calculated as the sum of the squared paths from the genetic and environmental components of the three measures of pubertal timing).

Discussion

Girls who mature earlier than their peers are at risk for excessive dieting and other forms of disordered eating. However, understanding the mechanisms underlying these associations has been hampered by difficulty discriminating between genetic vulnerabilities that precipitate early maturation and the social environments faced by early maturing girls. By comparing sisters of varying degrees of genetic relatedness, the current study offers a more nuanced understanding of the relative roles of environmental experience and genetic risk in the association between pubertal timing and dieting in adolescence. Overall, our results suggest that early maturing girls face dual sources of risk. First, the same genes that predispose girls to objectively early maturation also increase dieting. Second, to the extent that girls perceive their own bodies to be more mature than their peers’, this peer comparison confers an additional, environmentally mediated risk for dieting.

Specifically, our results indicate that the association between menarcheal age and dieting in adolescence is entirely due to common underlying genes. This pattern of results suggests that objectively early pubertal maturation is a marker for underlying genetic vulnerabilities. Genes involved in ovarian hormone synthesis and hormone receptors are promising candidates for explaining this association, as previous research has linked ovarian hormone genes to both eating disorders (Klump & Culbert, 2007) and food intake (Asarian & Geary, 2006; Edler *et al.* 2007; Klump *et al.* 2008), as well as individual differences in menarche (e.g. Stavrou *et al.* 2002, 2006; Gorai *et al.* 2003; Mitchell *et al.* 2008).

In contrast, subjective pubertal timing – whether a girl perceives herself as more pubertally advanced than other girls her age – is associated with dieting via a non-shared environmental pathway. There are myriad non-shared environmental experiences that may contribute to this association, and exploring mediators would be a fruitful avenue for future research. One hypothesis is that the girls' who perceive themselves (accurately or inaccurately) as more physically mature than their peers may be more likely to pursue dating or sexual relationships, which have been identified as important for the association between pubertal timing and eating-related problems (Smolak *et al.* 1993; Cauffman & Steinberg, 1996). Alternatively, these non-shared environmental effects could be mediated by body dissatisfaction, provoked by perceived differences between oneself and one's peers.

In addition to peer comparisons, the current study also examined girls' self-rated level of pubertal development. Although clearly related, there are important conceptual differences between these subjective measures of pubertal timing. The peer comparison measure represents the extent to which a girl perceives herself as more mature than she perceives other girls. The self-rated measure represents the extent to which a girl perceives herself as more mature than other girls her age perceive themselves. Notably, the peer comparison measure had the strongest association with dieting in adolescence, whereas self-rated pubertal development was not uniquely associated with dieting. This suggests that a girl's perception of between-person differences (e.g. 'I am more developed than other girls') may be a more important determinant of environmental risk for disordered eating than a girl's perception of within-person change ('I am more developed than I used to be').

We found minimal effects of the shared environment on both objective and subjective pubertal timing. Certainly, there has been a secular decline in the average at menarche among girls in industrialized

nations (e.g. Hwang *et al.* 2003), indicating the importance of macro-environmental determinants of pubertal timing. The lack of shared environmental influence in the current study may be due to minimal between-family variation in nutritional status. That is, within a modern US sample, being sufficiently underweight to delay menarche may reflect within-family differences in dietary restriction or athletic pursuits rather than between-family differences in wealth or access to adequate nutrition.

The AddHealth participants were older than is typical for a study of pubertal development; thus, objective pubertal timing was measured using retrospective reports of age at menarche. However, bodily changes important for the development of dieting (e.g. breast changes, increases in body weight) occur months or years before menarche. This temporal gap may account, in part, for the modest agreement between girls' self-rated pubertal development and their age at menarche. At the same time, the age of the AddHealth participants suggests that the effects of early pubertal timing may be relatively enduring in adolescence, as a number of previous studies have suggested (e.g. Graber *et al.* 2004; Zehr *et al.* 2007). Zehr *et al.* (2007) hypothesized that the relatively long-term effects of early pubertal timing were due to the organizational effects of gonadal steroid hormones on brain development during adolescence. This explanation suggests the possibility that early pubertal timing activates enduring genetic vulnerabilities for eating-related problems. Previous behavioral genetic studies have found that pubertal status modifies genetic influences on disordered eating, with greater genetic variance evident among post-pubertal *versus* pre-pubertal adolescents. However, no previous study has tested a moderation effect with pubertal timing, i.e. whether early maturing girls show persistently higher genetic variance in disordered eating than girls who mature at a later chronological age.

The measures of eating behavior in AddHealth are limited by their brevity. Obviously, there are important distinctions between occasional dieters and adolescents who severely restrict their food intake. Future research is necessary to examine whether the current associations generalize to more extreme forms of restriction. Moreover, self-report measures of dietary restraint have poor concordance with actual caloric intake and may be more appropriately considered a measure of eating intentions (Stice *et al.* 2004). Nevertheless, consistent with previous prospective studies (Jacobi *et al.* 2004; Stice *et al.* 2010), the brief measure of adolescent dieting used in these analyses predicted a variety of disordered eating behaviors 7 years later, when participants were in early

adulthood, suggesting that the current analyses describe an outcome of clinical significance.

Current clinical interventions, including those for eating pathology, often seek to modify distorted self-perceptions to ameliorate symptom presentation. While this study indicates that genetic differences in the timing of pubertal maturation are important for girls' differential risk for dieting, it is also clear that girls' self-perceptions – particularly comparisons with peers – constitute an independent and environmentally mediated mechanism of risk for dieting among adolescent girls.

Acknowledgements

Dr Paige Harden is a Faculty Research Associate of the Population Research Center at the University of Texas at Austin, which is supported by a center grant from the National Institute of Child Health and Human Development (5-R24-HD042849). This research uses data from Add Health, a program project designed by J. Richard Udry, Peter S. Bearman and Kathleen Mullan Harris, and funded by a grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 17 other agencies. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Persons interested in obtaining data files from Add Health should contact Add Health, Carolina Population Center, 123 W. Franklin Street, Chapel Hill, NC 27516–2524 (addhealth@unc.edu). No direct support was received from grant P01-HD31921 for this analysis.

Declaration of Interest

None.

Notes

¹ Given that menarche occurs relatively late in the process of pubertal development, girls who had not yet experienced menarche were nevertheless already likely experiencing pubertal change. That is, pre-menarcheal is not synonymous with pre-pubertal. Thus, we consider information on girls' eventual age of menarche to be a meaningful index of pubertal timing even for girls who were, at the time of the Wave I assessment, pre-menarcheal. To check that the inclusion of girls who were pre-menarcheal at Wave I did not bias our results, we conducted a series of follow-up analyses that excluded these 74 participants. Results from means comparisons and behavioral genetic models were unchanged (complete results available from first author).

² Previous research has used various cut-offs to categorize 'early' pubertal maturation; we chose 12 years as the cut-off because it was close to the mean menarcheal age in the full sample, thus yielding groups of approximately equal size. This choice is consistent with classifications used by numerous previous studies (e.g. Stattin & Magnusson, 1990; Caspi & Moffitt, 1991; Dearthoff *et al.* 2005). Ultimately, any dichotomization of menarche into 'early' versus 'late' is to some degree arbitrary and our subsequent, more rigorous behavioral genetic analyses therefore examine both menarche and perceived development as continuous variables.

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