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Genetic and Environmental Influences on the Association between Pubertal Maturation and Internalizing Symptoms

Kristine Marceau,

The Pennsylvania State University

Jenae M. Neiderhiser,

The Pennsylvania State University

Paul Lichtenstein, and

Karolinska Institutet

David Reiss

Yale University

Abstract

The literature consistently shows an association between pubertal maturation and internalizing problems in girls. The association for boys is less clear. The present study examines genetic and environmental influences on the association between pubertal maturation and internalizing problems for boys and girls in two primarily Caucasian adolescent twin/sibling studies: The Swedish Twin study of CHild and Adolescent Development (706 same-sex twin pairs aged 13-14, $M=13.7$ years, 50% female), and the Nonshared Environment in Adolescent Development sample (US-based, 687 same-sex twin/sibling pairs aged 10-18, $M=13.6$ years, 47% female). For girls, more advanced pubertal maturation was associated modestly with more internalizing problems, and that association was entirely explained by shared environmental influences. For boys, the association between pubertal maturation and internalizing problems was weak and inconsistent. Results for girls were remarkably consistent across samples. Findings suggest that nongenetic mechanisms mediate the association between pubertal maturation and internalizing problems. Findings have implications for intervention such that environmental influences shared by twins/siblings may provide the best targets for intervention strategies designed to minimize the potential negative effects of pubertal maturation on internalizing symptoms in girls.

Keywords

Puberty; Internalizing; Quantitative Genetics

Early pubertal maturation has been linked to exacerbated psychological, social, and emotional problems (e.g., Caspi & Moffit, 1991; Dorn, Susman, & Ponirakis, 2003; Mendel, Turkheimer, & Emery, 2007), particularly clinical and subclinical levels of internalizing problems (i.e. anxiety and depression symptoms) for girls. However, it remains unclear

whether genetic or environmental mechanisms mediate this association. Puberty may affect internalizing problems in multiple ways. Observed increases in adrenal and gonadal hormones are associated positively with internalizing problems, including depression and disordered eating attitudes and behaviors (e.g., Angold, Costello, Erkanli, & Worthman, 1999; Klump, Keel, Culbert, & Edler, 2008). Social environments also change during puberty in ways that may exacerbate internalizing problems (Zahn-Waxler, Shirtcliff, & Marceau, 2008). The present study uses a quantitative genetic framework to test whether genetic or environmental influences mediate the association between pubertal maturation and internalizing problems. Understanding whether genetic or environmental influences mediate this association will help us to understand the mechanisms by which puberty and internalizing problems are associated, which may prove useful for identifying potential targets of interventions aiming to reduce internalizing problems during adolescence.

Associations between Pubertal Maturation and Internalizing Problems

We begin with a brief overview of the operationalization of pubertal maturation. There are multiple ways to conceptualize and measure puberty (see Dorn, Dahl, Woodward, & Biro, 2006). Pubertal development typically is described as a combination of maturational stage, timing, and tempo (Marshall & Tanner, 1969; Marshall & Tanner, 1970), though most studies examine only maturational stage or timing. *Maturational Stage* refers to the child's current degree of physical maturation, often indexed by level of breast (in girls), genital (in boys), and pubic hair development (in both boys and girls), and sometimes indexed by levels of pubertal hormones (i.e. testosterone, estradiol, and dehydroepiandrosterone) (e.g., Shirtcliff, Dahl, & Pollak, 2009). *Timing* describes the relative rank of an individual's stage of development compared with same age and sex peers (e.g., Ge, Natsuaki, Neiderhiser, & Reiss, 2007) or the age at which an individual enters a particular stage of maturation (e.g., Marceau, Ram, Houts, Grimm, & Susman, 2011). *Tempo* describes how quickly or slowly an adolescent progresses through pubertal development (e.g., Marceau et al., 2011; Susman, Houts, Steinberg, Belsky, Kaufmann, Dehart et al., 2010). Thus, the developmental course of puberty is captured through timing and tempo, while maturational stage assesses a child's current degree of pubertal maturation at any given moment.

Though there is some evidence that maturational stage may better predict internalizing problems (e.g., Angold, Costello, & Worthman, 1998), pubertal timing (usually operationalized as the relative rank of an individual's stage of development compared with same age and sex peers) has been most commonly examined in association with internalizing problems. For girls, earlier timing of puberty has been associated consistently with more internalizing symptoms (both symptoms of specific disorders on the internalizing spectrum and internalizing symptoms more broadly defined) than girls with average or later timing (e.g., Angold et al., 1998; Marceau et al., 2011; see Mendel et al., 2007 for review). For boys, findings are mixed. Some studies show that early and/or late timing is associated with more internalizing symptoms in boys (e.g., Ge, Brody, Conger, Simmons, McBride-Murray, 2006; Graber, Seeley, Brooks-Gunn & Lewinsohn, 2004). However, other findings show no association between pubertal timing and internalizing symptoms for boys (e.g., Canals, Mart-Henneberg, Fernandez-Ballart, & Domonech, 1995; Angold et al., 1998). Still other studies have shown that early timing is a protective factor for boys, as earlier pubertal

timing is associated with fewer internalizing symptoms (e.g., Stattin & Magnusson, 1990). Together, these findings suggest that early timing of puberty is a risk factor for more internalizing symptoms in girls, but the association between the timing of puberty and internalizing symptoms for boys is unclear.

However, maturational stage more cleanly assesses the developmental status of an adolescent, and may better capture the physiological changes of puberty. In girls, more advanced maturational status has been associated with increased depressive symptoms (e.g., Angold et al., 1998; Ge et al., 2006; Hayward, Killen, Wilson, Hammer, Litt, Kraemer et al., 1997). More mature genital development stage also has been associated with depressive and anxiety symptoms in boys (e.g., Ge et al., 2006; Susman, Dorn, & Chorusos, 1991). Again, findings are mixed, and more advanced maturational status also has been associated with fewer depressive symptoms in boys (Angold et al., 1998). Together across multiple measures, findings suggest that pubertal timing and maturational status are associated consistently with internalizing symptoms in girls, though there is no strong consensus in the literature about the association between pubertal timing or maturational status and internalizing symptoms in boys. In this manuscript we focus on the role of maturational stage in the development of internalizing problems, measured via secondary sex characteristic growth in boys and girls.

Explanations for the Association between Pubertal Maturation and Internalizing Symptoms

There are several theories devised to explain why pubertal maturation and internalizing problems are associated (e.g., Cyranowski, Frank, Young, & Shear, 2000; Nolen-Hoeksema & Girgis, 1994). Notably, hypotheses and supporting evidence for the association between puberty and internalizing problems encompass genetic, neuroendocrine, and socio/emotional explanations (e.g., Angold et al., 1998; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Paikoff & Brooks-Gunn, 1991). We review two distinct, but not competing, hypotheses here.

According to the hormonal influence hypothesis, changes in adolescent moods and behavior may be influenced directly by pubertal hormones (Ge & Natsuaki, 2009; Paikoff & Brooks-Gunn, 1991). The hypothalamic-pituitary-adrenal (HPA) and hypothalamic-pituitary-gonadal (HPG) axes are both implicated in changes in pubertal hormones. Adrenal androgens (i.e., dehydroepiandrosterone [DHEA]) initiate adrenarche through the HPA axis. Then, the HPG axis drives gonadarche via gonadotropins (luteinizing hormone [LH], follicle-stimulating hormone [FSH]) and gonadal steroids (estrogens, testosterone). Both adrenal and especially gonadal hormones play a role in depression during puberty (Angold et al., 1999). Since the largest increase in gonadal hormones occurs around the center of pubertal development (around Tanner Stage 3, on average 12.5-13 years old; e.g., Angold et al., 1999), samples with a mean age around 13 years would be optimal for testing the relation between maturational status and internalizing symptoms from a hormonal influence perspective. Hormone mechanisms may act through gene expression, alterations in the stress response system, or may be mediated by social and environmental influences (Ge & Natsuaki, 2009). Pubertal hormones, particularly estrogen, may have a direct impact on gene

expression in such a way that confers risk for internalizing problems (Joffe & Cohen, 1998). The extent to which genes regulating pubertal hormones also contribute internalizing symptoms would present as genetic influences on the association in quantitative genetic studies, though this has not been investigated. Hormones operating through changes in the stress response system or mediated by social/environmental influences would present as environmental influences in quantitative genetic studies. Thus, quantitative genetic studies can help to clarify the mechanisms of hormonal influence.

Changes in adolescents' social environments also contribute to the association between puberty and internalizing problems (Cyranowski et al. 2000, Nolen-Hoeksema & Girgis, 1994). For example, maturational status may drive increases in internalizing problems environmentally if others around an adolescent perceive overt physical changes in the adolescent and treat the adolescent differently. The adolescent may then develop internalizing problems because of increased strain or change in social relationships. This has been hypothesized to be especially influential for girls, as girls become more focused on relationships during adolescence (Cyranowski et al., 2000; Zahn-Waxler et al., 2008). This hypothesis generally has been supported empirically. For example, peer and teacher popularity and being the target of rumors has been shown to partially mediate the effect of early timing of puberty on internalizing problems in girls across adolescence (Reynolds & Juvonen, 2011). Further, girls who were more developed physically and had a greater response to an interpersonal stressor exhibited more internalizing problems (Natsuaki, Klimes-Dougan, Ge, Shirtcliff, Hastings, & Zahn-Waxler, 2009). Taken together, the hormonal influence hypothesis and changing social environments suggest that pubertal adolescents may show internalizing problems both for genetic and environmental reasons.

Genetic and Environmental Influences on Pubertal Maturation and Internalizing Problems

Pubertal Maturation

Quantitative genetic studies allow researchers to parse the variance in a phenotype into genetic influences (heritability), shared environmental influences (non-genetic influences that cause family members to be more similar), and nonshared environmental influences (non-genetic influences that make family members different from one another). Among quantitative genetic studies examining genetic and environmental influences on pubertal maturation the heritability of puberty is most typically measured in girls using age at menarche (used both as a measure of maturational status and timing), and findings suggest age at menarche is moderately-to-highly heritable (e.g., Kaprio, Rimpela, Winter, Viken, Rimpela, & Rose 1995, Doughty & Rodgers, 2000; Meyer, Eaves, Heath, & Martin, 1991). However, using age at menarche as an indicator of pubertal development excludes boys, for whom there is no comparable measure. Measuring maturational status via secondary sex characteristics has yielded evidence of moderate genetic and nonshared environmental, and small shared environmental influences in boys and girls (Dick, Rose, Pulkinnen, & Kaprio, 2001). For example, a latent measure of pubertal status comprised from multiple items measuring secondary sex characteristic growth showed substantial heritability, while there were genetic, shared, and nonshared environmental influences unique to specific secondary

sex characteristic (Mustanski, Viken, Kaprio, Pulkkinen, & Rose, 2004). Generally, though less frequently examined, maturational status also shows genetic and environmental influence.

Each method for capturing pubertal development measures different aspects of pubertal maturation and thus has a slightly different meaning (Dorn et al., 2006). Pubertal maturation variables are created in different ways in each study, and measured at different ages, which helps to explain differences in the sizes of estimates of genetic and environmental influences on pubertal maturation found in the literature. Nonetheless, these studies show that pubertal timing and maturational status are influenced by both genes and the environment (e.g., Dick et al., 2001; Doughty & Rodgers, 2000; Ge & Natsuaki, 2007; Kaprio et al., 1995; Meyer et al., 1991; Mustanski et al., 2004).

Internalizing Problems

Quantitative behavioral genetic studies on internalizing problems often have examined relatively normally developing samples, though some have used clinical samples with heightened levels of internalizing problems, specifically depressive symptoms. The few studies examining extreme groups have yielded inconsistent evidence for whether genetic and environmental influences are different among youth with clinical levels, as opposed to normative or sub-clinical levels, of depressive symptoms (Rice & Thapar, 2009). Generally, there are moderate genetic influences (40-60%) on depressive and anxiety symptoms in childhood and adolescence, though estimates vary widely (see Rice & Thapar, 2009 for review). Further, there is consistent evidence of shared and nonshared environmental influences on both depressive symptoms and internalizing problems broadly (Rice & Thapar, 2009). Overall, studies show that variation in both genetic and environmental influences contribute to internalizing symptoms (i.e. some combination of anxiety and depressive symptoms). Genetic and environmental influences on internalizing problems have been reported previously for the samples used in this report. Genetic influences were substantial for a composite score of internalizing problems heavily weighted for depressive symptoms over anxiety symptoms in NEAD (70%), and the remaining variance was accounted for by nonshared environmental influences (29%) (O'Connor, McGuire, Reiss, Hetherington, & Plomin, 1998; Reiss, Neiderhiser, Heatherington, & Plomin, 2000). In the Swedish Twin Study of Child and Adolescent Development (TCHAD), Kendler, Gardener, & Lichtenstein (2008) reported a lower, but still substantial heritability (46%), significant shared environmental influences (32%), and similar nonshared environmental influences (22%) on internalizing problems broadly (fairly evenly assessed depressive and anxious symptoms). Taken together, there are generally genetic, shared, and nonshared environmental influences on internalizing problems during adolescence. Together, findings from quantitative studies suggest that both pubertal maturation and internalizing problems of varying severity are influenced by genes and the environment. Thus, the association between pubertal maturation and internalizing problems may be due to shared genes or environments.

Association between Pubertal Maturation and Internalizing Problems

Although there is a sizable literature on associations between puberty and internalizing problems, there has been little work in this area using genetically sensitive designs. Quantitative genetic designs can help to identify the source of associations. If common genetic influences are found for maturational status and internalizing problems, the same genes must regulate both puberty and internalizing problems. Alternatively, pubertal development may directly impact gene expression regulating internalizing problems, an indirect test of one mechanism of hormonal influence. If common environmental influences are found, then these genetic explanations are not supported. Instead, the influence of pubertal hormones must act through social and environmental mechanisms to affect internalizing problems. Depending on the type of environmental influences (shared or nonshared) underlying the association, different specific environmental factors may be identified which, if changed, break the association between advancing pubertal maturation and increasing internalizing symptoms during adolescence. Since the hypothesized genetic and neuroendocrine mechanisms driving this association apply to the maturational course of puberty, we have chosen to examine pubertal status rather than pubertal timing in the present study.

Only two genetically informed studies have examined both puberty and internalizing problems. The first showed that pubertal maturation moderates genetic influences on disordered eating attitudes and behaviors (Klump, Perkins, Burt, McGue & Iacono, 2007). The second showed that pubertal maturation moderates environmental influences on depressive symptoms (Edwards, Rose, Kaprio, & Dick, 2010), controlling on the associations between pubertal maturation and each phenotype. To date, no studies have examined whether genetic and/or environmental influences underlie the association between pubertal maturation and internalizing problems.

Present Study

The present study tests genetic and environmental mechanisms underlying concurrent and longitudinal associations between pubertal maturation and internalizing symptoms from mid-adolescence to young adulthood. We had three aims. First, we tested whether maturational status and internalizing problems were associated concurrently. Second, we tested whether puberty predicted later internalizing problems. Finally, we examined genetic and environmental influences on the association between maturational status and internalizing problems for boys and girls. We hypothesized that more advanced maturational status would be associated with more internalizing problems and that this association would be stronger for girls than boys, consistent with past research. We also hypothesized that the association between maturational status and internalizing problems would be attributable to both genetic and shared environmental influences, reflecting both the hormonal influence hypothesis and the changing social environments hypothesis. Because of sex differences in puberty, levels of internalizing problems during adolescence, and in the patterns of findings regarding the association between puberty and internalizing problems in extant literature, we chose to analyze boys and girls separately. Thus, the present study extends past research on the associations between puberty and internalizing problems by examining genetic and

environmental influences on the association between maturational status and internalizing symptoms in two adolescent twin/sibling samples.

Method

Participants

The present study utilizes two samples, the Nonshared Environment in Adolescent Development (NEAD) and the Swedish Twin study of CHild and Adolescent Development (TCHAD). The NEAD sample comes from a US-based twin and sibling study in which youth were assessed in middle adolescence, late adolescence and young adulthood. The TCHAD sample comes from a large Swedish population-based sample of same and opposite sex twins measured in childhood, mid-, and late- adolescence. Each study has strengths and weaknesses. For example the limited age range in TCHAD is optimal for the present hypotheses. However, the inclusion of non-twin siblings in NEAD allows for greater generalizability of findings, and the measures of internalizing problems are better in the NEAD sample. Replicating results across disparate studies adds strength to findings. Together, these studies help to provide a more complete picture of the genetic and environmental architecture underlying this association.

Nonshared Environment in Adolescent Development Study (NEAD) Sample—

The NEAD sample consists of 721 families of White (> 94%) twins and siblings. Most of the families were recruited through a national market survey of 675,000 families, though some were recruited through random digit dialing of 10,000 telephone numbers throughout the United States (Reiss et al., 2000). Zygosity was established using a validated questionnaire on which adolescent twins were rated for physical similarity (Nichols & Bilbro, 1966). Approximately 6% of the twin sample was excluded because they could not be classified with certainty. The analysis sample consists of 687 families: same-sex monozygotic twins (MZ: 46 male, 42 female), dizygotic twins (DZ: 50 male, 47 female), and full siblings (FI: 47 male, 44 female) in non-divorced families, and full siblings (FS: 86 male, 89 female), half siblings (HS: 60 male, 47 female), and genetically unrelated siblings (US: 74 male, 55 female) in step families. The analysis sample was slightly reduced because of missing data on either pubertal development or internalizing problems in one or both twins (N=34 pairs).

We used the first wave assessment in the current study, when adolescents were 10 to 18 years old ($M = 13.6$ years; $SD = 3$ years), and the third wave of assessment (aged 20-35, $M = 26.1$, $SD = 2.5$). Siblings were required to be the same sex, within 4 years of age of each other ($M = 1.6$ years; $SD = 1.3$ years), and to live in the same household for at least 5 years. At the first wave of assessment, parents varied in their level of education: 7% of mothers and 10% of fathers did not complete a high school education, while 42% of mothers and 35% of fathers completed high school, and 51% of mothers and 55% of fathers completed at least some post-high school education. These groups were combined into “high school or less” and “post-high school” categories in order to have categories comparable to education levels in the second sample in Table 1.

Socio-economic status was measured via mothers' and fathers' income and occupation at the first wave of assessment. Families participating in NEAD were primarily middle-class (on average earning \$25,000 to \$35,000 per year); 12% of families earned less than \$20,000/year, while 32% of families earned more than \$50,000/year. Demographic information was recoded using the Hollingshead Index (Zill & Furstenberg, 1981) and combined into a five-level social strata variable: (1) unskilled laborers, menial service workers; (2) machine operators, semiskilled workers; (3) skilled craftsmen, clerical, sales workers; (4) medium business, minor professional, technical; and (5) major business and professional. The first and second categories are combined in Table 1 in order to create comparable social strata categories in each sample. Additional sample and demographic information can be found in Reiss et al., (2000) and Neiderhiser, Reiss, & Hetherington (2007).

The Swedish Twin Study of Child and Adolescent Development (TCHAD)

Sample—The TCHAD sample consists of 915 White families of twins from the second wave of data collection, when all twins were 13-14 years of age. TCHAD is a study of all twins born in Sweden from May, 1985 to December, 1986. 1480 twin pairs were followed, assessed at four times starting in 1994 (time 1: 8-9 years of age, time 2: 13-14 years of age, time 3: 16-17 years of age, and time 4: 19-20 years of age). Of the 1480 twin pairs recruited, 75% of parents participated in wave 1 (91% after a follow up telephone interview), and 73% of parents and 78% of adolescents participated in wave 2. Participants who did not participate in Wave 2 did not differ from those who did on key variables, including sex ratio and externalizing symptoms (Lichtenstein, Tuvblad, Larsson, & Carlstrom, 2007). An independent samples t-test showed that participants who did and did not participate in Wave 2 had similar levels of internalizing problems at Wave 1, $t(1473) = 1.55, p = .12$.

Zygosity for same-sex twins was determined using parent and youth report separately on four questions about the physical similarity of the twins, and augmented by algorithms based on 106 twin pairs whose zygosity was determined previously in a clinical trial using 16 polymorphic DNA markers (see Lichtenstein et al., 2007 for more information on recruitment and zygosity information). The analysis sample consists of 706 families: same-sex MZ (207 male, 204 female pairs) and DZ twin pairs (145 male, 150 female pairs). The analysis sample is reduced because of missing data in either pubertal maturation or internalizing problems in one or both twins, and because opposite sex twin pairs ($N=209$) were excluded.

Twins were 12.8-14.5 years old ($M=13.7, SD=.47$) at the wave 2 assessment. Parents varied in educational levels: 21% of mothers and 31% of fathers completed compulsory school (9 years), 29% of mothers and 23% of fathers completed two years in a higher secondary school, 15% of mothers and 17% of fathers completed three-four years in a higher secondary school, and 36% of mothers and 29% of fathers completed a University degree. The first two categories are combined to create a category complementary to US high school education and less, and the higher two categories are grouped to form a category complementary to US post- high school education in Table 1.

Socio-economic status was coded into five classes: (1) unskilled blue collar (i.e. in production of goods, not specially trained; in production of services, not specially trained);

(2) skilled blue-collar or low-level white collar (i.e. in production of goods, specially trained; in production of services, specially trained; white collar, lower level, category I; white collar, lower level, category II); (3) mid-level white collar (i.e. white collar, intermediate level); (4) high-level white-collar (i.e. white collar, upper level; managerial capacity; self-employed, academic work); and (5) unspecified (i.e. self-employed; farmer; home wife or similar; student; retired; disability or sick leave; relief work). Detailed demographic and sample recruitment and assessment information can be found in Lichtenstein et al. 2007.

Measures

Internalizing Symptoms—NEAD families were visited in their homes, where children and parents completed questionnaires and participated in a series of 10-minute videotaped interactions. The depressive symptom composite was used in the current study. This measure has been used in previous reports (Reiss et al., 2000). The composite score was created using unit weights to combine twin/sibling report on the Child Depression Inventory (CDI; Kovacs, 1992), the internalizing subscale of the Behavior Problems Index (BPI; Zill, 1988) and the depressive factor of the Behavior Events Inventory (BEI; Hetherington & Clingempeel, 1992), mother and father report on the internalizing subscale of the BPI and the CDI, and observer ratings of twin/siblings' depressed mood (Hetherington & Clingempeel, 1992). Each measure had acceptable reliability (α 's $> .65$). The composite also showed good reliability ($\alpha = .84$ for sibling 1, $\alpha = .81$ for sibling 2). Because the NEAD sample consists of adolescents who span a wide age range, we regressed age and age difference (for nontwin sibling pairs) out of internalizing scores, and the standardized residuals were used for all subsequent analyses (McGue & Bouchard, 1984).

At wave 3 of the NEAD study, internalizing problems were measured using self report on the Center for Epidemiological Studies Depression Scale (CES-D). Reliability for this measure was also good ($\alpha = .91$ for sibling 1, $\alpha = .92$ for sibling 2). Because of skewness, these scores were square root transformed. As with the composite score for wave 1, age and age difference were regressed out, and standardized residuals were used. See Table 2 for means and standard deviations for internalizing problems at each wave of assessment.

Families participating in the TCHAD study completed mailed questionnaires and telephone interviews. Parents and twins reported on twins' internalizing problems using the Child Behavior Checklist and Youth Self Report at each wave (CBCL, YSR; Achenbach, 1991). The internalizing subscales of the CBCL and YSR include items indexing withdrawal, somatic complaints, and anxiety/depression rated on a scale from 0 (not true) to 2 (often true). Internal consistency was good for both twins and parents at each wave of assessment ($\alpha > .83$). Parent and youth report on the internalizing subscale were averaged to create a composite internalizing score. See Table 2 for means and standard deviations for internalizing problems at each wave of assessment. Only parent report was available at wave 1. Parent and youth report were correlated at each subsequent assessment, $r = .38$, $p = .001$ for wave 2, $r = .42$, $p < .001$ for wave 3, $r = .40$, $p < .001$ for wave 4. Because of skewness, scores at each wave were square root transformed. As noted above, age was controlled for and the standardized residuals were used.

Maturation Status—Pubertal development was measured using youth reports on the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988) in both studies. The PDS consists of 5 questions for boys and girls, (height growth spurt, growth of body hair, and skin changes for boys and girls; breast growth and menstruation for girls; voice deepening and facial hair growth for boys). Shirtcliff and colleagues (2009) developed a method of recoding the PDS that was designed to weight items so that the scores would map best onto hormone and physical exam scores and to weight items according to the reliability of a child's perception on each particular item. As a result of this coding scheme, scores on each scale range from 1 to 5, mirroring Tanner stages, and youth may be classified between stages. A detailed description of the coding scheme and decision rules used to create the coding scheme is available upon request (see Shirtcliff, Dahl, & Pollak, 2009 for further details).

In both studies, maturational status was normally distributed. See Table 3 for the distribution of youth in each stage by age. Because age and maturational status are highly confounded, twins necessarily will be closer for maturational status than nontwin siblings who differ in age. Thus, as with the measures of internalizing problems, we controlled on age difference in the NEAD sample in order to correct statistically for differences in maturational status between sibling pairs that are a product of the difference in age when siblings are not twins. However, we did not control for the age of each sibling in either study because of the age-maturational status within individuals. Further, controlling for age on measures of maturational status would change our measure to a measure of pubertal timing (see Ge et al., 2007).

Analytic Strategy

Phenotypic Analyses—We tested concurrent associations between maturational status and internalizing problems during middle adolescence (wave 1 in NEAD; wave 2 in TCHAD). In TCHAD, because data were available at a previous wave, we also tested whether pubertal stage predicted concurrent internalizing problems controlling on previous internalizing problems by regressing internalizing problems at the first wave from internalizing problems at the second wave, and correlating the standardized residual with maturational status. Finally, we tested the correlations between maturational status and later internalizing problems (from wave 3 of NEAD and TCHAD, and wave 4 of TCHAD), controlling for internalizing problems at all previous assessments.

Biometric Analyses—Biometric genetic analyses parse the variance of a phenotype into additive genetic (A), shared environmental (C) and nonshared environmental (E) components by examining the similarity and differences of family members. By definition, twins or siblings who live together share 100% of their shared environment (nongenetic influences that make family members similar) and none of their nonshared environment (nongenetic influences that make family members different, including measurement error). Different sibling types share different average proportions of their segregating genes: monozygotic (MZ) twins share 100% of their genes, dizygotic (DZ) twins and full siblings share an average of 50% of their segregating genes, half-siblings share an average of 25% of

their segregating genes, and step-siblings (genetically unrelated siblings) do not share any genes.

Bivariate biometric analyses (Cholesky decompositions, see Figure 1) were conducted using OpenMx (Boker et al., 2010) to test whether the association between maturational status and internalizing problems could be attributed to genetic, shared, or nonshared environmental influences when the associations were significant. In bivariate biometric analyses, the association between the variables (maturational status and internalizing problems) is decomposed into genetic, shared, and nonshared environmental influences. Unique genetic, shared, and nonshared environmental influences are also estimated for each variable. The overall fit of the model was tested by -2 times the log-likelihood of data (-2lnL) and the Akaike's Information Criterion (AIC; Akaike, 1987). A nonsignificant -2lnL and χ^2/df ratios > 2 indicate a good model fit; lower AIC values also indicate better model fit.

We tested a full model estimating all genetic, shared, and nonshared environmental influences on maturational status, common to maturational status and internalizing problems, and unique to internalizing problems. We then systematically dropped paths that were not significantly different from zero (as judged by 95% confidence intervals). Model differences were explored using a nested model approach that compared the constrained models to the fully saturated model. Differences in chi-square estimates between models tested whether the constrained models resulted in a significant deterioration of fit. If a model removing paths did not result in a significant decrement in fit from the fully saturated model, we concluded that the path was no different from zero and therefore the model dropping those paths (the constrained model) was more parsimonious, therefore, the better fitting model. Results from both the full model and the best-fitting model are presented. Note this approach favors parsimony and can lead to over-fitting models. Thus, we present both full and reduced models for readers to interpret.

Results from the bivariate Cholesky decomposition were then converted into a correlated factors solution for further interpretation of results. In the correlated factors solution, latent genetic, shared environmental, and nonshared environmental influences are estimated for each phenotype and then the correlation between genetic factors, shared environmental factors, and nonshared environmental factors on the phenotypes are each estimated. For example, the genetic correlation between maturational status and internalizing problems was specified as:

$$r_g = \frac{a_{21}a_{11}}{\sqrt{a_{11}^2 * (a_{21}^2 + a_{22}^2)}} \quad (1)$$

Where r_g is the correlation between the latent genetic factors estimated for maturational status and internalizing problems, a_{11} is the unstandardized path estimate for genetic influences on maturational status, a_{21} is the unstandardized path estimate for genetic influences common to maturational status and internalizing problems, and a_{22} is the unstandardized path estimate for genetic influences unique to internalizing problems. The same equation was applied to shared and nonshared environmental influences, respectively.

The genetic correlation (r_g) is interpreted as the amount of overlap in genetic influences on each phenotype. So, for example, a high genetic correlation would indicate a large overlap in the genes influencing each phenotype. A genetic correlation of 1 would indicate that 100% of the genes influencing the first phenotype also influence the second phenotype, and that there are no additional genes influencing the second phenotype.

Model Assumptions—These types of behavioral genetic decompositions are based on the assumption that shared and nonshared environmental effects are the same across sibling types and that assortative mating (i.e., nonrandom mating that could result in suppressed genetic influences because DZs and full siblings would share more than 50% of their genes) is not occurring. These assumptions previously have been tested and generally have been found to be valid in these samples and others (Kendler, Myers, Prescott, & Neale, 2001; Pike, McGuire, Hetherington, Reiss, & Plomin, 1996). Further, there were no systematic differences for the validity of equal twin and sibling environments in NEAD (Reiss et al., 2000), indicating that the equal environments assumption is reasonable for the current study. We also are assuming that the order of influence of predictors flows in the direction previously specified based on the literature explaining how pubertal development impacts internalizing problems.

Results

Phenotypic Analyses

We first examined zero-order correlations to test whether maturational status and internalizing problems was associated concurrently for boys and girls (see Table 4). Girls' maturational status and internalizing problems were correlated modestly in the NEAD sample, $r=.14$, $p<.05$, and the TCHAD sample $r=.09$, $p<.05$. Boys' maturational status and internalizing problems were correlated inversely in the NEAD sample, $r=-.09$, $p<.05$, but were uncorrelated for boys in the TCHAD sample: $r=.02$, $p=.58$.

For girls in TCHAD, maturational status was associated with concurrent internalizing problems after accounting for previous internalizing problems, $r = .13$, $p < .01$. Maturational status and concurrent internalizing problems remained uncorrelated for boys in TCHAD after accounting for previous internalizing problems, $r = .01$, $p = .82$. In both the NEAD and TCHAD samples, maturational status did not predict later internalizing problems, r 's $< .05$, $ps > .05$. Because the phenotypic correlations for boys were weak and inconsistent in the present report and in previous studies, we conducted bivariate biometric analyses only for girls.

Bivariate Biometric Analyses

Bivariate biometric analyses were conducted to examine genetic and environmental influences on the association between maturational status and internalizing problems for girls in both samples. Model fit statistics for both sets of analyses are presented in Table 5.

Bivariate model fitting results were remarkably consistent. The best fitting model included genetic, shared, and nonshared environmental influences on maturational status, only shared environmental influences common to maturational status and internalizing problems, and

unique genetic and nonshared environmental influences on internalizing problems. Parameter estimates are presented in Table 6 for girls in the NEAD and TCHAD samples. For girls in both samples, all of the variance common to maturational status is attributable to shared environmental influences and the shared environmental influences common to maturational status and internalizing problems account for all of the shared environmental influences on internalizing problems (i.e. there were no unique shared environmental influences on internalizing problems).

Finally, unstandardized parameter estimates from the full models were then converted into correlated factors solutions to determine the genetic, shared, and nonshared environmental correlations. Unique shared environmental influences on internalizing problems were not estimated for girls in the reduced model, so for the reduced models the shared environmental correlation is 1, while the genetic and nonshared environmental influences were 0 (common genetic and nonshared environmental influences also were not estimated). Thus, the following genetic and environmental correlations were computed using the full models.

For girls in the NEAD sample, the genetic correlation was essentially nonexistent, $r_g=.01$. There was, however, moderate to substantial overlap in both shared and nonshared environmental influences for maturational status and internalizing problems, $r_c=.39$; $r_e=.21$. For girls in the TCHAD sample, the genetic influences on maturational status and internalizing problems were entirely separate, $r_g=0$. There was substantial overlap in shared environmental influences, $r_c=.65$, and no overlapping nonshared environmental influences for maturational status and internalizing problems, $r_e=0$.

Discussion

This study was one of the first to examine the genetic and environmental influences underlying the well established association between maturational status and internalizing problems. Maturational status predicted concurrent internalizing problems even controlling for previous levels of internalizing problems for girls. Maturational status and internalizing problems were not associated consistently for boys, as has been previously reported in the literature. Using two samples, results consistently demonstrate the importance of environmental influences for the association between maturational status and concurrent internalizing problems for girls. The shared environment explained the association between more advanced maturational status and more internalizing problems (like studies of puberty and externalizing behavior; Burt, McGue, DeMarte, Krueger, & Iacono, 2006; Dick, Rose, Viken, & Kaprio, 2000; Eriksson, Kaprio, Pulkkinen, & Rose, 2005). However, this association appears to be limited to concurrent association; maturational status in mid-adolescence did not predict later internalizing problems for girls or boys.

Genetic and Environmental Influences on the Association between Puberty and Internalizing

The phenotypic correlations between perceived maturational status and internalizing problems for girls in both samples were significant and in the same direction as previously found in the literature (see Mendle et al., 2007 for review). The present study contributes to the literature by showing that the extent to which perceived maturational status and

internalizing problems are associated is explained entirely by shared environmental factors, or nongenetic factors that contribute to similarity between family members. The next step in this line of research is to test for specific shared environmental factors that influence both maturational status and internalizing problems. Because of the definition of shared environmental influences, these factors are likely to be family-level environmental experiences, such as family ideals, harsh parenting, or nutrition, which both siblings in the same family would likely experience similarly.

It is important to note that though it is common practice to drop non-significant paths from biometric models in pursuit of the most parsimonious model, doing so may sometimes oversimplify results, as demonstrated by the shared and nonshared environmental correlations for girls in the NEAD sample. In the full model, the magnitude of common shared and nonshared environmental influences were similar for NEAD, but not TCHAD. The correlations between the shared and nonshared environmental influences on maturational status and internalizing problems were modest whereas the correlation between the shared environmental influences on maturational status and internalizing problems was 1 in the reduced model. It is likely, then, that both shared and nonshared environmental influences contribute to the association between maturational status and internalizing problems for girls in the NEAD sample. This difference in findings could be explained by cultural differences in views on pubertal maturation (e.g., talking about puberty is much more open in Sweden than in the U.S.), or differences in exposure to media and body ideals. However, cultural factors are more likely to present as shared environmental influences (environmental influences that both members of a twin pair experience that make them more similar) within each sample. Thus, the shared environmental mechanism by which constructs like family ideals or cultural environment influence pubertal maturation and internalizing problems appear to apply to multiple cultures, even if the specific cultural influences may differ.

Instead, the difference in findings across samples may be attributable to the constellation of the sample, since NEAD includes nontwin siblings of differing ages. Nonshared environmental influences, or nongenetic factors whose effect makes family members less similar, contribute to the association between pubertal maturation and internalizing problems. Environmental factors that one twin or sibling experiences could contribute to more internalizing problems in that twin/sibling. Nontwin siblings differ in age, in this sample between 1 and 4 years. Though we controlled for age difference in non-twin siblings, we could not control for any differences the difference in age could have caused. For example, a younger sibling may have more experience with understanding maturational status because they watched an older sibling progress through puberty, or feel less isolated or different because of pubertal maturation than children who did not watch a sibling go through the same process. These differences between siblings (who did vs. did not witness pubertal development of an older sibling) could present as nonshared environmental influences on maturational status, internalizing, or the association in results. Or, siblings differing in age may have different cognitive abilities and view their pubertal changes from different perspectives. Siblings differing in age also may be in different grades at school and may be more likely to have different teachers and friends. Indeed, increased popularity and being the target of rumors among peers was related to being more physically developed and was shown to predict increases in internalizing problem in girls (Reynolds & Juvonen,

2011). In this case, peers may act as nonshared environmental influences. Finally, siblings differing in age likely have different responsibilities and relationships at home. Thus, siblings may differ in their perceptions of the same environmental event or stimuli, presenting as nonshared environmental influences in the present study. Therefore, candidates for specific nonshared environmental factors include cognitive ability as it relates to perception of their own development, peer influence, differential treatment by parents, and prior experience with the concept of pubertal development for younger but not older siblings. Future research is needed to determine which specific factors explain this nonshared environmental effect of maturational status, and whether these factors are amenable to intervention.

Explanations for the Associations between Pubertal Maturation and Internalizing Problems

Results from the current study are consistent with existing hypotheses for the association between pubertal timing and maturation status and internalizing problems. Findings suggest that common genetic influences are not directly responsible for the association between maturational status and internalizing problems. In other words, the genes responsible for advancing puberty are not the same genes conferring risk for internalizing problems in adolescence. Instead, shared and nonshared environmental influences contribute to the association between maturational status and internalizing problems. These findings suggest that social and emotional changes during adolescence may mediate the influence of pubertal hormones on internalizing problems (e.g., Susman et al., 1991). The effect of pubertal hormones would then appear as environmental, not genetic influences on the association between maturational status and internalizing problems. This conclusion has potential implications for how we understand the effects of pubertal hormones during adolescence. Our findings suggest that the mechanism underlying the association is entirely environmental. That is, the psychological effects of hormones which drive pubertal development likely operate through social, environmental pathways. Therefore, breaking the environmental mechanism of the association between advancing development and internalizing problems would be an appropriate intervention technique, once the specific environmental mechanisms are identified and if evidence from intervention trials suggests the environmental mechanisms are malleable. Necessarily, the changing environments hypothesis is also supported. Here, we draw from both explanations, rather than attempting to support one over the other. Both hypotheses continue to present viable mechanisms by which pubertal maturation and internalizing problems are associated.

Results suggesting the importance of environmental influences on the association between maturational status and internalizing problems may be interpreted in three ways. First, a third environmental variable may influence both maturational status and internalizing problems in adolescence. Nutrition affects pubertal timing, tempo, and maturational status across development such that poor diet and childhood obesity may accelerate pubertal maturation (Davison, Susman, & Birch, 2003; Ellis, 2004). Obese children are also at risk for developing internalizing problems (Datar & Sturm, 2006; Erermis, Cetin, Tamar, Bukusoglu, Akdeniz, & Goksen, 2004). However, obesity, and the influence of obesity on internalizing problems, is not a female phenomenon. Thus, while a viable mechanism, we

would expect also to find an association between measures of puberty and internalizing symptoms in boys if the association was wholly explained by obesity.

Second, a third environmental variable may mediate the association between pubertal maturation and internalizing problems, as in the case of family ideals. One hypothesis is that the way the adolescent perceives their own maturational status could be explained by how the family views the body and the pubertal transition. For example, if a family believes that women should be thin, and the adolescent girl begins to perceive changes in her body that are not consistent with that thin ideal, she may show internalizing problems. Adversity in the early rearing environment, including insecure attachment, negative or harsh parenting, and father absence, also has been shown to impact the timing of puberty in girls (e.g., Belsky, Steinberg, Houts, Friedman, DeHart, Cauffman, et al. 2007; Belsky, Houts, & Fearon 2010) and the development of internalizing problems (e.g., Zahn-Waxler et al. 2008). In adolescence, pubertal status is associated with less cohesion and calm discussions with mothers and fathers for boys and girls, and increases in parent-adolescent conflict in girls (e.g., Steinberg, 1987). Further, maternal and paternal negativity are associated with internalizing problems in part because of shared and nonshared environmental influences (Reiss et al., 2000). Therefore, family environmental influences including parental negativity may contribute to both pubertal development and internalizing problems, and completely or partially account for the environmental association between pubertal maturation and internalizing problems though this hypothesis has not been tested.

Finally, perceived pubertal development may act as an environmental factor influencing individuals to develop internalizing problems. Related findings suggesting that environmental influences on depressive symptoms are greater among youth in more advanced stages of pubertal maturation than among less mature youth generally support this hypothesis (Edwards et al., 2011). Adolescents may develop internalizing symptoms in reaction to their own feelings of being different or isolated from peers as they witness their own development. It is also possible that the direction of effects is in the opposite direction, that adolescents with internalizing problems also show a reporting bias in measures of maturational status. However, this is unlikely because the association between maturational status and internalizing problems also is found when using hormonal and clinician reported measures of maturational status in the literature. Future research using longitudinal samples would help to determine whether perceived maturational status or pubertal timing and tempo directly influences the development of internalizing problems, or whether family body ideals, childhood obesity, and/or early rearing environment are mechanisms that mediate the shared environmental influences explaining the association between pubertal maturation and internalizing problems. Identifying and exploring the influence of these potential mechanisms, and identifying whether the mechanisms are malleable may help future researcher to develop interventions to reduce the effect of pubertal maturation on the increase in internalizing symptoms observed during adolescence.

Limitations and Future Directions

There were several limitations in the present study that are important to consider when interpreting the results. First, we used the PDS as our measure of maturational status. The

PDS combines indices of adrenal and gonadal axis maturation, which mature at different times (Dorn et al., 2006) and may cloud the hormonal mechanisms driving correlates of pubertal change (thus limiting our ability to find support for the gene expression mechanism of hormonal influences). Also, adolescents are not the best reporters of their own maturational status, often reporting themselves as closer to average development than a clinician would report (Shirtcliff et al., 2009). Accordingly, results are interpreted considering that the PDS is a youth report, and therefore tests the influence of self-perceived maturational status. Recoding the PDS to give less weight to those items that youth are not as reliable reporting, as done in this study, should help to ameliorate some of the objections to the measure. Though recoding the PDS has advantages, the coding system was based on correlations with other measures of puberty within a different, small sample. Thus, there may be sample-specific error in the coding scheme.

Second, as with many studies examining the association between maturational status and internalizing problems, in the current study maturational status is confounded with age. This is especially true of the NEAD sample, where there is quite a wide age range. While the wide age range did guarantee participants in each stage of pubertal development, this age range is particularly problematic because it inflates the correlation between maturational status and age. Because of the relatively small sample size and age range configuration of nontwin siblings in NEAD, we were unable to test the questions posed in this report using a subsample with a restricted age range. However, we believe that findings reflect primarily maturational status and not age because of the consistency in findings in both samples, and because the associations were consistent with past research on this association. The phenotypic correlations between maturational status and internalizing problems were relatively low, though the magnitude of the associations does not differ substantially from many published reports (e.g., Ge et al., 2006; Mendle et al., 2007; Negri, Fung, & Trickett, 2008). For example, for girls in the TCHAD sample, the correlation was only $r = .09$, which translates to less than 1% of the variance in internalizing problems explained by maturational status. For girls in the NEAD sample, the correlation was slightly higher, $r = .14$, translating to still only about 2% of the variance in internalizing problems being explained by maturational status. These correlations are small likely because both the NEAD and TCHAD samples are normative and participants show few internalizing symptoms (only about 7% of each sample would qualify as having clinical levels of internalizing problems, measured by the CDI norms in NEAD and the CBCL T-score norms in TCHAD). Though the magnitudes of these correlations are modest, the findings clarify why the correlation arises. We are confident that we detected meaningful effects because the associations did reach significance, were consistent with past research, and findings were replicated across both samples. However, it is important to remember that internalizing problems are predicated in a complex nexus of experiences and predispositions, of which pubertal stage is only one component.

Relatedly, the distributions of maturational status were different for boys and girls such that more girls were at or above Tanner Stage 3 than boys (as expected). This could have implications for differences in findings between boys and girls. Specifically, if hormonal changes occurring around Stage 3 do have a causal effect of increasing internalizing symptoms, we would expect to see more internalizing symptoms for girls in these samples,

and stronger associations between pubertal maturation and internalizing symptoms for girls than for boys in these samples in part because there are more girls above Stage 3 than there are boys. However, because findings showing increased internalizing symptoms in girls compared with boys after puberty, and stronger associations between pubertal development and internalizing symptoms in girls are so consistent across the literature, we believe that the small differences in distributions in these samples are not entirely driving our findings.

We used broadly defined measures of internalizing problems. In NEAD, we used a multi-rater, multi-method approach which asked about a number of distinct types of internalizing symptoms (e.g., anxiety and especially depressive symptoms) on several different time-scales. In the TCHAD sample, we also used a multi-rater approach, but only on one measure assessing somatic, anxiety, withdrawal, and depressive symptoms. It is possible that results may differ depending on the specific type of internalizing symptom assessed. We chose to assess broadly defined internalizing constructs in order to capture as many types of problems as possible from these low-risk samples, which is not uncommon in the literature.

Finally, as with many twin studies, there are some limitations to the generalizability of our results. The inclusion of nontwin siblings does allow us to generalize the present findings beyond twins. However, the implications of having a similarly aged, same-sex sibling on pubertal maturation and its association with internalizing problems is unknown. Further, the lack of ethnic diversity in these samples limits generalizations to White individuals.

Conclusion

The current study is an important initial step in examining the underlying processes that influence the association between pubertal maturation and internalizing problems. Results show that maturational status and internalizing problems are associated for entirely environmental reasons. The implications of these findings should guide subsequent research looking for specific environmental factors influencing the association between maturational status and internalizing problems. Implications of specific environmental factors influencing this association could be particularly important for intervention. Because the association is environmentally influenced, there is an opportunity in earlier in childhood to change specific shared or nonshared environmental influences found to mediate the association between pubertal maturation and internalizing problems, once specific mechanisms are identified and shown to be malleable in intervention trials.

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MZ = 1.00; DZ & Full Siblings = 0.50; Half Siblings = 0.25; Genetically Unrelated Siblings = 0.0

Figure 1.

Bivariate Cholesky Decomposition. The latent factors A1, C1 and E1 represent genetic, shared environmental and nonshared environmental contributions to covariation between pubertal timing and externalizing behavior and A2, C2, and E2 represent genetic, shared and nonshared environmental influences on externalizing behavior that are uncorrelated with pubertal timing.

Table 1
Sample Characteristics for NEAD and TCHAD

Characteristic	NEAD		TCHAD	
	Male	Female	Male	Female
Number of sibling pairs by sibling type				
MZ	46	42	207	204
DZ	50	47	145	150
FI	47	44		
FS	86	89		
HS	60	47		
US	74	55		
	Sibling 1	Sibling 2	Sibling 1	Sibling 2
Age	14.6 (2.2)	13.0 (2.3)	13.7 (.5)	13.7 (.5)
Age Range	10-18	9-18	12.9-14.5	12.9-14.5
Age Difference	1.6 (1.3)		0	
	Mother	Father	Mother	Father
Parent Education				
Lower*	41.2%	34.6%	58.1%	57%
Higher*	58.8%	65.4%	41.9%	43%
Family SES				
Unskilled Labor	12%		10%	
Skilled Labor	28%		28%	
Mid-level business	22%		45%	
High-level business	30%		16%	

Note: Lower education means completed high school or less for NEAD, and completed two years of higher secondary education or less for TCHAD. Higher education means more than high school education in NEAD and completed 3 or more years of higher secondary education or a university degree in TCHAD.

Table 2
Age and internalizing problems for each sample by wave of assessment

Sample	NEAD		TCHAD	
	Boys	Girls	Boys	Girls
Wave 1				
Age Range	10-18*		8-9	
Internalizing	8.8 (5.6)	10.8 (4.9)	3.1 (3.7)	3.7 (5.5)
Wave 2				
Age Range	-----		13-14*	
Internalizing			4.7 (3.9)	6.9 (5.3)
Wave 3				
Age Range	21-35		16-17	
Internalizing	13.7 (10.1)	13.8 (10.1)	4.2 (3.6)	7.3 (5.6)
Wave 4				
Age Range	---		18-19	
Internalizing			4.5 (4.3)	7.4 (4.3)

* Note: Wave 1 of NEAD is comparable to wave 2 of TCHAD based on mean age. No other waves are comparable.

Table 3
Distributions of pubertal status for boys and girls in each sample

"Tanner" stage		1	1.5	2	2.5	3	3.5	4	4.5	5
TCHAD	Girls	6 (<1%)	18 (2.3%)	66 (8.5%)	65 (8.3%)	113 (14.5%)	108 (13.8%)	149 (19.1%)	189 (24.2%)	66 (8.5%)
	Boys	75 (10.4%)	145 (20.2%)	83 (11.5%)	93 (12.9%)	97 (13.5%)	105 (14.6%)	99 (13.8%)	17 (2.4%)	5 (<1%)
NEAD	Girls	31 (4.7%)	30 (4.7%)	41 (6.4%)	40 (6.3%)	47 (7.3%)	47 (7.3%)	98 (15.3%)	137 (21.4%)	169 (26.4%)
	Boys	22 (3.1%)	152 (21.1%)	90 (12.5%)	105 (14.6%)	85 (11.8%)	76 (10.6%)	64 (8.9%)	79 (11.0%)	46 (6.4%)

Note: "Tanner" stage scores were computed by weighting items according to the reliability of children's perceptions on each item, and in order to map best onto hormone and physical exam scores according to methodology described in Shirliff et al., (2009).

Table 4
Correlations between pubertal status and internalizing problems

		Internalizing Problems			
Sample	Concurrent Association	Accounting for previous Internalizing Problems	Time 3 Internalizing Problems	Time 4 Internalizing Problems	
Girls	NEAD	.14*	N/A	-.03	N/A
	TCHAD	.09*	.13*	-.04	-.04
Boys	NEAD	-.09*	N/A	-.04	N/A
	TCHAD	.02	.01	.05	.01

* *Note:* = significant correlation ($p < .05$).

Table 5

Bivariate Cholesky Decomposition Model Fit Statistics

NEAD Girls						
Model	-2lnL	Df	AIC	Change in χ^2	Change in df	p-value
Full	6155.0	1350	3461.0	NA	NA	NA
Best-fitting	6160.2	1353	3460.24	5.20	3	.16
TCHAD Girls						
Model	-2lnL	df	AIC	Change in χ^2	Change in df	p-value
Full	4075.7	1569	937.7	NA	NA	NA
Best-fitting	4075.9	1572	931.9	.26	3	.97

Note: The best-fitting model included genetic, shared environmental, and nonshared environmental influences on maturational status, only shared environmental influences common to maturational status and internalizing symptoms, and unique genetic and nonshared environmental influences on internalizing problems in both samples.

Table 6
Unstandardized parameter estimates and 95% Confidence Intervals for the bivariate Cholesky decomposition for girls in the NEAD sample

Sample	NEAD				TCHAD			
	Full model		Best-fitting model		Full model		Best-fitting model	
Parameter	Pubertal Maturation	Internalizing Problems	Pubertal Maturation	Internalizing Problems	Pubertal Maturation	Internalizing Problems	Pubertal Maturation	Internalizing Problems
Common Genetic Influences	.73 [.63, .83]	.003 [-.23, .21]	.73 [.64, .83]	---	.71 [.53, .85]	0 [0, .17]	.72 [.54, .85]	---
Unique Genetic Influences		.54 [-.79, .79]		.67 [.48, .79]		.73 [.52, .83]		.77 [.59, .83]
Common Shared Environmental Influences	.60 [.47, .69]	.14 [-.06, .32]	.60 [.47, .69]	.19 [.06, .32]	.48 [.13, .66]	.22 [-.05, .52]	.46 [.15, .65]	.23 [.06, .53]
Unique Shared Environmental Influences		.33 [-.54, .54]		---		.26 [-.55, .55]		---
Common Nonshared Environmental Influences	.32 [.26, .39]	.16 [-.08, .40]	.32 [.26, .39]	---	.52 [.47, .58]	0 [0, .04]	.52 [.47, .58]	---
Unique Nonshared Environmental Influences		.75 [.59, .90]		.72 [.59, .85]		.60 [.54, .66]		.59 [.54, .65]

Note: Common genetic and environmental influences represent genetic and environmental influences on pubertal maturation, and genetic and environmental influences shared between pubertal maturation and internalizing problems. Unique genetic and environmental influences represent the remaining genetic and environmental influences on internalizing problems.