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Developmental changes in genetic and environmental influences on rule-breaking and aggression: age and pubertal development

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Background: Antisocial behavior (ASB) can be meaningfully divided into nonaggressive rule-breaking versus aggressive dimensions, which differ in developmental course and etiology. Previous research has found that genetic influences on rule-breaking, but not aggression, increase from late childhood to mid-adolescence. This study tested the extent to which the developmental increase in genetic influence on rule-breaking was associated with pubertal development compared to chronological age. **Method:** Child and adolescent twins (n = 1,031), ranging in age from 8 to 20 years (M age = 13.5 years), were recruited from public schools as part of the Texas Twin Project. Participants reported on their pubertal development using the Pubertal Development Scale and on their involvement in ASB on items from the Child Behavior Checklist. Measurement invariance of ASB subtypes across age groups (≤12 years vs. >12 years old) was tested using confirmatory factor analyses. Quantitative genetic modeling was used to test whether the genetic and environmental influences on aggression and rule-breaking were moderated by age, pubertal status, or both. **Results:** Quantitative genetic modeling indicated that genetic influences specific to rule-breaking increased as a function of pubertal development controlling for age (a gene × puberty interaction), but did not vary as a function of age controlling for pubertal status. There were no developmental differences in the genetic etiology of aggression. Family-level environmental influences common to aggression and rule-breaking decreased with age, further contributing to the differentiation between these subtypes of ASB from childhood to adolescence. **Conclusions:** Future research should discriminate between alternative possible mechanisms underlying gene × puberty interactions on rule-breaking forms of antisocial behavior, including possible effects of pubertal hormones on gene expression. **Keywords:** Antisocial behavior, aggression, rule-breaking, puberty, adolescence, behavior genetics.

Introduction

Antisocial behavior (ASB) encompasses actions that violate laws, social norms, and/or the rights of other people. One well-validated way of parsing the heterogeneity of ASB is by type of behavior (Burt, 2012; Tackett, Krueger, Iacono, & McGue, 2005) – aggression (acts that directly victimize another person) versus nonaggressive rule-breaking (acts, such as theft, that violate laws or norms but do not directly victimize another person). Compared to aggression, rule-breaking behavior increases more dramatically, on average, during adolescence (see Burt, 2012 for comprehensive review). In addition, *genetic* influences on rule-breaking, but not aggression, also increase from childhood to adolescence (Burt & Klump, 2009; Burt & Neiderhiser, 2009), a finding that has been replicated across four independent twin and adoption studies (Burt, 2014).

Currently, it is unclear why genetic influences on rule-breaking increase with development. One hypothesis, originally advanced by Burt and Neiderhiser (2009), is that these increases are due to *gene-environment transactions*, a dynamic process in which people select and are selected into environmental experiences on the basis of their genetically influenced characteristics. These environments reciprocally influence their behavior, thus reinforcing and magnifying initial genetically based differences between people (Tucker-Drob & Harden, 2012). For example, adolescents with genetic propensities toward sensation seeking might select more deviant peer groups, and these peer groups might, in turn, facilitate rule-breaking (Harden, Quinn, & Tucker-Drob, 2012; Mann, Kretsch, Tackett, Harden, & Tucker-Drob, 2015). As children are given more autonomy to select their own environments over development, transactional processes will compound, amplifying earlier genetic influences on rule-breaking. Such a transactional process is an 'outside of the skin' mechanism for increasing genetic influence, as it does not necessarily involve any biological changes in gene expression, but rather depends on feedback from the environment.

Alternatively, increases in genetic influence may be due to 'inside the skin' mechanisms, such as developmental changes in gene expression. Genetic variance in rule-breaking has been found to increase most markedly between ages 10 and 15, a developmental span that overlaps substantially with pubertal change. During puberty, average levels of testosterone, progesterone, estradiol, DHEA, and

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DHEA-S increase in both sexes (Biro, Lucky, Huster, & Morrison, 1995; Granger, Schwartz, Booth, & Arentz, 1999). These adrenal and gonadal hormones can directly affect gene expression via binding to DNA-transcription factors that are distributed throughout the central nervous system (Nilsson & Gustafsson, 2000; Witt, 2007). It is therefore possible that the neuroendocrine changes of puberty lead to changes in gene expression.

No previous twin study has directly examined puberty as a potential moderator of the genetic etiology of ASB subtypes, but a few previous studies provide evidence that supports the plausibility of this hypothesis. First, among adolescent girls, genes related to earlier pubertal timing contribute to genetic variance in rule-breaking and aggressive forms of ASB (Harden & Mendle, 2012) – indicating that there is a source of genetic variance in ASB not relevant before the onset of puberty. Second, among adult men, testosterone levels were found to interact with *MAOA* genotype in predicting antisocial personality disorder symptoms, with the largest differences between genotypes evident among men with higher testosterone levels (Sjöberg et al., 2008). This result is consistent with the hypothesis that gonadal hormones may exacerbate genotypic differences, although the study has not yet been independently replicated and must therefore be considered preliminary. Finally, providing more general evidence that puberty may be critical for developmental transitions in genetic effects, previous twin research on gene × puberty interactions has found evidence that genetic influences on eating disorder behaviors are higher among girls with more advanced pubertal status and higher estradiol levels (Klump, Keel, Sisk, & Burt, 2010; Klump et al., 2012).

The transition from childhood to adolescence thus involves a complex array of both social and biological changes that may contribute to increasing genetic influence on ASB, via both 'outside the skin' and 'inside the skin' mechanisms. As an initial step toward understanding these mechanisms, this study uses a sample of child and adolescent twin pairs to examine both puberty and age as moderators of genetic influences on aggression and rule-breaking behaviors.

Methods

Participants

Twins were identified and recruited from public school rosters as part of the Texas Twin Project (Harden, Tucker-Drob, & Tackett, 2013). The analytic sample consisted of $n = 1{,}031$ twins nested within 547 pairs from 504 unique families. (One family had two sets of twins. Additionally, there were 21 sets of triplets, each of which contributed three pairwise combinations per family.) Participants ranged in age from 7.9 to 20.1 years ($M = 13.5$, $SD = 2.9$). Most participants (72%) were between the ages of 10 and 16, 11% were <10 years old, and only 3% were >18 years old. Sixty-eight percent of twins were non-Hispanic White, 20% were Hispanic/Latino, 10% were African American, and the remaining 2% were another race/ethnicity. Using maternal education as a metric of socioeconomic status, 12% of mothers had a high school diploma or less than a high school education, 29% had some college or vocational school, 29% had a college degree, 31% had some education beyond a college degree.

Procedure

Details regarding identification of potential twin families are given in Harden et al. (2013). Participants who were currently in grades 3 through 12, or had finished high school within the last 3 months, were recruited to participate in an in-laboratory study. All recruitment, consent, and study procedures received ethics approval from the university Institutional Review Board. Each twin was assessed in a separate room by a different research assistant. Potentially sensitive survey questions were computer-administered.

Measures

Zygosity. All opposite-sex pairs were classified as dizygotic (DZ). Zygosity for same-sex twin pairs was determined based on responses to survey items regarding twins' physical similarity (e.g. facial appearance) and the frequency with which the twins are/would be mistaken for one another. These items were completed by a parent and at least two research assistants; twins ages 14 and older also provided self-reports. Zygosity items were entered into a latent class analysis (LCA). LCA of questionnaire zygosity data has been shown to have a classification error rate of >1%, as compared to classification using genotyping (Heath et al., 2003). Final zygosity classifications were as follows: 178 monozygotic (MZ) pairs (96 female; 82 male) and 369 DZ pairs (93 female, 102 male, and 174 opposite-sex).

Antisocial behaviors. Antisocial behaviors were assessed using youth self-report on an abbreviated set of 25 items from the Child Behavior Checklist (Lizotte, Chard-Wierschem, Loeber, & Stern, 1992). Nonaggressive rule-breaking behaviors (e.g. 'I break rules at home, school or elsewhere') and aggressive behaviors (e.g. 'I physically attack people') were rated on a 3-point scale ranging from 0 = *Not True* to 2 = *Very True or Often True*. Construction of scale scores was informed by measurement invariance analyses, and scale scores were residualized for potential confounds (see 'Behavior genetic models' in the Results section).

Pubertal development. Pubertal development was assessed using the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988). Both males and females responded to items about growth in height, growth of body hair, and skin changes (such as pimples). Males also rated growth of facial hair and deepening of voice, whereas females rated breast growth and whether they had begun to menstruate. Items were rated on a 4-point scale ranging from 1 = *Not Yet Begun to Change* to 4 = *Finished Changing*, and the menstruation item was recoded to be consistent with the 4-point scale (*No* = 1, *Yes* = 4). Fifty-six percent of the females reported having begun menstruation. An average of the reports on the five items was calculated, resulting in a pubertal development score ranging from 1 to 4. Boys' average pubertal development score ($M = 2.42$, $SD = 0.83$) was slightly lower than girls' ($M = 2.76$, $SD = 0.94$). Pubertal development was highly correlated with age ($r = .76$). Figure S1 in the supporting information (available online) illustrates the distribution of pubertal development at each age, separately by sex. Twin pair

correlations for pubertal development were substantial in both MZ (r = .78) and DZ (r = .67) pairs.

Results

All analyses were conducted using M*plus* version 7.1 (Muthén & Muthén, 1998–2012). The complex sampling option was used to correct results for nonindependence of twins within pairs (in phenotypic analyses) and nonindependence of multiple pairs within the same family (in behavioral genetic analyses).

Exploratory and confirmatory factor analyses

We first conducted an exploratory factor analysis (EFA) to evaluate which items loaded onto the rule-breaking versus aggressive dimensions in our sample.[1] The fit of a one-factor EFA solution was marginal (χ^2 = 933.42, df = 275, p < .001, RMSEA = .049, CFI = .857, TLI = .844), whereas a two-factor solution fit the data well (χ^2 = 630.69, df = 251, p < .001, RMSEA = .039, CFI = .917, TLI = .901).[2] Based on the two-factor EFA solution, we specified a CFA model of rule-breaking and aggression, in which the latent rule-breaking and aggression factors were correlated and were regressed on sex, age, age-squared, African American race, Hispanic/Latino ethnicity, and pubertal status. This model fit the data well (χ^2 = 897.48, df = 412, p < .001, RMSEA = .035, CFI = .908, TLI = .900). Factor loadings, factor correlations, and regression coefficients for this model are summarized in Table 1. Males reported higher rule-breaking and aggressive behavior. Rule-breaking increased with age. In contrast, aggression decreased with age, although this effect was not significantly different than zero. Compared to Whites, Latino and African American youth reported higher rule-breaking, and African American youth reported higher aggression. There were no significant main effects of pubertal status.

Behavior genetic models

Prior to fitting behavioral genetic models, we conducted a series of measurement invariance models that examined whether the measures of rule-breaking and aggression were invariant across age, because lack of measurement invariance could bias estimates of genetic and environmental influence (Tucker-Drob, Harden, & Turkheimer, 2009). Measurement invariance analyses and results are described in the Appendix S1. Loadings were invariant, indicating that the CBCL items were equivalently sound indicators of the underlying latent factors in children versus adolescents. In contrast, thresholds were not invariant, indicating that age differences in how frequently certain antisocial behaviors were endorsed were not all mediated through the effects of age on the latent factor. We therefore constructed scale scores for behavioral genetic models as follows: First, the effects of sex,[3] African-American race, Hispanic/Latino ethnicity, age, and age-squared were regressed out of each item using a Poisson regression model.[4] (Race/ethnicity and sex were partialled to prevent any results from being potential artifacts of group differences in pubertal development and/or ASB). The item-specific residuals were then summed for rule-breaking and aggression items separately; the sums were log-transformed to correct positive skew; and the resulting values were scaled to z-scores. Twin pair correlations for rule-breaking

Table 1 Standardized parameter estimates from CFA of rule-breaking and aggression

	Aggression factor	Rule-breaking factor
CBCL items	Factor loadings	
I physically attack people	.76***	
I threaten to hurt people	.75***	
I destroy things belonging to others	.66***	
I get in many fights	.66***	
I scream a lot	.66***	
I am mean to others	.65***	
I destroy my own things	.62***	
I tease others a lot	.62***	
I have a hot temper	.59***	
I argue a lot	.54***	
I brag	.46***	
I am stubborn	.44***	
I talk too much	.37***	
I disobey at school		.81***
I break rules at home, school, or elsewhere		.79***
I steal from places other than home		.74***
I disobey my parents		.72***
I steal at home		.68***
I swear or use dirty language		.64***
I cut classes or skip school		.64***
I lie or cheat		.64***
I hang around kids who get in trouble		.61***
I run away from home		.59***
I set fires		.57***
I don't feel guilty after doing something I shouldn't		.34***
	Factor correlation	
	.70***	
Covariates	Regression coefficients	
Male	.19*	.44***
African American	.53***	.43***
Latino	.03	.26*
Age	−.03	.05*
Age^2	−.001	.004
Pubertal development	.11	.09

Regression coefficients for all covariates except for pubertal development are standardized with respect to the latent rule-breaking and aggression factors only.
*p < 0.05; ***p < .001.

were as follows: r_{MZ} = .54, r_{DZ} = .26. Twin pair correlations for aggression were as follows: r_{MZ} = .38, r_{DZ} = .20. The rule-breaking and aggression scores were not correlated with age, age-squared, pubertal development, or pubertal development-squared; therefore, the possibility of spurious interaction findings due to nonlinear effects of the moderators (Rathouz, Van Hulle, Rodgers, Waldman, & Lahey, 2008) is not a particular concern.

Using these scale scores, we fit a series of quantitative genetic models that decomposed variance shared between and unique to aggression and rule-breaking into additive genetic (*A*), shared environmental (*C*), and nonshared environmental (*E*) factors, as illustrated in Figure 1 (Neale & Maes, 2004). The model was parameterized as a bivariate common and specific factors model (Loehlin, 1996), in which one set of ACE factors contributed to both rule-breaking and aggression, and then two additional sets of ACE factors captured variance unique to each ASB subtype.

As a preliminary analysis, we tested whether the magnitude of the paths from the ACE components to the ASB phenotypes differed between males and females. A model that allowed these paths to differ between males and females fit no better than a model in which the ACE paths were constrained to be equal between sexes (scaled χ^2 difference test = 5.62, $\Delta df = 9$, $p = .78$). This is consistent with previous research suggesting that the average level of involvement in ASB, but not its etiology, differs between males and females (Rhee & Waldman, 2002). Results from the no-sex-moderation model are summarized in Table 2.

We next fit three sets of moderation models, in which the paths from the ACE components to the phenotypes were allowed to vary as a function of age, pubertal status, or both age and pubertal status (illustrated in Figure 1). The squares of the paths from the ACE components to the phenotypes quantify the variance in the phenotype that is due to genetic and environmental differences, as a function of the moderator values (Purcell, 2002).

Moderation by age only. Results from an age moderation model are summarized in Table 2. Initial estimates indicated that the main effects and age interactions for the shared environmental components unique to rule-breaking and aggression were negligible; these parameters were subsequently fixed to zero to facilitate subsequent model convergence. Genetic variance specific to rule-breaking increased with age, whereas shared and nonshared environmental variance common to both aggression and rule-breaking decreased with age. The total model-implied heritability of rule-breaking thus increased from 17% at age 8, to 40% at age 12, to 58% at age 16 (illustrated in the top panel of Figure 2). Neither genetic nor environmental influences specific to aggression showed any age-related changes. Overall, results from the age moderation model replicate previous research showing developmental increases in genetic influences on rule-breaking. The finding of decreased shared environmental overlap between rule-breaking and aggression is also consistent with previous meta-analytic results (Burt, 2013).

We then conducted a follow-up analysis to probe whether the moderating effects of age were consistent across the entire age range of the sample. Specifically, we fit a spline model that estimated two linear effects of age on the ACE paths: the effect of age in childhood (ages 8–12.9 years) and the effect of age in adolescence (ages 13–20 years). Notably, this model did not fit significantly better than a model in which the effects of age were constrained to be constant across childhood and adolescence (scaled χ^2 difference test = 11.29, $\Delta df = 7$, $p = .13$).

Model 2: Moderation by pubertal status only. Results from a puberty moderation model are summarized in Table 2. Initial estimates indicated that

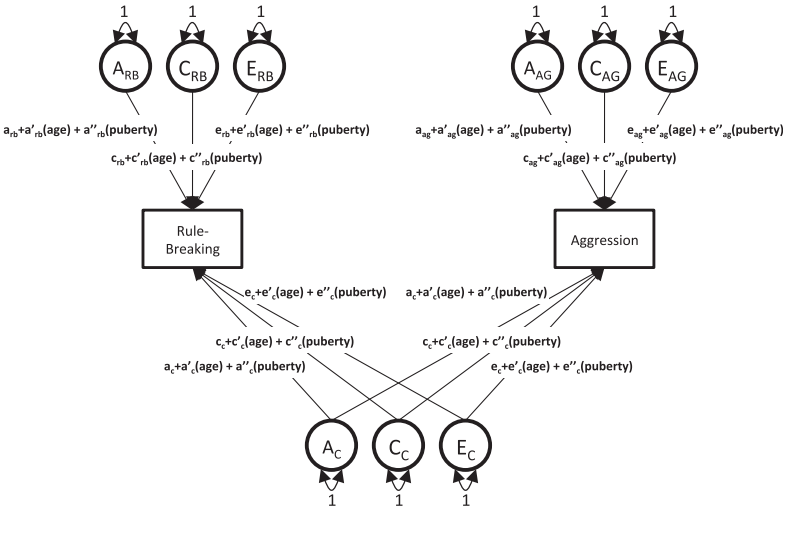

Figure 1 Behavioral genetic model of rule-breaking and aggression. *A* = Additive genetic. *C* = Shared environmental. *E* = Nonshared environmental. Only one twin per pair shown.

Table 2 Parameter estimates from behavior genetic models

	No moderation	Age moderation	Puberty moderation	Age + puberty moderation (Full)	Age + puberty moderation (Trimmed)
Variance common to RB and AGG					
Main effect of A (a_c)	.55*** (0.32, 0.77)	.50*** (0.38, 0.61)	.36 (−0.02, 0.74)	.85*** (0.48, 1.22)	.81*** (0.49, 1.14)
Age interaction (a_c')	–	.02 (−0.04, 0.08)	–	.12** (0.04, 0.20)	.14*** (0.08, 0.20)
Puberty interaction (a_c'')	–	–	.08 (−0.08, 0.24)	−.26* (−0.52, −0.01)	−.32** (−0.54, −0.10)
Main effect of C (c_c)	.14 (−0.51, 0.80)	.07 (−0.19, 0.32)	.52*** (0.22, 0.82)	.05 (−0.39, 0.50)	.36*** (0.16, 0.57)
Age interaction (c_c')	–	−.08*** (−0.13, −0.03)	–	−.09* (−0.17, −0.02)	−.05 (−0.10, 0.01)
Puberty interaction (c_c'')	–	–	−.20** (−0.35, −0.05)	.13 (−0.13, 0.39)	[0]
Main effect of E (e_c)	.43*** (0.32, 0.54)	.43*** (0.34, 0.52)	.51*** (0.33, 0.69)	.38* (0.002, 0.76)	.45*** (0.36, 0.54)
Age interaction (e_c')	–	−.03* (−0.06, 0.00)	–	−.02 (−0.08, 0.04)	[0]
Puberty interaction (e_c'')	–	–	−.06 (−0.14, 0.03)	.02 (−0.18, 0.22)	[0]
Variance Unique to RB					
Main effect of A (a_{rb})	.47*** (0.35, 0.60)	.48*** (0.36, 0.61)	.27* (0.02, 0.52)	.23 (−0.15, 0.60)	.25* (0.03, 0.47)
Age interaction (a_{rb}')	–	0.05* (0.01, 0.10)	–	−0.001 (−0.07, 0.07)	[0]
Puberty interaction (a_{rb}'')	–	–	.13* (0.01, 0.26)	.14 (−0.03, 0.32)	.14* (0.03, 0.25)
Main effect of C (c_{rb})	.00 (0.00, 0.00)	[0]	[0]	[0]	[0]
Main effect of E (e_{rb})	.52*** (0.43, 0.61)	.52*** (0.43, 0.61)	.58*** (0.40, 0.77)	.91*** (0.60, 1.23)	.87*** (0.62, 1.12)
Age interaction (e_{rb}')	–	.02 (−0.1, 0.05)	–	.08** (0.01, 0.15)	.07** (0.02, 0.11)
Puberty interaction (e_{rb}'')	–	–	−.04 (−0.14, 0.06)	−.24** (−0.41, −0.07)	−.22** (−0.35, −0.08)
Variance Unique to AGG					
Main effect of A (a_{ag})	.23* (0.02, 0.45)	.23* (0.02, 0.44)	.28 (−0.07, 0.64)	.20 (−0.49, 0.88)	.25* (0.05, 0.45)
Age interaction (a_{ag}')	–	−.01 (−0.04, 0.03)	–	−.02 (−0.11, 0.08)	[0]
Puberty interaction (a_{ag}'')	–	–	−.04 (−0.23, 0.15)	.02 (−0.37, 0.40)	[0]
Main effect of C (c_{ag})	.001 (−0.002, 0.003)	[0]	[0]	[0]	[0]
Main effect of E (e_{ag})	.66*** (0.58, 0.73)	.65*** (0.57, 0.72)	.72*** (0.57, 0.87)	.65*** (0.39, 0.91)	.66*** (0.59, 0.73)
Age interaction (e_{ag}')	–	−.02 (−0.04, 0.004)	−.04 (−0.11, 0.03)	−.02 (−0.06, 0.02)	[0]
Puberty interaction (e_{ag}'')	–	–		.002 (−0.14, 0.15)	[0]

Age centered at 14 and pubertal status centered at 1. Rule-breaking (RB) and aggression (AGG) scores standardized. 95% confidence intervals in parentheses.
*$p < .05$; **$p < .01$; ***$p < .001$.

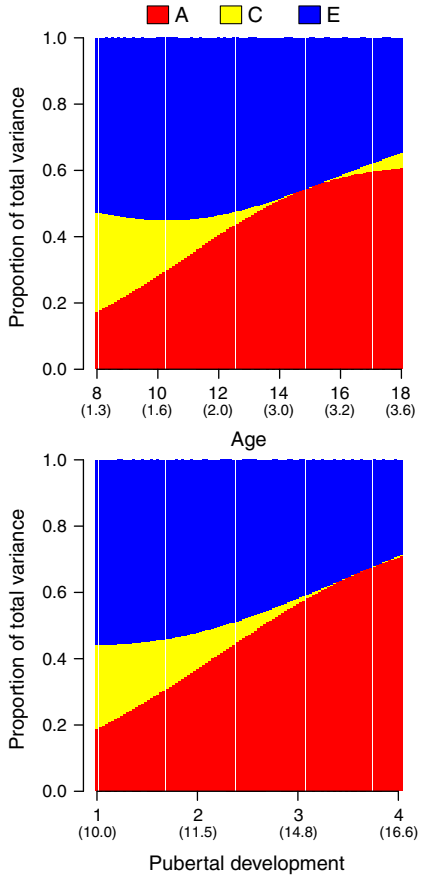

Figure 2 Age- and puberty-related differences in genetic and environmental influences on rule-breaking. Figure based on parameters from 'Age Moderation' (top panel) and 'Puberty Moderation' (bottom panel) models summarized in Table 2. Results do not control for interactions with other moderator. Areas represent the proportion of total variance in rule-breaking (both shared with and unique from aggression) due to additive genetic (*A*), shared environmental (*C*), and nonshared environmental (*E*) influences. The proportion of variance due to *A* is the heritability. Values in parentheses under the *X* axis represent the median level of pubertal development at each age (top panel) and the median age at each level of pubertal development (bottom panel).

the shared environmental components unique to rule-breaking and aggression were zero; these parameters were subsequently fixed to zero to facilitate model convergence. The pattern of results was nearly identical to what was observed for the age-moderation model: genetic variance specific to rule-breaking increased with pubertal development, whereas shared environmental variance common to both aggression and rule-breaking decreased. As illustrated in the bottom panel of Figure 2, the total model-implied heritability of rule-breaking thus increased from 19% in pre-pubertal youth (PDS = 1) to 71% in post-pubertal youth (PDS = 4). There were no puberty-related changes in variance specific to aggression.

To probe whether the moderating effects of pubertal status were constant across early versus late pubertal development, we fit a spline model that estimated two linear effects of pubertal development on the ACE paths: the effect of early pubertal development (PDS score ranging from 1 to 2.5) and the effect of late pubertal development (PDS score ranging from 2.5 to 4.0). This model did not fit significantly better than a model in which the effects of pubertal development were constrained to be constant across both early and late pubertal development (scaled χ^2 difference test = 5.93, $\Delta df = 7$, $p = .55$).

Moderation by age and pubertal status. Results from a model in which both pubertal development and age were simultaneously entered as moderators are summarized in Table 2. Three sets of results are particularly noteworthy. First, the gene × puberty interaction on rule-breaking was essentially unchanged compared to the puberty-moderation model ($a'' = .143$ in age + puberty model vs. .133 in the puberty-only model). In contrast, the gene × age interaction on rule-breaking was reduced to zero after entering puberty into the model ($a' = -.001$). The standard errors of both the puberty and age moderation effects, however, were larger when considered simultaneously than when entered separately, because age and pubertal development were highly correlated. Second, the moderating effect of age on shared environmental influences common to both rule-breaking and aggression was still evident in the full interaction model. Third, there were a number of interaction effects that were not apparent in a puberty-only or age-only moderation model; these might have been masked because puberty and age had opposing trends. Specifically, age was associated with increasing nonshared environmental influences specific to rule-breaking and with increasing genetic influences common to both rule-breaking and aggression, whereas pubertal development was associated with decreases in these same factors.

Finally, we fit a trimmed model that fixed parameters with *p* values greater than .10 in the full model to zero. This model did not fit significantly worse than the full interaction model (scaled χ^2 difference test = 8.32, $\Delta df = 8$, $p = .40$). Results from the trimmed model are summarized in the final column of Table 2 and illustrated in Figure 3. Focusing on the left column of Figure 3, non-shared environmental influences unique to rule-breaking decreased with pubertal development but increased with age; these countervailing developmental trends essentially cancel each other out, such that there is expected to be relatively little difference in the magnitude of within-MZ-twin-pair differences if comparing younger, less developed children with older, more developed adolescents. The major developmental shift in rule-breaking was an increase in genetic variance as a function of pubertal development. Moving to the middle column of Figure 3, the magnitude of genetic influences specific to aggression was relatively minimal across both age and pubertal development; most of the genetic influences on aggression were shared with rule-breaking. When considering the variance common to both rule-

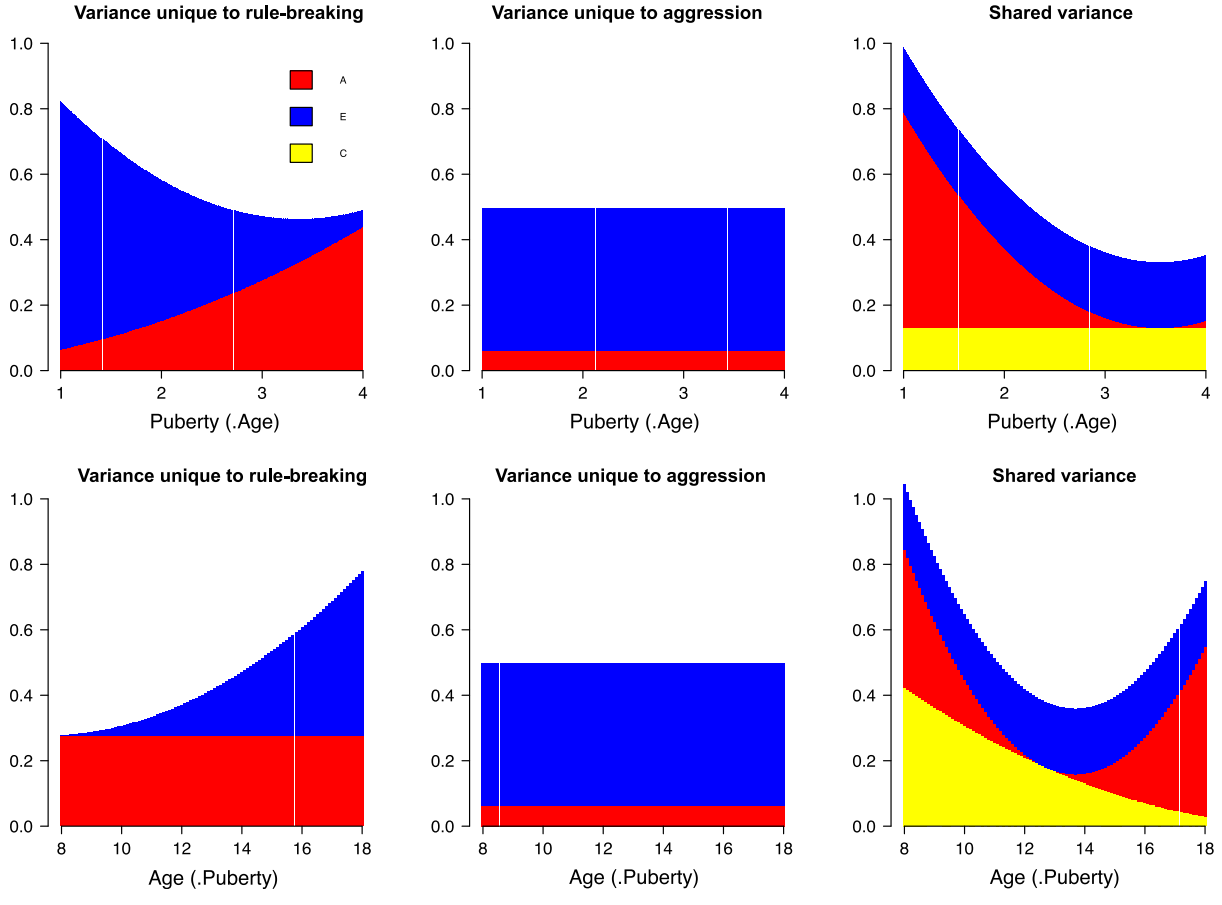

Figure 3 Developmental differences in genetic and environmental influences on rule-breaking and aggression. Figure based on parameter estimates of trimmed age + puberty moderation model (final column of Table 2). Puberty (.Age) = moderating effects of pubertal development controlling for age. Age (.Puberty) = moderating effects of age controlling for puberty. *A* = additive genetic; *C* = shared (family) environmental; *E* = nonshared (unique) environmental. Unstandardized variance components are represented.

breaking and aggression (right column of Figure 3), there were opposing developmental trends in genetic influences – initial developmental decreases are countervailed by a subsequent age-related increase. The major developmental shift in variance shared between rule-breaking and aggression was a decrease in family-level environmental influences as a function of age.

Discussion

This study extended previous research by simultaneously investigating age and pubertal development as moderators of genetic and environmental influences on aggressive and nonaggressive subtypes of ASB. Developmental increases in genetic influences specific to nonaggressive rule-breaking were better accounted for by pubertal status than by age. At the same time, there were age-related decreases in family-level environmental influences common to both rule-breaking and aggression, contributing to the developmental differentiation of these ASB subtypes.

A key issue for future research is to better understand the mechanisms underlying this gene × puberty interaction, as puberty involves a coordinated suite of changes in biology, social relationships, and self-perception. In an 'outside the skin' process, puberty could usher in a constellation of social changes, such as autonomy from parents, that provide the adolescent with new opportunities to select environmental niches consistent with her own genetically influenced motivations and interests. Alternatively, in an 'inside the skin' process, the neuroendocrine events of puberty, particularly elevated concentrations of gonadal hormones, may directly affect gene expression, leading to innovative genetic effects on behavior. Distinguishing between socially mediated versus hormonally mediated processes will require a very large sample of twins combined with careful measurement of both hormones and social environments, such that gene × social environment and gene × hormone interactions can be tested simultaneously as mediators of the moderation effect. Technology for measuring hormonal concentrations cheaply and noninvasively in saliva and hair continues to progress, offering new opportunities to incorporate direct hormonal measurement in large behavior genetic samples. Additionally, if gene × hormone interactions are indeed detected, this opens a new avenue for uncovering the specific genetic underpinnings of ASB, by focusing on genes whose transcription is known to be regulated by gonadal hormones.

Limitations

We use a sample with a wide age range (8–20 years). Such age-heterogeneity is well suited for examining age moderation effects, but age and pubertal development are necessarily strongly confounded in a sample that spans the entire second decade of life. An alternative strategy for the study of pubertal effects is to focus on a very narrow age cohort, typically around ages 11–13; however, this approach conflates *pubertal status* (a person's level of physical development at particular point in time) and *pubertal timing* (individual differences in *when* a person experiences a particular pubertal status). Moreover, average levels of adrenal and gonadal hormones continue to increase for many years after early adolescence (Harden, Kretsch, Tackett et al., 2014; Šulcová, Hill, Hampl, & Starka, 1997). Ultimately, longitudinal research in which all adolescents are followed from prepuberty through young adulthood is necessary to disentangle fully the effects of age, pubertal status, and pubertal timing (Harden, Kretsch, Moore, & Mendle, 2014).

Second, pubertal development was measured using self-reports on the PDS. Previous studies have found that self-reports of pubertal development correlate with testosterone, estradiol, and DHEA as well as – or even better than – Tanner stages as determined by physical exam (reviewed in Harden, Kretsch, Moore et al., 2014; Shirtcliff, Dahl, & Pollak, 2009). In a subsample of the participants used in this study (boys ages 14 +), PDS scores were substantially correlated with salivary testosterone levels ($r = .51$), and this correlation was due to shared underlying genetic influences (Harden, Kretsch, Tackett et al., 2014). Nevertheless, the correspondence between self-reports of pubertal development and hormonal concentrations is far from perfect (Dorn, Dahl, Woodward, & Biro, 2006), and future research will benefit from measuring pubertal development using multiple methods (Harden, Kretsch, Moore et al., 2014).

Finally, the CBCL measure of aggression includes a diverse set of behaviors, including physical aggression ('I physically attack other people'), relational aggression ('I am mean to others'), and oppositionality ('I am stubborn'). Factor analytic results support combining these items into a broadband aggression scale, but the operationalization of the aggression construct nevertheless remains relatively broad. The inclusion of behaviors beyond physical aggression might perhaps account for the fact that we find lower heritability for aggression than has been found in many previous behavioral genetic studies (Burt, 2012).

Conclusions

Adolescence is a period of peak risk for antisocial behavior, and this developmental spike in socially problematic behavior is driven by engagement in nonaggressive rule-breaking behaviors. This study suggests that puberty is a critical juncture for the expression of genetic risk for nonaggressive forms of ASB, but future research is necessary to distinguish potential social and biological mechanisms underlying this gene × puberty interaction.

Supporting Information

Additional Supporting Information may be found in the online version of this article:

Appendix S1. Measurement invariance models.

Figure S1. Observed distribution of pubertal development by age and sex.

Figure S2. Observed distributions of rule-breaking and aggression scale scores.

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Key points

- Previous research has found that aggressive behavior differs from non-aggressive rule-breaking in etiology and developmental course. Most of the adolescent spike in antisocial behavior can be accounted for by rule-breaking.
- Using a sample of twins ages 8–20, this paper examined how genetic and environmental influences on rule-breaking aggression differed as a function of age and pubertal status.
- Genetic influences specific to rule-breaking increased with pubertal development, whereas genetic influences specific to aggressive behavior are not moderated by pubertal development.

- Puberty is a critical juncture for the emergence of genetic propensities toward certain types of antisocial behavior.
- Future research should examine mechanisms for how puberty activates genetic risk for rule-breaking forms of antisocial behavior, including effects of gonadal and adrenal hormones on gene expression.

Notes

1. Two items ('disobedient at school' and 'disobedient at home') are categorized as aggression items on the CBCL (Achenbach & Rescorla, 2001). The ostensible content of these items, however, is more consistent with the rule-breaking construct, and these items were both used (with factor analytic support) as indicators of rule-breaking by Burt & Neiderhiser (2009) – whose results we aim to replicate and extend in this paper. The purpose of the EFA, therefore, was to examine how these items performed in our sample. As shown in Table 2, both items loaded strongly on the rule-breaking factor (standardized loadings > .7). All other items loaded on the rule-breaking and aggression factors consistent with the CBCL scoring.
2. In their simulations, Hu and Bentler (1999) found that nearly 100% of misspecified models were rejected when a criterion of RMSEA <.045 in large sample sizes (n > 1,000) and recommended a cut-off of RMSEA <.06 as evidence for good fit to the data.
3. Previous studies have established the measurement invariance of CBCL items across sex (Fonseca-Pedrero, Sierra-Baigrie, Lemos-Giráldez, Paino, & Muñiz, 2012; Yarnell et al., 2013).
4. Alternatively, items could be residualized for covariates using ordinal logistic regression (OLR), which would be consistent with how the items were modeled in measurement invariance models. Scale scores constructed using OLR were nearly perfectly correlated with scores constructed using Poisson regression (rs > .99); however, scores constructed using Poisson regression were preferred because they were easily log-transformed to produce normally distributed variables (see Figure S2).

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