Development and Psychopathology **29** (2017), 155–172

doi:10.1017/S0954579416000109

Antisocial peer affiliation and externalizing disorders: Evidence for Gene × Environment × Development interaction

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Abstract

Gene × Environment interaction contributes to externalizing disorders in childhood and adolescence, but little is known about whether such effects are long lasting or present in adulthood. We examined gene–environment interplay in the concurrent and prospective associations between antisocial peer affiliation and externalizing disorders (antisocial behavior and substance use disorders) at ages 17, 20, 24, and 29. The sample included 1,382 same-sex twin pairs participating in the Minnesota Twin Family Study. We detected a Gene × Environment interaction at age 17, such that additive genetic influences on antisocial behavior and substance use disorders were greater in the context of greater antisocial peer affiliation. This Gene × Environment interaction was not present for antisocial behavior symptoms after age 17, but it was for substance use disorder symptoms through age 29 (though effect sizes were largest at age 17). The results suggest adolescence is a critical period for the development of externalizing disorders wherein exposure to greater environmental adversity is associated with a greater expression of genetic risk. This form of Gene × Environment interaction may persist through young adulthood for substance use disorders, but it appears to be limited to adolescence for antisocial behavior.

Externalizing disorders, such as substance use disorders and adult antisocial personality disorder, are common (British Psychological Society, 2010; Esser et al., 2014; Grant et al., 2006; Substance Abuse and Mental Health Administration, 2013) and associated with a myriad of poor psychosocial and physical health outcomes (British Psychological Society, 2010; Centers for Disease and Control, 2014; Grieg, Baker, Lewin, Webster, & Carr, 2006). Externalizing disorders are complex in that they entail a dynamic interplay of both heritable and environmental influences (Hicks, Foster, Iacono, & McGue, 2013; Rhee & Waldman, 2002). Two processes that are essential to delineating the mechanisms underlying externalizing disorders are gene–environment correlation and interaction. Gene–environment correlation refers to the process whereby genetically influenced traits are associated with exposure to environmental risk (Scarr & McCartney, 1983). Gene × Environment interaction refers to the process wherein genetic influences vary as a function of environmental context (Dick, 2011; Rutter, Moffitt, & Caspi, 2006).[1]

Both gene–environment correlation and interaction have long been shown to contribute to adolescent externalizing problems (i.e., antisocial behavior and problematic substance use; Benner, Kretsch, Harden, & Crosnoe, 2014; Button, Lau, Maughan, & Eley, 2008; Byrd & Manuck, 2014; Cicchetti, Rogosch, & Thibodeau, 2012; Cleveland, Wiebe, & Rowe, 2005; Dick et al., 2007; Feinberg, Button, Neiderhiser, Reiss, & Hetherington, 2007; Hicks, South, Dirago, Iacono, & McGue, 2009). Few studies, however, have examined whether these processes are also present in young adulthood, or whether exposure to environmental risk factors in adolescence has long-lasting effects in terms of moderating genetic risk for externalizing disorders in adulthood. This is a critical gap to fill because it remains imperative to understand how both early and later developmental context may affect the etiology of externalizing disorders across time.

Gene–Environment Interaction and Correlation in Adolescence

The ubiquity of gene–environment correlation processes in adolescence was demonstrated by a landmark meta-analysis that showed many variables assumed to be "environmental" (e.g., parenting and peer interaction) are substantially influenced by additive genetic factors (Kendler & Baker, 2007). Furthermore, research has consistently shown that the associations between such environmental factors and externalizing disorders are at least partially attributable to common additive genetic influence (e.g., Marceau et al., 2013). These findings are often interpreted as supporting the notion of

This research was supported by Grants DA05147 and DA024417 from the National Institute on Drug Abuse and Grant AA09367 from the National Institute of Alcohol Abuse and Alcoholism. Support was also provided by Hatch Project 1006129 from the USDA National Institute of Food and Agriculture (to D.R.S.). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

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1. It is statistically equivalent to say that gene–environment interaction refers to the process in which genetic main effects vary in the context of environmental risk as it is to say environmental main effects vary in the context of genetic risk.

gene–environment correlation (Scarr & McCartney, 1983), or how exposure and selection into specific environmental contexts are related to our unique genotypes. One such process is *evocative* gene–environment correlation, or how children and adolescents evoke certain types of responses from parents and others based on their genetically influenced traits (e.g., some children may evoke frustration or negativity whereas others may evoke warmth or positivity based on their genetically influenced temperament). An alternative process is *active* gene–environment correlation, or how children and adolescents actively select out a particular environment based on their unique genotype (e.g., children likely seek out affiliation with like-minded antisocial or prosocial friends).

Potentially as crucial is that once people have been exposed or selected into specific environmental contexts, such contexts also appear to either amplify or offset genetic risk for externalizing disorders, a process known as Gene × Environment interaction. For example, Hicks (2009) showed a consistent pattern of Gene × Environment interaction for externalizing disorders and several environmental risk factors in adolescence. Specifically, environmental factors that included antisocial peer affiliation, parent–child relationship problems, academic engagement, and stressful life events all moderated additive genetic influences on externalizing disorders such that genetic influences were greater in the context of greater environmental adversity. Because of the consistency of the findings across environmental variables, Hicks et al. speculated that the pattern of increased genetic risk as a function of greater environmental adversity may be a general mechanism underlying risk for externalizing disorders.

Gene × Environment × Development Interplay?

Although research has clearly supported the notion of gene–environment correlation and interaction involving key family, peer, and school factors in relation to adolescent externalizing disorders (see citations above), less research has examined the constancy of gene–environment interplay beyond adolescence and into young adulthood when key externalizing attributes are more common. Specifically, antisocial behavior tends to peak in late adolescence (Blumenstein, Cohen, & Farrington, 1988; Loeber et al., 2012; Moffitt, 1993), and heavy substance use and substance use disorders tend to peak in the early 20s (Centers for Disease and Control, 2012; Johnston, O'Malley, Bachman, & Schulenberg, 2009; Schulenberg & Maggs, 2002; Substance Abuse and Mental Health Administration, 2014). It may be that gene–environment interaction is more relevant to the development of externalizing disorders at the end of adolescence or in early adulthood when they are most common. Alternatively, it may be that gene–environment interaction may be more relevant earlier in development when individuals may be more malleable or sensitive to environmental context (Kendler, Gardner, & Dick, 2011).

Converging evidence supports the latter notion in that gene–environment interaction involving externalizing disorders may be developmentally limited to adolescence. For example, follow-up analyses using the same sample as Hicks et al. (2009) found that poor parent–child relationship quality (Samek, Hicks, et al., 2015) and low academic achievement (Johnson, McGue, & Iacono, 2009) did not moderate genetic risk for externalizing disorders at age 24. Instead, gene–environment correlation explained much of the concurrent and long-term associations between parent–child relationship quality and young adult externalizing disorders (Samek, Hicks, et al., 2015), and shared environmental influences explained much of the association between higher educational attainment and young adult's antisocial behavior symptoms (Johnson et al., 2009). Thus, other confounding family factors (genetic and environmental) explain much of the long-term association between these adolescent environmental contexts and adult outcomes rather than Gene × Environment interaction processes.

However, it remains unclear whether other aspects of young adult's environmental context may amplify or offset genetic risk for externalizing disorders: particularly those aspects that may be more important to young adults than parenting or school. Because antisocial peer affiliation is the one of the strongest environmental correlates of externalizing problems across childhood, adolescence, and young adulthood (Brendgen, 2012; Dishion & Owen, 2002; Lansford, Yu, Pettit, Bates, & Dodge, 2014; Monahan, Rhew, Hawkins, & Brown, 2014; Monahan, Steinberg, & Cauffman, 2009; Wichers, Gillespie, & Kendler, 2013), it may continue to be a factor that amplifies or offsets genetic risk for externalizing disorders in early adulthood.

Antisocial Peer Affiliation

A large body of research has demonstrated important bidirectional influence of peers and substance use in adolescence (Curran, Stice, & Chasin, 1997; Van Ryzin & Dishion, 2014; Van Ryzin, Fosco, & Dishion, 2012); however, comparatively fewer investigations have evaluated such effects in early adulthood. In a landmark study, Dishion and Owen (2002) showed evidence of both selection and socialization in the linkages between substance use and deviance within friendships. Specifically, Dishion and Owen showed that affiliation with substance-using friends in early adolescence was associated with subsequent substance use in middle adolescence, which then reinforced a pattern of associating with deviant friends in late adolescence and subsequent substance use in early adulthood. In relation to antisocial behavior more generally, other research suggests little impact of peer socialization beyond age 20 (Monahan et al., 2009), particularly in relation to an aggregate externalizing measure (Samek, Goodman, Erath, McGue, & Iacono, in press). Thus, it may be that gene–environment interaction involving antisocial peer affiliation and externalizing disorders is less relevant in adulthood than in adolescence.

Prior research has shown that child and adolescent antisocial peer affiliation is heritable (Bullock, Deater-Deckard, & Leve, 2006; Cleveland et al., 2005) and that the association

https://doi.org/10.1017/S0954579416000109 Published online by Cambridge University Press

between adolescent antisocial peer affiliation and adolescent externalizing problems is largely explained by shared genetic factors (Fowler et al., 2007; Teneyck & Barnes, 2015). Furthermore, Kendler et al. (2007) showed heritability estimates of retrospective reports of antisocial peer affiliation increased and then stabilized over time. Specifically, the heritability of antisocial peer affiliation increased from about 30% for 8- to 11-year-olds to about 50% for ages 15–17, 18–21, and 22–25. One possibility is that as antisocial peer affiliation becomes more heritable over time, gene–environment correlation processes become even more important to the emergence and maintenance of externalizing disorders, but this hypothesis has not yet been evaluated.

Prior research has also consistently shown a Gene × Environment interaction such that the additive genetic influences on adolescent externalizing problems are greater in the context of greater antisocial peer affiliation (Fowler et al., 2007; Harden, Hill, Turkheimer, & Emery, 2008; Hicks et al., 2009). It is unknown, however, if antisocial peer affiliation continues to amplify genetic risk for externalizing disorders in young adulthood or if this Gene × Environment interaction is developmentally limited to adolescence as has been found for parenting and school factors (Johnson et al., 2009; Samek, Hicks, et al., 2015). An effect of antisocial peer affiliation limited to adolescence would be consistent with several nongenetically informed studies demonstrating that individuals become more resistant to antisocial peer influences over time (Gardner & Steinberg, 2005; Sumter, Bokhorst, Steinberg, & Westenberg, 2009), potentially due to continued brain development and improvement in cognitive skills throughout adolescence and young adulthood (Albert, Chein, & Steinberg, 2013).

Failure to detect a Gene × Environment interaction between antisocial peer affiliation and adult externalizing disorders, combined with similar findings for parenting and academic achievement (Johnson et al., 2009; Samek, Hicks, et al., 2015), would suggest that adolescence may be a critical period wherein greater autonomy and exposure into high-risk environments provides a catalyst for previously unexpressed genetic risk. Once initiated, however, selection or active gene–environment correlation processes may maintain the association between externalizing disorders and contextual risk in adulthood.

We tested this research question by examining gene–environment interplay between externalizing disorders and antisocial peer affiliation at multiple time points spanning adolescence (age 17), early adulthood (ages 20 and 24), and later adulthood (age 29). Further, we tested whether adolescent antisocial peer affiliation had moderating effects on genetic influences on adult externalizing disorders. Given prior research (Johnson et al., 2009; Monahan et al., 2009; Samek, Hicks, et al., 2015), as well as our theory that adolescence may be a developmentally limited critical period wherein high-risk environments provide a catalyst for previously unexpressed genetic risk, we predicted that antisocial peer affiliation would moderate additive genetic influences on externalizing disorders in adolescence. We also expected that adolescent antisocial peer affiliation would not moderate additive genetic influences on adult externalizing disorders. Rather, we predicted that externalizing disorders and antisocial affiliation would exhibit significant additive genetic correlations (i.e., common genetic influences), both concurrently and prospectively across adolescence and young adulthood (thus evidencing greater selection or active gene–environment correlation than gene–environment interaction mechanisms postadolescence).

We evaluated these hypotheses using an aggregated count of symptoms of clinical externalizing disorders, including several substance use disorders and the adult criteria for antisocial personality disorder (hereafter referred to as adult antisocial behavior symptoms). However, given the slight difference in age at which rates of antisocial behavior versus substance use disorder peak (e.g., see Loeber et al., 2012; Moffitt, 1993; Substance Abuse and Mental Health Administration, 2014), we also evaluated the extent of differences in gene–environmental interplay involving antisocial behavior *versus* substance use disorder by developmental stage.

Method

Participants were members of the Minnesota Twin Family Study (Iacono, Carlson, Taylor, Elkins, & McGue, 1999), a longitudinal, cohort-sequential study of twins born in Minnesota. Twin families were identified using publicly available birth certificates (birth years 1972 to 1984) and were located using several public databases. Participating families had twins who were the biological offspring of their parents and lived within a day's drive of the university laboratory; neither twin could have a mental or physical handicap that would impair study participation. About 90% of twins were successfully located, and 83% of eligible and located families agreed to participate. After a description of the study to twins and parents, parents provided written consent for minors and minors provided written assent, while those 18 years and older provided written consent. The University of Minnesota Institutional Review Board approved all study protocols.

The sample used both the older and younger cohort of twins, thus included 2,764 individuals from 1,382 same-sex twin pairs (52% female, 65% monozygotic), assessed at the target ages of 17 ($M = 17.8$ years, $SD = 0.69$, $N = 2{,}577$), 20 ($M = 21.0$ years, $SD = 0.82$, $N = 2{,}450$), 24 ($M = 25.0$ years, $SD = 0.90$, $N = 2{,}499$), and 29 ($M = 29.4$ years, $SD = 0.67$, $N = 2{,}496$) years old (see Iacono et al., 1999, for a detailed study overview). Consistent with demographics of Minnesota for the relevant birth years, nearly all participants were of European American ancestry (95%). Participation rates ranged from 88% to 93% across follow-up assessments. To evaluate attrition, we compared mean differences in symptoms of externalizing disorder symptoms at age 17 for those who did or did not complete adult assessments. Those who participated in adult assessments had slightly fewer externalizing symptoms at age 17 than those who did not participate,

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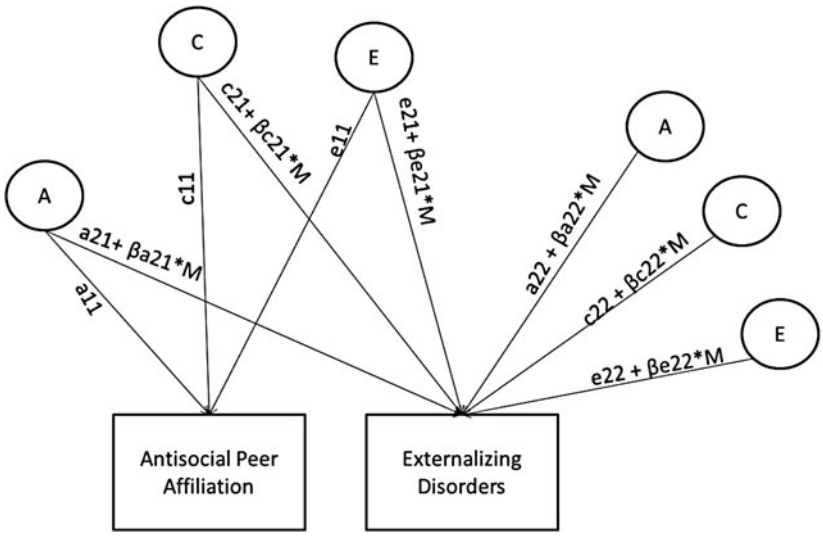

Figure 1. Gene–environment interaction in the presence of gene–environment correlation. Separate models were evaluated for antisocial peer affiliation in relation to externalizing disorders at ages 17, 20, 24, and 29, in addition to models evaluating the prospective relationship between antisocial peer affiliation at age 17 with externalizing disorders at ages 20, 24, and 29. A, Genetic influences; C, shared environmental influences; E, nonshared environmental influences. Parameters a11, c11, and e11 refer to the genetic and environmental influences on the moderator (antisocial peer affiliation). Parameters a21, c21, and e21 refer to the genetic and environmental influences on the moderator (antisocial peer affiliation) in common with the dependent variable (externalizing disorders). Parameters a22, c22, and e22 refer to the unique genetic and environmental influence on the dependent variable (externalizing disorders); β describes the magnitude and direction of moderation effect; and M indicates the level of the moderator. Moderation can influence both/either the common or unique variance for externalizing disorders.

but the effect sizes were small with mean Cohen *d* values of –0.21, –0.16, and –0.03 at ages 20, 24, and 29, respectively, indicating little evidence of attrition effects.

Measures

Antisocial peer affiliation. Participants rated characteristics of their peer groups using the Friends survey (Burt, McGue, & Iacono, 2009; Walden, McGue, Lacono, Burt, & Elkins, 2004), which consisted of 7 to 9 items depending on developmental stage.[2] Items (e.g., "my friends enjoy getting drunk" and "my friends know where to buy drugs") were rated from 1 (*all of my friends are like that*) to 4 (*none of my friends are like that*); αs ranged from 0.83 to 0.88.

Externalizing disorders. Structured interviews were used to assess DSM-III-R symptoms of nicotine dependence, alcohol use disorder, illicit drug use disorder, and adult antisocial behavior (adult criteria for antisocial personality disorder) at ages 17, 20, 24, and 29. Substance use disorders were assessed using the Substance Abuse Module of the Composite International Diagnostic Interview (Robins, Babor, & Cottler, 1987), and adult antisocial behavior was assessed using an interview adapted from the Structured Clinical Interview for DSM-III-R Axis II (Spitzer, Williams, & Gibbon, 1987). Interviews were reviewed by at least two individuals with advanced training in clinical diagnoses, and consensus among the reviewers was reached prior to symptom assignment. Reliability was assessed by double-coding a randomly selected subsample of 600 Minnesota Twin Family Study participants. Kappa coefficients indexing diagnostic reliability were >0.90 for all substance use disorders and 0.79 for adult antisocial behavior. Abuse and dependence symptoms were combined to calculate symptom count variables for alcohol and illicit drug disorders. Because we were interested in evaluating a general measure of externalizing disorders, symptom counts were standardized (*z* scored) and averaged to calculate an externalizing composite at each age (mean *r* values among *z* score scales were .50 at age 17, .45 at age 20, and .39 at ages 24 and 29). Subsequent analyses evaluated differences in antisocial behavior versus substance use disorders. Here, substance use disorder symptom counts were standardized and averaged within each age (mean *r* values among *z* score scales were .53 at age 17, .41 at age 20, .34 at age 24, and .35 at age 29).

Analytic plan

Structural equation modeling was used to evaluate gene–environment interplay for the concurrent and prospective associations between antisocial peer affiliation and externalizing disorders using Mx software (Neale, 2006). Full information maximum likelihood was used to adjust parameter estimates for missing data (Enders & Bandalos, 2001; Johnson & Young, 2011). To better approximate normality, externalizing composites were log-transformed prior to analysis. Consistent with prior research, sex, age, age^2, and Age × Sex were

2. Items were added, removed, or worded slightly differently in the adolescent and adult versions; for example, "My friends get into trouble at school" was removed after age 17 because schooling was over after this assessment.

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Table 1. *Means (standard deviations) of externalizing symptom counts across sex*

	Males ($n = 1,333$)[a]	Females ($n = 1,436$)	Sex Difference Cohen *d*
Age 17			
Nicotine dependence	0.97 (1.83)	0.75 (1.66)	0.13
Alcohol use disorder	0.86 (1.78)	0.40 (1.24)	0.30
Illicit drug use disorder	0.65 (1.88)	0.29 (1.16)	0.23
Adult antisocial behavior	0.99 (1.39)	0.53 (1.00)	0.38
Age 20			
Nicotine dependence	1.43 (1.94)	0.91 (1.64)	0.29
Alcohol use disorder	1.70 (2.18)	0.50 (1.26)	0.67
Illicit drug use disorder	0.96 (2.05)	0.36 (1.31)	0.35
Adult antisocial behavior	1.27 (1.28)	0.55 (0.80)	0.67
Age 24			
Nicotine dependence	1.32 (1.84)	0.92 (1.62)	0.23
Alcohol use disorder	1.56 (2.02)	0.60 (1.42)	0.55
Illicit drug use disorder	0.73 (1.80)	0.32 (1.17)	0.27
Adult antisocial behavior	1.20 (1.12)	0.65 (0.85)	0.55
Age 29			
Nicotine dependence	1.24 (1.78)	0.80 (1.53)	0.27
Alcohol use disorder	1.03 (1.80)	0.34 (1.09)	0.46
Illicit drug use disorder	0.61 (1.73)	0.22 (1.07)	0.27
Adult antisocial behavior	0.99 (1.01)	0.50 (0.73)	0.56

Note: The sample sizes reported for males and females reference the eligible sample size (total sample); however, specific numbers (*n*s) for each measure varied across assessment and by externalizing disorder (at age 17, *n*s = 1,191–1,246 for males and 1,336–1,369 for females; at age 20, *n*s = 1,103–1,112 for males and 1,326–1,336 for females; at age 24, *n*s = 1,130–1,172 for males and 1,296–1,317 for females; at age 29, *n*s = 1,181–1,182 for males and 1,314–1,314 for females). Nicotine dependence symptom counts ranged from 0 to 7. Alcohol and illicit drug use disorder symptom counts (abuse + dependence) ranged from 0 to 10. Adult antisocial behavior (adult criteria for adult antisocial personality disorder) symptom counts ranged from 0 to 10. Males had significantly higher mean symptom counts than females for all externalizing disorders ($p < .05$). The Cohen *d* values show the magnitude of the gender difference effect: 0.2–0.3 is considered small, 05 medium, and >0.8 large.

[a]This include five triplets that were not used in subsequent twin analyses.

covaried out of all phenotypes prior to modeling (age and sex adjustments were conducted within assessment). Univariate and bivariate models were first fit to estimate the additive genetic (A), shared environmental (C), and nonshared environmental (E) influences on study phenotypes, where A refers to genetic influences on twin similarity, C refers to environmental influences on twin similarity, and E refers to environmental influences on twin differences. ACE parameters are estimated by comparing the relative similarity of monozygotic (MZ) and dizygotic (DZ) twin pairs. Additive genetic effects are inferred when MZ correlations are greater than DZ correlations. Shared environmental effects are inferred when DZ correlations are greater than half of MZ correlations. Nonshared environmental effects are inferred when MZ correlations are less than 1.0 (for a detailed overview of ACE modeling, see Rijsdijk & Sham, 2002).

Table 2. *Phenotypic correlations*

	1	2	3	4	5	6	7	8
1. EXT at age 17	1.0							
2. Antisocial peers at age 17	.63	1.0						
3. EXT at age 20	.58	.57	1.0					
4. Antisocial peers at age 20	.39	.58	.57	1.0				
5. EXT at age 24	.53	.49	.71	.50	1.0			
6. Antisocial peers at age 24	.37	.51	.48	.65	.54	1.0		
7. EXT at age 29	.48	.44	.64	.43	.70	.47	1.0	
8. Antisocial peers at age 29	.40	.45	.47	.56	.49	.64	.51	1.0

Note: EXT, Externalizing disorders. All variables were age, sex, Age × Age, and Age × Sex adjusted prior to phenotypic and biometric analysis. All correlations were significantly different from zero at $p < .001$.

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Table 3. *Twin correlations and standardized ACE estimates*

	MZ Twin (n = 902 pairs)[a]	DZ Twin (n = 480 pairs)[a]	a^2	c^2	e^2
EXT at age 17	.70 (.66, .73)	.46 (.38, .53)	0.55 (0.41, 0.70)	0.16 (0.02, 0.29)	0.29 (0.26, 0.32)
EXT at age 20	.59 (.54, .63)	.38 (.30, .46)	0.45 (0.29, 0.62)	***0.15*** ***(0.00, 0.30)***	0.40 (0.36, 0.45)
EXT at age 24	.58 (.53, .62)	.32 (.24, .41)	0.56 (0.39, 0.64)	***0.03*** ***(0.00, 0.19)***	0.41 (0.37, 0.45)
EXT at age 29	.53 (.48, .58)	.32 (.23, .40)	0.46 (0.28, 0.59)	***0.08*** ***(0.00, 0.24)***	0.46 (0.41, 0.51)
Antisocial peer affiliation at age 17	.67 (.63, .72)	.55 (.46, .62)	0.29 (0.15, 0.46)	0.40 (0.24, 0.53)	0.31 (0.27, 0.35)
Antisocial peer affiliation at age 20	.60 (.55, .64)	.44 (.35, .51)	0.32 (0.16, 0.49)	0.28 (0.12, 0.43)	0.40 (0.36, 0.44)
Antisocial peer affiliation at age 24	.53 (.48, .59)	.41 (.33, .49)	0.21 (0.03, 0.40)	0.32 (0.15, 0.48)	0.47 (0.42, 0.53)
Antisocial peer affiliation at age 29	.50 (.44, .55)	.24 (.15, .33)	0.51 (0.34, 0.56)	***0.00*** ***(0.00, 0.15)***	0.49 (0.44, 0.54)

Note: EXT, Externalizing disorders. This table shows intraclass twin correlations for monozygotic (MZ) and dizygotic (DZ) twins as well as standardized estimates of additive genetic (a^2), shared environmental (c^2), and nonshared environmental (e^2) influences from univariate decompositions.
[a]Sample sizes (n) shown for MZ and DZ are based on eligible sample size (total sample); actual numbers (ns) associated with correlations ranged from 681 to 823 for MZ pairs and 369 to 442 for DZ pairs (across measure and age of assessment). Ninety-five percent confidence intervals are provided in parentheses. Coefficients are significant if the confidence interval does not cross zero (nonsignificant coefficients are shown in bold italic for clarity of presentation). All variables were adjusted for age, sex, Age × Sex, and age^2 prior to analysis.

Table 4. *Phenotypic, genetic, and environmental correlations between antisocial peer affiliation and externalizing disorders from ages 17 to 29*

	r	rA	rC	rE	A	C	E
Cross-Sectional Correlations in Adolescence							
Antisocial peers at age 17 and EXT at age 17	.63 (.60, .66)	.64 (.48, .79)	1.0 (.92, 1.0)	.29 (.21, .36)	.36 (.21, .54)	.50 (.34, .64)	.14 (.10, .18)
Cross-Sectional Correlations in Young Adulthood							
Antisocial peers at age 20 and EXT at age 20	.57 (.53, .60)	.65 (.47, .86)	1.0 (.69, 1.0)	.26 (.20, .33)	.42 (.27, .55)	.18 (.05, .31)	.40 (.36, .45)
Antisocial peers at age 24 and EXT at age 24	.54 (.51, .57)	.86 (.63, 1.0)	1.0 (.54, 1.0)	.21 (.14, .28)	.52 (.30, .77)	.31 (.08, .51)	.17 (.11, .23)
Antisocial peers at age 29 and EXT at age 29	.51 (.47, .54)	.61 (.45, .76)	***1.0*** ***(−.52, 1.0)***	.34 (.28, .40)	.56 (.30, .75)	***.12*** ***(−.03, .35)***	.32 (.25, .40)
Longitudinal Correlations of Adolescent Peer Affiliation Predicting Young Adult EXT							
Antisocial peers at age 17 and EXT at age 20	.57 (.53, .61)	.70 (.53, .92)	1.0 (.75, 1.0)	.14 (.05, .22)	.45 (.26, .67)	.47 (.26, .64)	.08 (.03, .14)
Antisocial peers at age 17 and EXT at age 24	.49 (.44, .54)	.68 (.49, .93)	1.0 (.62, 1.0)	.10 (.02, .18)	.55 (.33, .79)	.38 (.15, .58)	.07 (.01, .13)
Antisocial peers at age 17 and EXT at age 29	.44 (.39, .49)	.47 (.19, .74)	1.0 (.63, 1.0)	.14 (.05, .23)	.38 (.12, .67)	.50 (.24, .74)	.12 (.04, .19)

Note: EXT, Externalizing disorders. The table shows phenotypic correlations (r) as well as additive genetic (rA), shared environmental (rC), and nonshared environmental (rE) correlations from bivariate, full ACE Cholesky decompositions. It also shows the proportion of phenotypic covariance that is due to additive genetic (A), shared environmental (C), and nonshared environmental (E) influence, that is, how much genetic and environmental influences contributed to the total phenotypic covariation (r in column 1); note that the last three columns add to 1.0. For example, 36% of the total covariation between antisocial peer affiliation and EXT at age 17 was due to additive genetic influences, 50% due to shared environmental influences, and 14% due to nonshared environmental influences (36 + 50 + 14 = 100%). All variables were adjusted for age, sex, Age × Sex, and age^2 prior to analysis. Significant coefficients are those with a confidence interval that does not cross zero (those that are not significant are denoted in bold italic for clarity of presentation).

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Table 5. *Fit statistics for gene–environment interplay models of antisocial peer affiliation and EXT at ages 17, 20, 24, and 29*

	−2LL	*df*	$\Delta\chi^2$	Δdf	*p*	AIC	Adj. BIC	DIC
Cross-Sectional Associations in Adolescence								
Antisocial Peer Affiliation at Age 17 and EXT at age 17								
1. Full ACE moderation	7499.71	3371				757.71	−2260.65	−4515.54
2. No ACE moderation	7982.39	3377	482.68	6	<.001	1228.39	−2030	−4288.91
3. Best fitting model: common A, unique AE moderation only; c22 parameter dropped	7499.89	3375	0.18	4	.99	749.89	−2267.69	−4525.26
Cross-Sectional Associations in Adulthood								
Antisocial Peer Affiliation at Age 20 and EXT at Age 20								
4. Full ACE moderation	10922.21	4399				2124.21	−2963.98	−5907.71
5. No ACE moderation	11022.03	4405	99.82	6	<.001	2212.03	−2925.55	−5873.31
6. Best fitting model: common A and unique E moderation only; c22 parameter dropped	10927.39	4404	5.18	5	.39	2119.39	−2970.96	−5918.04
Antisocial Peer Affiliation at Age 24 and EXT at Age 24								
7. Full ACE moderation	10559.59	4195				2169.59	−2655.56	−5462.6
8. No ACE moderation	10629.32	4201	69.73	6	<.001	2227.32	−2632.04	−5443.10
9. Best fitting model: unique E moderation only; c22 and a22 parameters dropped	10566.97	4202	7.38	7	.39	2162.97	−2665.11	−5476.83
Antisocial Peer Affiliation at Age 29 and EXT at Age 29								
10. Full ACE moderation	11657.6	4563				2531.6	−2993.42	−6047.05
11. No ACE moderation	11778.37	4569	120.77	6	<.001	2640.37	−2944.64	−6002.28
12. Best fitting model: common AE and unique AE moderation only; c11, c21, and c22 parameters dropped	11659.65	4568	2.05	5	.84	2523.65	−3002.07	−6059.04
Longitudinal Associations: Adolescent Peer Affiliation and Adult EXT								
Antisocial Peer Affiliation at Age 17 and EXT at Age 20								
13. Full ACE moderation	7870.57	3275				1320.57	−1904.05	−4094.73
14. No ACE moderation	7963.48	3281	92.91	6	<.001	1401.48	−1868.29	−4062.98
15. Best fitting model: unique CE moderation only; c22 parameter dropped	7877.01	3280	6.44	5	.27	1317.01	−1909.75	−4103.77
Antisocial Peer Affiliation at Age 17 and EXT at Age 24								
16. Full ACE moderation	8141.25	3262				1617.25	−1745.53	−3927.51
17. No ACE moderation	8167.84	3268	26.59	6	<.001	1632.84	−1742.93	−3928.93
18. Best fitting model: common A and unique E moderation only; a22, c21, c22, and e21 parameters dropped	8152.8	3270	11.55	8	.17	1612.8	−1754.02	−3941.35
Antisocial Peer Affiliation at Age 17 and EXT at Age 29								
19. Full ACE moderation	8103.61	3254				1595.61	−1750.09	−3926.72
20. No ACE moderation	8146.76	3260	43.15	6	<.001	1626.76	−1739.21	−3919.85
21. Best fitting model: unique E moderation only; c22 parameter dropped	8109.56	3260	2.95	6	.82	1589.56	−1757.81	−3938.45

Note: –2LL, –2 log likelihood; $\Delta\chi^2$, chi-square change; AIC, Akaike information criterion; BIC, Bayesian information criterion; DIC deviance information criterion; A, additive genetic effects; C, shared environmental effects; E, nonshared environmental effects; EXT, externalizing disorders. The baseline model of comparison used in chi-square difference tests is the full ACE moderation model, which allows for ACE moderation on all common and unique parameters. The $\Delta\chi^2$ is the difference between the –2LL in the baseline model (full ACE moderation) compared to the other modes tested.

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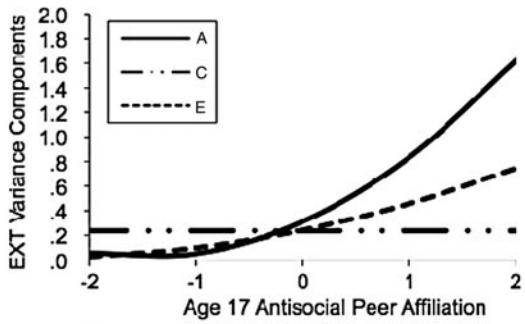
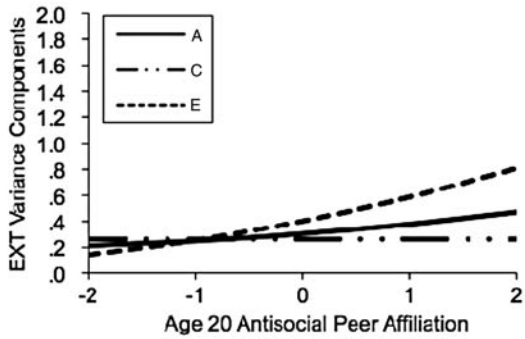
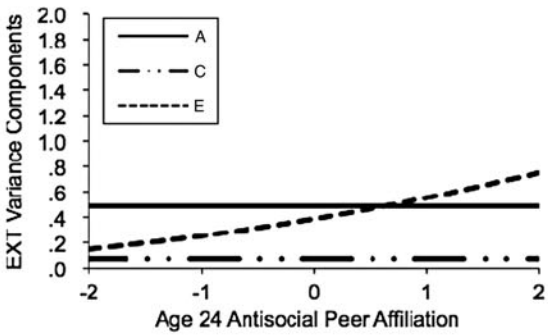
Cross-Sectional Relationships



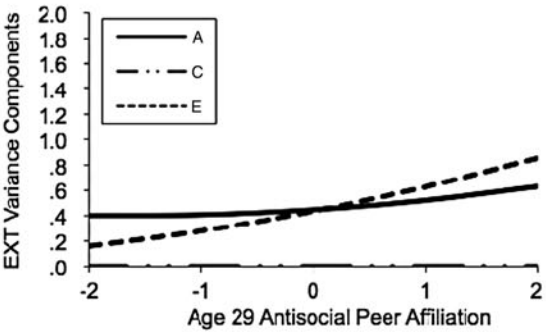
(a) Antisocial peer affiliation at age 17 moderating genetic and environmental influences on EXT at age 17



(b) Antisocial peer affiliation at age 20 moderating genetic and environmental influences on EXT at age 20

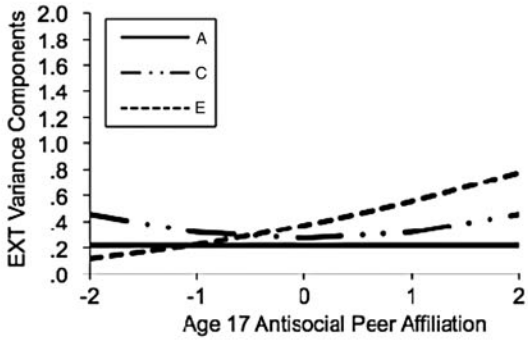


(c) Antisocial peer affiliation at age 24 moderating genetic and environmental influences on EXT at age 24

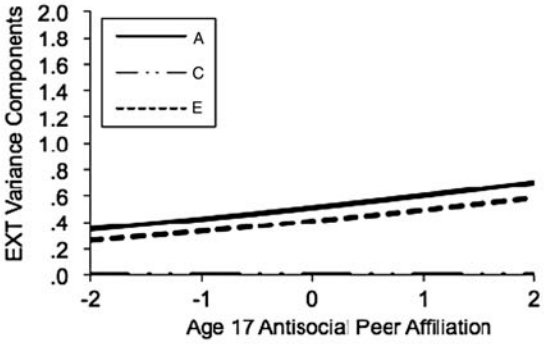


(d) Antisocial peer affiliation at age 29 moderating genetic and environmental influences on EXT at age 29

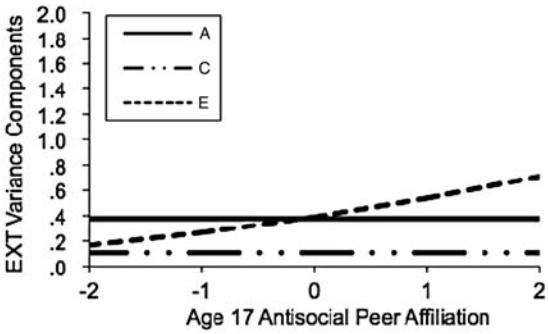
Longitudinal Relationships



(e) Antisocial peer affiliation at age 17 moderating genetic and environmental influences on EXT at age 20



(f) Antisocial peer affiliation at age 17 moderating genetic and environmental influences on EXT at age 24



(g) Antisocial peer affiliation at age 17 moderating genetic and environmental influences on EXT at age 29

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Next, we tested for Gene × Environment interaction in the presence of gene–environment correlation (Purcell, 2002). As illustrated in Figure 1, this bivariate analysis decomposes the ACE contributions on the covariance between antisocial peer affiliation and externalizing disorders (a11*a21, c11*c21, e11*e21) and the variance unique to externalizing disorders (a22, c22, e22). In this bivariate decomposition, the genetic and environmental covariance (e.g., a11*a21) is standardized to give estimates of genetic and environmental correlations (range –1.0 to 1.0). As shown in Figure 1, in the case of a Gene × Environment interaction, ACE parameters are also adjusted for the direction and size of the moderation (β) and the level of the moderator (M), here antisocial peer affiliation. Moderation can occur on ACE effects common to antisocial peer affiliation and externalizing disorders (a21 + βa21 × M, c21 + βc21 × M, e21 + βe21 × M) or unique to externalizing disorders (a22 + βa22 × M, c22 + βc22 × M, e22 + βe22 × M). Model fit was evaluated using the –2 log likelihood (–2LL) and testing the likelihood ratio test between comparison models. Several information theoretic fit indices were also used to evaluate fit including the Akaike information criterion, the sample-size adjusted Bayesian information criterion, and the deviance information criterion, in which smaller values indicated better fit. For all model comparisons, we compared the full ACE moderation model to a model that dropped all ACE moderation parameters. If the full ACE moderation model fit better than the no ACE moderation model, follow-up comparisons were made by dropping nonsignificant parameters (i.e., 95% confidence intervals that included zero) to identify the best fitting, most parsimonious model.

Results

Preliminary analyses

Descriptive statistics and phenotypic correlations for study phenotypes are provided in Tables 1 and 2, respectively. Males had significantly higher mean externalizing symptom counts than females across all externalizing disorders (all *p*s $< .05$). All phenotypes were significantly and substantially correlated (mean $r = .52$, range $= .37$–$.71$, all *p*s $< .001$).

Twin correlations and univariate ACE estimates are reported in Table 3. Additive genetic influences on the externalizing composites were moderate and stable over time (a^2 range $= 0.46$–0.56). Shared environmental influences on externalizing disorders were small at age 17 ($c^2 = 0.16$) and not significantly different from zero after age 17. Additive genetic influences on antisocial peer affiliation were small to moderate across time (a^2 range $= 0.21$–0.51). Shared environmental influences on antisocial peer affiliation were moderate through age 24 and not significantly different from zero at age 29. Nonshared environmental influence on externalizing disorders and antisocial peer affiliation tended to increase over time.

Gene–environment correlations

Cross-sectional and longitudinal phenotypic, genetic, and environmental correlations between antisocial peer affiliation and externalizing composites are reported in Table 4. Genetic correlations between antisocial peer affiliation and the externalizing composite were medium to large (range $= 0.47$–0.86), and all were significantly different from zero (as indicated by the 95% confidence intervals not crossing zero). Shared environmental correlations were also large, though total shared environmental variance was small for the externalizing composites, suggesting little practical effect. Nonshared environmental correlations were moderate for the cross-sectional correlations at each time point and small for the longitudinal correlations (i.e., age 17 to 20, age 17 to 24, and age 17 to 29). Table 4 also shows the proportion of the phenotypic covariance due to genetic and environmental influence (columns add to 1.0). As expected given the genetic correlations, common genetic influences were substantial within and across time.

Gene × Environment interaction in adolescence

Table 5 shows the fit statistics for all Gene × Environment interaction models. For the cross-sectional association between antisocial peer affiliation and the externalizing composite at age 17, as reported in rows 1 and 2, the no ACE moderation model fit significantly worse than the full ACE moderation model. This was evidenced by the significant likelihood ratio test and poorer fit in Akaike information criterion, adjusted the sample-size adjusted Bayesian information criterion, and deviance information criterion values. For the best fitting model (row 3), only parameters that were significantly different from zero (i.e., 95% confidence interval did not include zero) were retained. The best fitting model included common A and unique AE moderation parameters only; common CE

Figure 2. Antisocial peer affiliation moderating genetic and environmental influences on externalizing disorders: cross-sectional and longitudinal relationships. Changes in the unstandardized ACE variance components of externalizing disorders (EXT) are given as a function of antisocial peer affiliation for the best fitting models (see Table 5). A, additive genetic influence; C, shared environmental influences; E, nonshared environmental influence. The *y*-axis represents the unstandardized variance component score (shown for A, C, and E). The *x*-axis represents the value of antisocial peer affiliation (shown in 0, ±1, and ±2 *SD*). All composites were adjusted for age, sex, Age × Sex, and age^2 by regressing these covariates out prior to analysis. Presented are the (a) results for the cross-sectional associations at age 17, (b) cross-sectional associations at age 20, (c) results for the cross-sectional association at age 24, (d) cross-sectional associations at age 29, (e) results for the longitudinal association between adolescent peer affiliation (age 17) and early emerging adult EXT (age 20), (f) interaction results for the longitudinal association between adolescent peer affiliation (age 17) and late emerging adult EXT (age 24), and (g) interaction results for the longitudinal association between adolescent peer affiliation (age 17) and late young adult EXT (age 29).

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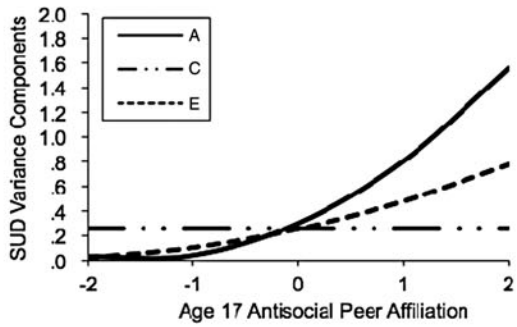
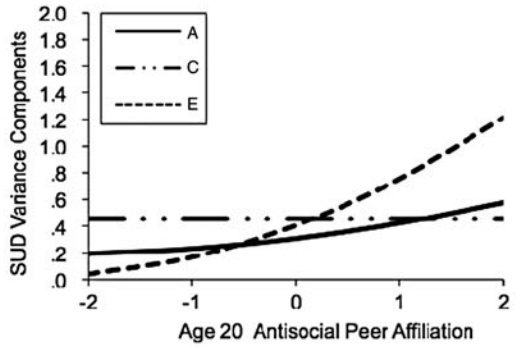
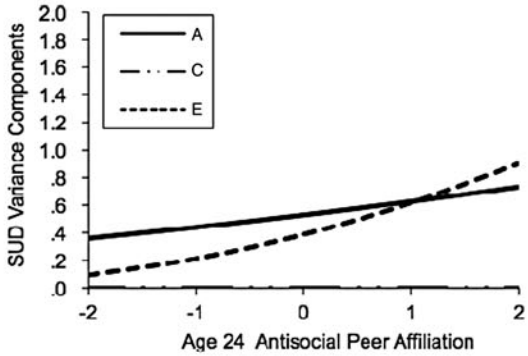
Cross-Sectional Relationships



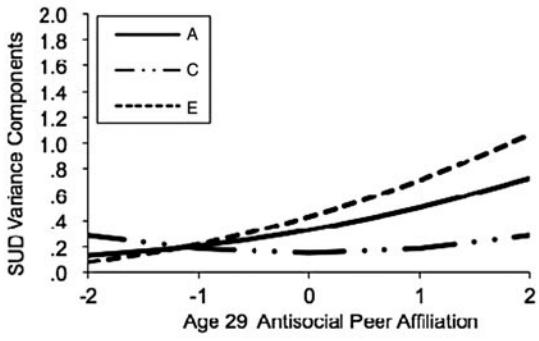
(a) Antisocial peer affiliation at age 17 moderating genetic and environmental influences on SUD symptoms at age 17



(b) Antisocial peer affiliation at Age 20 moderating genetic and environmental influences on SUD symptoms at age 20

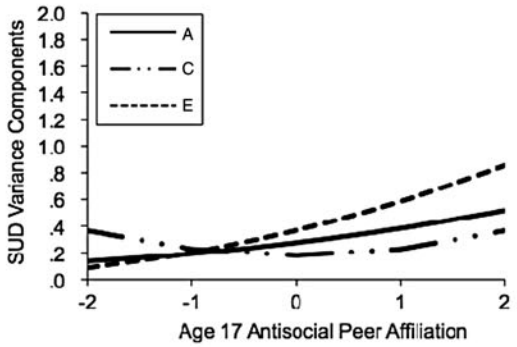


(c) Antisocial peer affiliation at age 24 moderating genetic and environmental influences on SUD symptoms at age 24

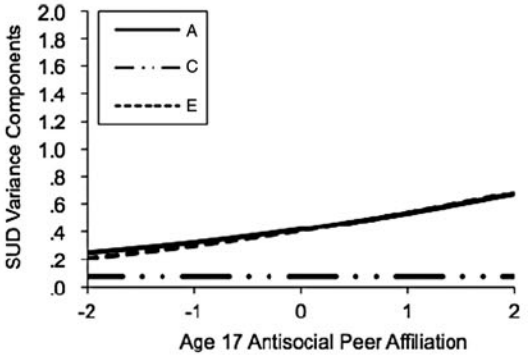


(d) Antisocial peer affiliation at age 29 moderating genetic and environmental influences on SUD symptoms at age 29

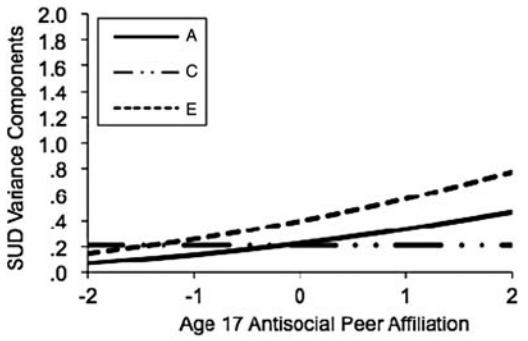
Longitudinal Relationships



(e) Antisocial peer affiliation at age 17 moderating genetic and environmental influences on SUD symptoms at age 20



(f) Antisocial peer affiliation at age 17 moderating genetic and environmental influences on SUD symptoms at age 24



(g) Antisocial peer affiliation at age 17 moderating genetic and environmental influences on SUD symptoms at age 29

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and unique C moderation parameters (and the residual C influence on externalizing disorders, i.e., c22) could be dropped without a decrement to model fit (fit statistics reported in row 3). The A moderation parameters could not be dropped without a decrement to model fit; thus, antisocial peer affiliation at age 17 moderated the additive genetic influences on externalizing disorders at age 17. Gene × Environment interaction results for the best fitting model are plotted in Figure 2a. The figure depicts how the additive genetic influence on externalizing disorders was greater in the context of greater antisocial peer affiliation. A similar pattern was also observed for nonshared environmental influences. Antisocial peer affiliation did not moderate the shared environmental influences on externalizing disorders.

Gene × Environment interaction in young adulthood

As depicted by the fit statistics in Table 5 (rows 4–12) and the plot of variance components in Figure 2(b–d), there was little evidence that antisocial peer affiliation moderated genetic influences on externalizing disorders at ages 20 (Fig. 2b), 24 (Fig. 2c), or 29 (Fig. 2d). Although the best fitting models at ages 20 and 29 included parameters for the moderation of genetic influences on externalizing disorders, the changes in additive genetic influence at ages 20 and 29 were much smaller in magnitude in comparison to the additive genetic changes at age 17 (see the additive genetic moderation line in Fig. 2b and d in comparison to the additive genetic moderation line Fig. 2a). However, and like the age 17 results, nonshared environmental influences on externalizing disorders were greater in the context of greater antisocial peer affiliation at ages 20, 24, and 29, and the effects sizes were moderate in magnitude across time.

Long-term Gene × Environment interaction

Finally, we tested whether antisocial peer affiliation at age 17 moderated the genetic and environmental influences on externalizing disorders at ages 20, 24, and 29. The results for the best fitting models are depicted in Figure 2e–g (model fit statistics are reported in Table 5, rows 13–24). There was no evidence that adolescent antisocial peer affiliation moderated genetic influences on adult externalizing disorders at ages 20 or 29. However, at age 24, there was evidence of longitudinal common genetic moderation such that the genetic influences on externalizing disorders at age 24 were greater in the context of a greater affiliation with antisocial peers at age 17. Thus, in this sole case, there was evidence for longitudinal genetic moderation. Nevertheless, the effect size at age 24 was clearly not as substantial as the cross-sectional results at age 17 (compare Fig. 2a and f). Across longitudinal analyses, unique nonshared environmental moderation was significant, such that greater antisocial peer affiliation in adolescence was associated with greater nonshared environmental influences on externalizing disorders in adulthood. Thus, longitudinal results were generally consistent with cross-sectional associations at ages 17, 20, 24, and 29.

To control for prior antisocial peer affiliation and externalizing disorders, we also fit Gene × Environment interaction models between externalizing disorders and antisocial peer affiliation in adulthood after regressing out adolescent externalizing and antisocial peer affiliation on the target phenotypes at the adult ages (i.e., models were fit using residualized scores). The results for these analyses are provided in online-only supplementary Tables S.1–S.3 and Figure S.1. All results were consistent with those reported for the unadjusted phenotypes in that there was no evidence for additive genetic moderation of externalizing disorders past age 17.

Finally, to evaluate whether effects depended on substance misuse versus general antisocial behavior, we tested Gene × Environment interaction models for substance use disorder symptom counts (see Figure 3) and adult antisocial behavior symptom counts in separate models (see Figure 4; detailed fit statistics shown in Table 6). Following results for the externalizing composite, for both substance use disorders and adult antisocial behavior, results showed clear evidence for additive genetic moderation for the cross-sectional relationships at age 17, such that the genetic influence was greater in the context of a greater degree of antisocial peer affiliation. For adult antisocial behavior, however, there was no evidence for genetic moderation for the cross-sectional relationships at age 20, 24, and 29 or for the longitudinal relationships between antisocial peer affiliation at age 17 and adult antisocial behavior at ages 20, 24, and 29 (see Figure 4). Conversely, for substance use disorders, there was evidence of both cross-sectional and longitudinal genetic moderation at each time point. As shown in Figure 3, the genetic moderation results were generally strongest in effect size for the cross-sectional relationship at age 17 (see moderation line in [a]) in comparison to the cross-sectional relationships at ages 20, 24, and 29 (see b–d) or the longitudinal associations (see e–g).

Figure 3. Antisocial peer affiliation moderating genetic and environmental influences substance use disorder (SUD) symptoms: Cross-sectional and longitudinal relationships. Changes in the unstandardized ACE variance components of SUD symptoms are given as a function of antisocial for the best fitting models (see Table 4). The *y*-axis represents the unstandardized variance component score (shown for A, C, and E). The *x*-axis represents the value of antisocial peer affiliation (shown in 0, ±1, and ±2 *SD*). A, additive genetic influence; C, shared environmental influences; E, nonshared environmental influence. All composites were adjusted for age, sex, Age × Sex, and age^2 by regressing these covariates out prior to analysis. Presented are the (a) results for cross-sectional associations at age 17, (b) results for cross-sectional associations at age 20, (c) results for the cross-sectional association at age 24, (d) the cross-sectional associations at age 29, (e) results for the longitudinal association between adolescent peer affiliation (age 17) and early adult substance use disorder (age 20), (f) interaction results for the longitudinal association between adolescent peer affiliation (age 17) and late early adult substance use disorder (age 24), and (g) interaction results for the longitudinal association between adolescent peer affiliation (age 17) and young adult substance use disorder (age 29).

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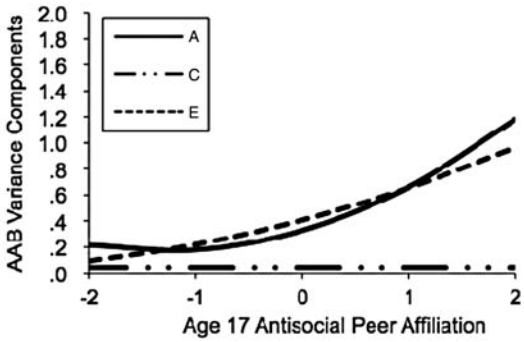
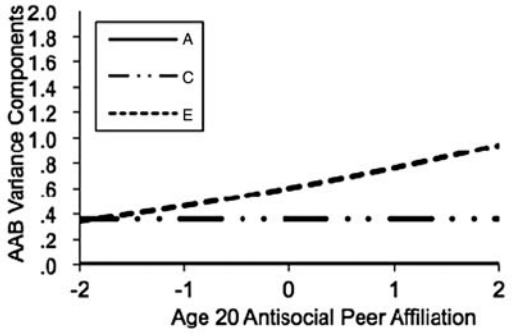
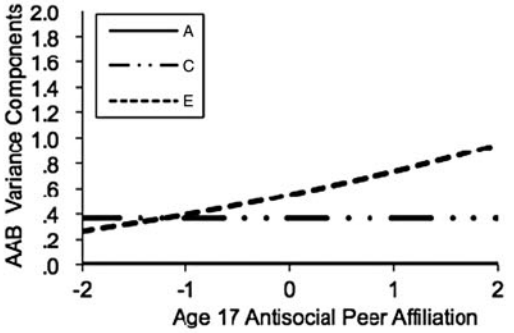
Cross-Sectional Relationships **Longitudinal Relationships**



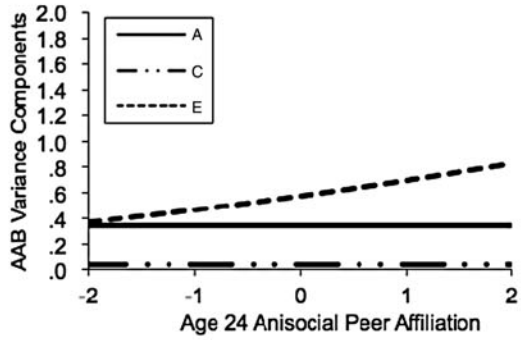
(a) Antisocial peer Affiliation at age 17 moderating genetic and environmental influences on AAB symptoms at age 17



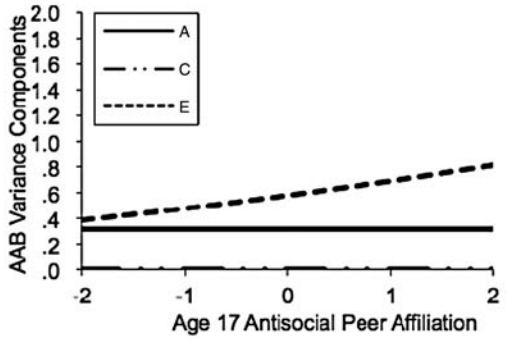
(b) Antisocial peer affiliation at age 20 moderating genetic and environmental influences on AAB symptoms at age 20



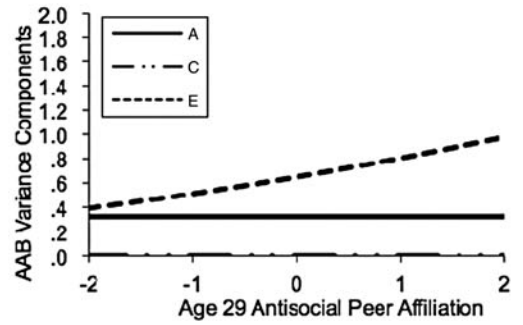
(e) Antisocial peer affiliation at age 17 moderating genetic and environmental influences on AAB symptoms at age 20



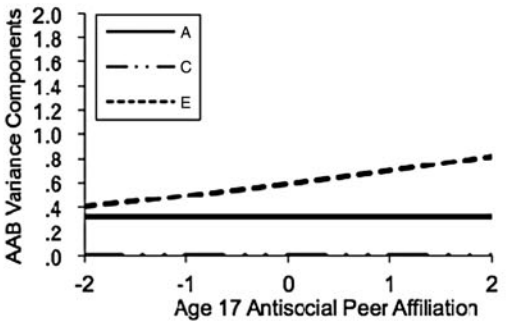
(c) Antisocial peer affiliation at age 24 moderating genetic and environmental influences on AAB symptoms at age 24



(f) Antisocial peer affiliation at age 17 moderating genetic and environmental influences on AAB symptoms at age 24



(d) Antisocial peer affiliation at age 29 moderating genetic and environmental influences on AAB symptoms at age 29



(g) Antisocial peer affiliation at age 17 moderating genetic and environmental influences on AAB symptoms at age 29

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Discussion

Previous studies have shown evidence of both gene–environment correlation and interaction involving child and adolescent environmental contexts and adolescent externalizing problems (Benner et al., 2014; Byrd & Manuck, 2014; Cicchetti et al., 2012; Cleveland et al., 2005; Feinberg et al., 2007; Hicks et al., 2009), but fewer studies have evaluated whether these processes are specific to adolescence or are also present in young adulthood. Here, we extended these findings by examining the consistency of Gene × Environment interplay involving antisocial peer affiliation and externalizing disorders across adolescence (age 17), early adulthood (ages 20 and 24), and later adulthood (age 29).

It is important to point out that overall genetic and environmental influences on externalizing disorders are generally comparable to earlier studies (Bergen, Gardner, & Kendler, 2007; Rhee & Waldman, 2002). We showed that the heritability estimate of externalizing disorders was generally stable from ages 17 to 29 (range = ~45%–55%), although in that same age period, shared environmental influences decreased and nonshared environmental influences increased. A similar pattern was found for antisocial peer influences across time, although shared environmental influences remained significant and moderate in effect size through age 24. Genetic influences on antisocial peer affiliation were stable and moderate in magnitude from age 17 to 24 (estimates arranged from ~20% to 30%) and substantial by age 29 (51%). This is somewhat different than earlier studies (Kendler et al., 2007), which reported substantial heritability estimates (~50%) from ages 12 to 25. Differences may be due to sample characteristics. For example, Kendler et al. used males only and retrospective report whereas we used equivalent numbers of males and females and prospective reports. Nonetheless, the results across both the Kendler et al. study and this study show nearly half the total variance in antisocial peer affiliation by age 29 is accounted for by additive genetic factors.

More important, and following previous research on parenting (Samek, Hicks, et al., 2015) and school factors (Johnson et al., 2009), we failed to detect any meaningful genetic moderation as a function of greater antisocial peer affiliation beyond age 17. This was true for our evaluation of the externalizing disorder composite and in subsequent evaluations of antisocial behavior alone. However, for symptoms of substance use disorders, we found evidence of genetic moderation as a function of greater antisocial peer affiliation from ages 17 through 29. The largest effect sizes for genetic moderation of substance use disorders were found at age 17, with comparatively smaller but nonetheless significant moderating genetic influences at ages 20, 24, and 29. Thus, results support the notion that antisocial peers continue to have a moderating influence on genetic and environmental risk for substance use disorders through young adulthood; perhaps because substance use disorders are more common in early and later adulthood relative to antisocial behavior (e.g., Moffitt, 1993; Substance Abuse and Mental Health Administration, 2013), exposure to antisocial peers continues to have an important socializing effect.

These results have a number of important implications. The first is that, given the differences in findings by substance use disorder in comparison to adult antisocial behavior alone, it remains important for future research to evaluate individual facets of externalizing disorders in addition to a conglomerate externalizing measure. Our findings suggest this may be especially true for analyses involving externalizing disorders in adulthood. The second is that greater expression of genetic risk for externalizing disorders as a function of greater antisocial peer affiliation seems to be particularly important in late adolescence. This is consistent with a previous study by Kendler et al. (2011) that used retrospective reports of alcohol use and several environmental variables and detected a similar Gene × Environment interaction in early and middle adolescence, but not in adulthood. Here, we confirmed what can be described as a Gene × Environment × Development interaction effect between externalizing disorders (particularly the adult antisocial symptom criteria of adult antisocial personality disorder) and antisocial peer affiliation using a longitudinal design. This finding is consistent across other environmental variables; that is, other analyses using this sample have observed the same pattern for parent-relationship quality (Samek, Hicks, et al., 2015), academic engagement (Johnson et al., 2009), and prosocial peer affiliation (Samek, Hicks, Keyes, Iacono, & McGue, 2014). These findings suggest that adolescence may be a critical period for the emergence of externalizing disorders, when exposure to environmental risk factors seems to potentiate what may have been unexpressed genetic risk for externalizing psychopathology.

Following adolescence, gene–environment correlation or common genetic influences primarily account for the association between externalizing disorders and antisocial peer affiliation, as well as several other environmental risk factors

Figure 4. Antisocial peer affiliation moderating genetic and environmental influences on adult antisocial behavior symptoms (AABs): cross-sectional and longitudinal relationships. Changes in the unstandardized ACE variance components of AABs are given as a function of antisocial for the best fitting models (see Table 4). A, additive genetic influence; C, shared environmental influences; E, nonshared environmental influence. The *y*-axis represents the unstandardized variance component score (shown for A, C, and E). The *x*-axis represents the value of antisocial peer affiliation (shown in 0, ±1, and ±2 *SD*). All composites were adjusted for age, sex, Age × Sex, and age^2 by regressing these covariates out prior to analysis. Presented are the (a) results for cross-sectional associations at age 17, (b) results for cross-sectional associations at age 20, (c) results for cross-sectional association at age 24, (d) cross-sectional associations at age 29, (e) results for the longitudinal association between adolescent peer affiliation (age 17) and early adult antisocial behavior symptoms (age 20), (f) interaction results for the longitudinal association between adolescent peer affiliation (age 17) and late early adult antisocial behavior symptoms (age 24), and (g) the interaction results for the longitudinal association between adolescent peer affiliation (age 17) and young adult antisocial behavior symptoms (age 29).

https://doi.org/10.1017/S0954579416000109 Published online by Cambridge University Press

Table 6. *Fit statistics for gene-environment interplay models of antisocial peer affiliation and externalizing disorders, shown separately for substance use disorder and adult antisocial behavior symptoms at ages 17, 20, 24, and 29*

	−2LL	*df*	$\Delta\chi^2$	Δdf	*p*	AIC	Adj. BIC	DIC
Cross-Sectional Associations in Adolescence								
Antisocial Peer Affiliation at Age 17 and Substance Use Disorder Symptoms at Age 17								
1. Full ACE moderation	7653.94	3371				911.94	−2183.53	−4438.42
2. No ACE moderation	8055.95	3377	402.01	6	<.001	1301.95	−1993.22	−4252.13
3. Best fitting model: unique AE and common A moderation only; c22 parameter dropped	7659.64	3375	5.7	4	.22	909.64	−2187.82	−4445.38
Antisocial Peer Affiliation at Age 17 and Adult Antisocial Behavior Symptoms at Age 17								
4. Full ACE moderation	7948.00	3366				1216.00	−2027.59	−4279.14
5. No ACE moderation	8214.36	3372	266.64	6	<.001	1470.64	−1904.96	−4160.53
6. Best fitting model: common A, unique AE moderation only; c22 parameter dropped	7949.01	3370	1.01	4	.91	1209.01	−2034.22	−4288.44
Cross-Sectional Associations in Adulthood								
Antisocial Peer Affiliation at Age 20 and Substance Use Disorder Symptoms at Age 20								
7. Full ACE moderation	10914.23	4398				21188.23	−2966.05	−5909.12
8. No ACE moderation	11117.86	4404	203.63	6	<.001	2309.86	−2875.73	−5822.81
9. Best fitting model: unique A, common A moderation only	10917.06	4402	2.83	4	.57	2113.06	−2972.29	−5918.04
Antisocial Peer Affiliation at Age 20 and Adult Antisocial Behavior Symptoms at Age 20								
10. Full ACE moderation	11389.09	4396.00				2597.09	−2724.79	−5666.52
11. No ACE moderation	11438.70	4402.00	49.61	6	<.001	2634.70	−2711.47	−5657.22
12. Best fitting model: unique E moderation only, parameters a21, a22, c22 dropped	11399.55	4404	10.46	8	.23	2591.55	−2734.88	−5681.96
Antisocial Peer Affiliation at Age 24 and Substance Use Disorder Symptoms at Age 24								
13. Full ACE moderation	10500.13	4195				2110.13	−2685.29	−5492.33
14. No ACE moderation	10666.12	4201	165.99	6	<.001	2264.12	−2613.64	−5424.70
15. Best fitting model: unique E and common A moderation only, parameters c21, c22, and a22 dropped	10505.34	4202	5.21	7	.63	2101.34	−2695.92	−5507.65
Antisocial Peer Affiliation at Age 24 and Adult Antisocial Behavior Symptoms at Age 24								
16. Full ACE moderation	11041.08	4192				2657.08	−2409.14	−5214.17
17. No ACE moderation	11075.09	4198	34.01	6	<.001	2679.09	−2403.48	−5212.53
18. Best fitting model: unique E moderation only, parameters c22, a22 dropped	11053.38	4199	12.30	7	.09	5655.38	−2416.23	−5225.95
Antisocial Peer Affiliation at Age 29 and Substance Use Disorder Symptoms at Age 29								
19. Full ACE moderation	11628.59	4563				2502.59	−3007.93	−6061.56
20. No ACE moderation	11856.78	4569	228.19	6	<.001	2718.78	−2905.43	−5963.08
21. Best fitting model: unique ACE and common E moderation only, drop c22	11630.91	4566	2.32	3	.51	2498.91	−3012.57	−6068.21

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Table 6 (*cont.*)

	−2LL	*df*	$\Delta\chi^2$	Δdf	*p*	AIC	Adj. BIC	DIC
Antisocial Peer Affiliation at Age 29 and Adult Antisocial Behavior Symptoms at Age 29								
22. Full ACE moderation	12192.6	4563				3066.6	−2725.92	−5779.55
23. No ACE moderation	12233.83	4569	41.23	6	<.001	3095.83	−2716.91	−5774.56
24. Best fitting model: unique E moderation only, parameters c11, c21, and c22 dropped	12195.84	4571	3.24	8	.92	3053.84	−2739.77	−5798.76
Longitudinal Associations: Adolescent Peer Affiliation and Adult EXT								
Antisocial Peer Affiliation at Age 17 and Substance Use Disorders Symptoms at Age 20								
25. Full ACE moderation	7858.32	3274				1310.32	−1908.39	−4098.40
26. No ACE moderation	8013.06	3280	154.74	6	<.001	1453.06	−1841.72	−4035.74
27. Best fitting model: unique ACE moderation only, parameter c22 dropped	7861.92	3278	3.6	4	.46	1305.92	−1913.72	−4106.41
Antisocial Peer Affiliation at Age 17 and Adult Antisocial Behavior Symptoms at Age 20								
28. Full ACE moderation	8208.2	3259				1690.2	−1706.71	−3886.68
29. No ACE moderation	8262.02	3265	53.82	6	<.001	1732.02	−1690.49	−3874.48
30. Best fitting model: unique E moderation only, parameters a21, a22, and c22 dropped	8217.33	3267	9.13	8	.33	1683.33	−1716.41	−3901.73
Antisocial Peer Affiliation at Age 17 and Substance Use Disorder Symptoms at Age 24								
31. Full ACE moderation	8089.51	3262				1565.51	−1771.40	−3953.38
32. No ACE moderation	8158.06	3268	68.55	6	<.001	1622.06	−1747.82	−3933.82
33. Best fitting model: unique AE moderation only, parameter c22 dropped	8094.73	3267	5.22	5	.39	1560.73	−1777.71	−3963.03
Antisocial Peer Affiliation at Age 17 and Adult Antisocial Behavior Symptoms at Age 24								
34. Full ACE moderation	8304.24	3240				1824.24	−1624.81	−3792.08
35. No ACE moderation	8330.09	3246	25.85	6	<.001	1838.09	−1622.58	−3793.86
36. Best fitting model: unique E moderation only, parameters a22, c21, c22, and e21 dropped	8319.54	3249	15.3	9	.08	1821.12	−1633.2	−3806.49
Antisocial Peer Affiliation at Age 17 and Substance Use Disorder Symptoms at Age 29								
37. Full ACE moderation	8025.62	3254				1517.62	−1789.08	−3965.71
38. No ACE moderation	8123.69	3260	98.07	6	<.001	1603.69	−1750.45	−3931.39
39. Best fitting model: unique AE moderation only, parameters a21 and c22 dropped	8031.63	3260	6.01	6	.42	1511.63	−1796.77	−3977.42
Antisocial Peer Affiliation at Age 17 and Adult Antisocial Behavior Symptoms at Age 29								
40. Full ACE moderation	8441.56	3254				1933.56	−1581.11	−3757.74
41. No ACE moderation	8461.06	3260	19.5	6	.003	1941.06	−1582.06	−3762.71
42. Best fitting model: unique E moderation only; parameters c21, c22, and e21 dropped	8448.57	3262	7.01	8	.54	1924.57	−1591.87	−3773.85

Note: –2LL, –2 log likelihood; $\Delta\chi^2$, chi-square change; AIC, Akaike information criterion; BIC, Bayesian information criterion; DIC deviance information criterion; A, additive genetic effects; C, shared environmental effects; E, nonshared environmental effects; EXT, externalizing disorders. The baseline model of comparison used in chi-square difference tests is the full ACE moderation model, which allows for ACE moderation on all common and unique parameters. The $\Delta\chi^2$ is the difference between the –2LL in the baseline model (full ACE moderation) compared to the other modes tested. In all best-fitting models, all parameters were significantly different from zero.

https://doi.org/10.1017/S0954579416000109 Published online by Cambridge University Press

(Johnson et al., 2009; Samek, Hicks, et al., 2015). That is, once genetic risk factors have been triggered by exposure to environmental risk, selection effects (i.e., active gene–environment correlation) appear to help maintain the mutual stability of externalizing disorders and high-risk environmental contexts. In addition, for substance use disorders but not adult antisocial behavior, we continued to detect a small Gene × Environment interaction of greater additive genetic and nonshared environmental variance as a function of greater antisocial peer affiliation. This suggests antisocial peers continue to have a potentiating influence on genetic and environmental risk for substance use disorders through young adulthood.

This study had several important limitations. One is that the sample had little racial and ethnic diversity. In addition, it is unclear if these results would replicate in more extreme samples (e.g., clinical or "at-risk" samples), although we might expect gene–environment interaction to be even more relevant to those experiencing extreme environmental adversity in adolescence, given our results on Gene × Environment interaction in adolescence. Another limitation is that the age ranges and environmental moderators we examined also limit the scope of our interpretations. For example, selection effects or gene–environment correlations and Gene × Environment interactions are likely to be present prior to age 17, and so it will be important to examine the stability of gene–environment interplay between externalizing disorders and contextual risk from childhood through adolescence. Relevant to this, prior research has shown that peer influences may be more critical in early or middle adolescence relative to later adolescence (Gardner & Steinberg, 2005; Steinberg & Monahan, 2007; Sumter et al., 2009); thus, it could be Gene × Environment interaction is even more critical in earlier adolescence relative to later adolescence. This remains to be tested because we evaluated only one time point in late adolescence here.

Further, we relied on a self-report assessment of antisocial peers. Prior research has demonstrated differences in genetic and environmental influence on peer measures depending on the method employed to assess peer deviance. For example, Bullock et al. (2006) showed that coder impressions of deviant friendship processes tended to be influenced more by shared environmental rather than by heritable influences, while teacher-reported peer deviance tended to be influenced more by heritable influences than by shared environmental influences. It remains important for future research to explore whether findings may be impacted by other measurement methods, including direct observation. Finally, environmental variables other than peers (e.g., characteristics of romantic partners, marriage, and parenthood) may be important moderators of genetic risk for adult externalizing psychopathology, and this area remains important for future research to address. Strengths of the study include the large sample with equivalent numbers of males and females, prospective design and analysis, and inclusion of diagnostic measurement of externalizing symptoms.

In total, the results show that it is necessary to investigate gene–environment interplay broadly across development, because what may seem like a ubiquitous effect at one time point (e.g., adolescence) may operate differently at other developmental periods and across different (but highly correlated) traits. While the evidence is becoming more convincing, it remains necessary to evaluate other, possibly more salient adult environmental contexts as moderators of genetic risk of adult externalizing disorders (e.g., romantic partner relationship characteristics) to better understand the etiology of externalizing psychopathology across development.

Supplementary Material

To view the supplementary material for this article, please visit http://dx.doi.org/10.1017/S095457941600010.

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