Published in final edited form as:
J Crim Justice. 2013 ; 41(5): . doi:10.1016/j.jcrimjus.2013.06.014.

Aggression and Rule-breaking: Heritability and stability of antisocial behavior problems in childhood and adolescence

Sharon Niv[1], **Catherine Tuvblad**[1], **Adrian Raine**[2], and **Laura A. Baker**[1]

[1]Department of Psychology, University of Southern California

[2]Departments of Criminology, Psychiatry and Psychology, University of Pennsylvania

Abstract

Purpose—This twin study examined the structure of genetic and environmental influences on aggression and rule-breaking in order to examine change and stability across the span of childhood to mid-adolescence.

Methods—Behavioral assessments were conducted at two time points: age 9–10 years and 14–15 years. Using behavioral genetics biometric modeling, the longitudinal structure of influences was investigated.

Results—Aggression and rule-breaking were found to be influenced by a latent common factor of antisocial behavior (ASB) within each wave of data collection. The childhood-age common factor of ASB was influenced by 41% genetics, 40% shared environment and 19% nonshared environment. In adolescence, 41% of influences on the common factor were novel and entirely genetic, while the remainder of influences were stable across time. Additionally, both aggression and rule-breaking within each wave were found to have unique influences not common across subscales or across waves, highlighting specificity of influences on different problem behaviors at both ages.

Conclusions—This research sheds light on the commonality of influences on etiology of different forms of antisocial behavior, and suggests future directions for research into intervention for antisocial behavior problems in youth, such as investigation of adolescence-specific environmental influences on the development of antisocial behavior problems.

Introduction

Concern about violence and crime within society is pervasive, as these forms of behavioral problems encompass broad antisocial behavior ranging from crime to drug use, homicide to risky sexual behavior. These are broadly referred to as antisocial behavior problems, and are arguably a problem for society on the whole; high rates of crime, drug use, gang warfare, or risky sexual activity pose risks for the safety and well-being for all members of society, not only those who propagate such behavior. Hence, considerable research is aimed at understanding the etiology of this behavior in order to better prevent and treat it. However,

Correspondence to: Sharon Niv, University of Southern California, 3620 S. McClintock Ave. Department of Psychology (SGM 501), Los Angeles, CA 90089-1061 USA, Fax: 213-746 9082, Phone number: 213-821-2859, sniv@usc.edu.
Address reprint requests to: Sharon Niv, University of Southern California, 3620 S. McClintock Ave., Department of Psychology (SGM 501), Los Angeles, CA 90089-1061 USA, Fax: 213-746 9082, Phone number: 213-740 2261, sniv@usc.ed

within the broad category of antisocial behavior problems, the question of aggressive behavior versus rule-breaking behavior (rule-breaking) is an important one. Also referred to as overt (aggressive) and covert (nonaggressive, delinquency or rule-breaking) in past work (Loever & Hay, 1997), these patterns of behavior are correlated and are known to co-occur at rates higher than would be expected by chance (Eley, et al. 1999). However, evidence has also emerged to support that these are distinct forms of behavior that should be considered separately when studying antisocial behavior.

For example, different developmental trajectories have emerged for these sets of behaviors, highlighting their distinctness. Lack of control in children of ages 3–5 years was found to predict aggression but not rule-breaking in adolescence, suggesting that rule-breaking was more influenced by peers and aggression more innate (Caspi et al. 1995). Rule-breaking also tends to emerge later developmentally than aggression, and is considered by some researchers to be less lifetime persistent and more likely to be adolescence-specific, although both sets of behaviors increase during the period of adolescence (Moffitt, 1993).

Over the course of adolescence, antisocial behavior problems may increase in inherent risk. Whereas younger children are unlikely to engage in significant substance use behavior, to commit serious crimes, or to behave sexually, adolescents prone to antisocial behavior problems may become involved in these forms of activities. Additionally, forms of antisocial behavior in youth are strongly predictive of adult criminality and antisocial behavior (Loeber & Dishion, 1983), and hence it is important to investigate how influences on aggression and rule-breaking change over the course of childhood to adolescence. Additionally important to consider are sex differences - while males have consistently been found to show higher prevalence (and mean levels) of antisocial behavior, disagreements exist in the literature about whether the genetic and environmental influences on antisocial behavior are equal between the sexes: some meta-analyses conclude equal genetic influence between the sexes (Rhee & Waldman, 2002), and some find higher heritability in males (Miles & Carey, 1997). Further clarifying this discrepancy is an aim of this study.

Antisocial behavior has been found to be heritable in past research. As measured by the Child Behavior Checklist (CBCL), the instrument used in this study, heritability estimates on antisocial behavior problems range from 38–56% (Gjone & Stevenson, 1997; Pesenti-Gritti et al., 2005). Specifically, aggression has been estimated to be heritable at a rate of 38–50% and rule-breaking at 24–40% (Rodgers, et al. 2001). Two recent meta-analysis reviews approached the questions of the distinctions and shared etiologies of aggression and rule-breaking. The first of these concluded that aggression showed higher heritability than rule-breaking, at 65% and 48%, respectively (Burt, 2009). Rule-breaking was also found to be influenced by shared environmental factors, at 18%, further highlighting etiological distinctions between the two forms of antisocial behavior. The second meta-analysis examined covariation between aggression and rule-breaking, and found that 38.4% of genetic influences on these forms of antisocial behavior were shared, whereas the rest of the genetic variance was unique to each (Burt, 2012). In addition, only 10.2% of shared environmental influences were common to both forms of behavior.

At least one previous study has attempted to examine the aggression and rule-breaking subscales of the CBCL longitudinally in late childhood and mid-adolescence, similar to the present research. Correlated genetic factors were found among all four subscales (both subscales at both time points), as well as stronger genetic stability to aggression than to rule-breaking (Eley, et al. 2003). However, this study did not investigate different potential structures to explain influences on the data, such as latent pathways using sophisticated structural equation modeling, which is a strength of the present paper. It is important to discern the structure of influences in order to understand their relationships more precisely

and guide future research into molecular genetics or environmental conditions relevant to etiology.

In general, there have been more longitudinal examinations of aggression or widely-defined antisocial behavior than of rule-breaking on its own. In young childhood, over the ages of 3–7 years, genetics and shared home environment were found to contribute to stability in overall antisocial behavior (van der Valk, et al. 2003). Stability in maternal ratings of aggression across the age span of 3–12 years was 65% accounted for by genetic factors and 25% by shared environmental factors. However, sex differences emerged with genetics as more influential on stability in males and shared environment in females (van Beijsterveldt, et al. 2003).

The purpose of this study is threefold: 1. To examine the structure of genetic and environmental influences on rule-breaking and aggression in order to both examine the nature of etiology and the manner in which influences take effect; 2. To investigate the longitudinal stability and change of influences on aggression, rule-breaking and the covariation between them from late childhood to mid-adolescence; 3. To examine sex differences in these relationships in order to determine whether male and female antisocial behavior should be approached differently.

Methods

Participants

This study uses data collected through the University of Southern California (USC) Risk Factors for Antisocial Behavior (RFAB) twin study, a longitudinal study of over 750 participating families from the greater Los Angeles area. Currently, in its fifth wave of data collection, this study has followed the twins from the age of 9–10 years to their present age of 19–20 years, and concentrates on biological and environmental risk factors for antisocial behavior. The sample is both ethnically diverse and representative of the Los Angeles population breakdown (44% Hispanic, 25% Caucasian, 16% African American, 3% Asian, and 12% mixed or other). Attrition analysis conducted with this sample found no demographic predictors of study continuation. To account for non-returning families from Wave 1, new families were recruited in the third wave of data collection. The analyses in this study utilize data collected in the first and third waves of collection, at which times the twins were 9–10 and 14–15 years of age, respectively. This study uses data from 1204 individuals (269 MZ male, 288 MZ female, 170 DZ male, 184 DZ female, 293 DZ opposite sex) in Wave 1 and 1148 individuals in Wave 3 (249 MZ male, 229 MZ female, 175 DZ male, 212 DZ female, 283 ZD opposite sex). In this study, 73% of Wave 1 families had also participated in Wave 3. Regression analysis found that scores on neither scale were predictive of discontinuation. For full description of the project including zygosity determination, see (Baker et al., 2013; Baker, et al. 2006).

Procedure

The testing protocol was 6–8 hours long in Wave 1, and 4–6 hours long in Wave 3. The twins participated in clinical interviewing and neurocognitive testing, and also psychophysiological testing. Their accompanying parent (>90% biological mothers) participated in daylong clinical interviewing and questionnaire answering aimed at assessing home and school environment, behavior, personality, and psychopathology of both twins as well as of the parent. A portion of families in Wave 3 participated via mail (N=135), phone (N=15) or internet surveys (N=63), while the majority participated in laboratory visits. An analysis of variance found no significant differences in CBCL scores for different participation types.

Measures

CBCL—The CBCL is a widely used caregiver-response instrument for research and clinical work. It measures a wide range of behavior problems in children, both internalizing (consisting of scales for depression, anxiety, and social withdrawal) and antisocial (aggression, rule-breaking) behavior problems (Achenbach, 1991). This instrument has 113 items that use a three-point scale (0 for not true, 1 for sometimes true, and 2 for very or often true). Parents are asked to consider their child's behavior over the last six months. The Rule-breaking subscale of the CBCL (20 items) examines such behavior tendencies as lying, stealing, and destroying possessions. Internal consistencies of the rule-breaking scale were 0.88 and 0.89 at Waves 1 and 3, respectively. The Aggression subscale of the CBCL consists of 13 items and includes behaviors such as arguing, fighting with other children, and bullying others. The internal consistencies in Waves 1 and 3 were found to be 0.61 and 0.71, respectively. This study's internal consistencies of the CBCL antisocial total subscale, which combines the 33 items from the Aggression and Rule-breaking subscales, – 0.88 and 0.91 for Waves 1 and 3, respectively – are consistent with estimates from past research (Pesentti-Gritt, 2005; Arseneault, et al. 2003).

Descriptive Statistics and Correlations

Descriptive statistics and phenotypic analyses were conducted with untransformed raw data. In twin analysis, intrapair twin correlations (correlations between the two twins) can serve as a first step to investigate heritability effects on any given observable. Then MZ correlations are approximately twice as large as DZ correlations, it provides evidence of additive genetic effects (A); when MZ correlations are less than twice DZ correlations, it provides evidence for shared environmental influence (C); other variance is accounted for by nonshared environment (E) (Neale & Cardon, 1992). To achieve more accurate estimates of the proportions of these influences, more sophisticated genetic modeling is employed.

Genetic Model Analyses

Genetic modeling was performed with the software package Mx (Neale, et al. 2003). This program determines model fit by comparing observed and expected values in each model and yielding a likelihood ratio statistic (−2LL) on this basis. Fit is assessed with a log-likelihood ratio test statistic, which compares the difference between −2LLs of models, yielding a χ^2 value the distribution for which has degrees of freedom (df) equal to the difference between df of the two models. Model fit was further assessed using two additional fit statistics, the Akaike Information Criterion (AIC) and the Bayesian Information Criterion (BIC). Lower AIC and BIC values are indicative of more parsimonious explanations of the data.

Prior to genetic modeling, aggressive and nonaggressive ASB scores were transformed using Blom normalization (Blom, 1958). This was performed using a pooled mean and standard deviation from both waves simultaneously, in order to retain information about mean changes and variance across waves. Each variable's heritability on its own was initially estimated using univariate genetic models that estimate A, C, and E individually without examining common influences with any other variable. Models included: 1. ACE (estimates all three types of influences on the measure); 2. AE (examines only genetic and non-shared environmental influences); 3. CE (examines only shared and non-shared environmental influences); 4. and E (examines only non-shared environmental influences). These, as well as all multivariate models, were compared to saturated models that freely estimate means and variances across zygosity groups.

Next, a series of multivariate models were employed to determine the structure of influences on the two waves of data. The first model was an ACE model utilizing Cholesky-

decomposition. The Cholesky decomposition estimates A, C, and E on each variable separately as well as influences of each type shared between variable by partitioning shared variance into genetic and environmental components at different times and on different subscales. This model uses cross-twin cross-trait covariances in modeling how the twins correlate with one another on a given trait as opposed to across traits. Hence, the model estimates how much of each type of influence is unique to each variable as opposed to common between variables. The next examined model is an independent pathway model, which supposes one source of A, C, and E influences that affect all variables. In addition, each variable may be influenced by specific a, c, and e that do not influence other variables. Next, three common pathway models were examined, in which latent variables are estimated through which the A, C and E influences exert effect. The first of these, modeled one latent factor that loads onto all four subscales (i.e., aggression waves 1 and 3, rule-breaking behavior waves 1 and 3) simultaneously. Next, two models with two latent variables were modeled. In the first, two different latent factors were modeled – one that loads onto Rule-breaking behavior in both waves, and one that loads onto Aggression in both waves. In the second, one latent factor loaded onto both subscales in Wave 1, and one loaded onto both subscales in Wave 3.

Results

Descriptive Statistics

Means, standard deviations, and Ns are presented in Table 1, by sex, for both CBCL subscales at each wave. Significant sex differences emerged with males showing higher Aggression in both Waves 1 and 3 ($t=4.69$ $df=1211$, $p<0.01$; $t=2.25$ $df=1151$, $p=0.02$) and males showing higher Rule-breaking in Wave 1 ($t=4.69$ $df=1151$, $p<0.01$). Mixed model analyses of variance (ANOVA) using Time as a repeated measure and Sex as a between group effect found that Time significantly affected both CBCL subscales, with Rule-breaking significantly increasing and Aggression significantly decreasing between childhood and mid-adolescence. However, interactions between Time and Sex were not significant, suggesting that developmental trajectories did not differ between the sexes.

Correlations

Table 2 presents phenotypic correlations among the different measures, separately by sex. Correlations between Aggression and Rule-breaking within wave were approximately $r=0.60–0.70$ for both sexes in both waves. Longitudinal stability of Rule-breaking was $r=0.41$ for females and 0.59 for males, and longitudinal stability of Aggression was $r=0.45$ for females and $r=0.44$ for males.

As presented in Table 3, MZ correlations are higher than DZ correlations for Aggression and Rule-breaking scores in Wave 1, and considerably higher in Wave 3. This increasing difference between MZ and DZ twin similarity across age suggests emergence of additional genetic influence on aggression and rule-breaking in adolescence than in childhood. Univariate genetic analyses conducted that examined each subscale separately within each wave found that the best fitting models equated the influences on the sexes for all but Rule-breaking in Wave 1, in which females showed CE influences and males showed ACE influences. These Univariate results are available in Appendix A.

Table 4 presents model fit statistics for the multivariate genetic models of Aggression and Rule-breaking across both waves of data collection. When compared to the full ACE model using Cholesky decomposition (Model 1), equating influences between the two sexes was found to produce a more parsimonious model (Model 1a) as assessed by AIC and BIC, which is consistent with univariate model results. Hence, all subsequent models (2–5) were

tested with estimates equated between the sexes. Models 1a, 2, 3, 4, and 5 were compared using AIC and BIC values, as well as their relevance to research questions. While the lowest AIC is found in model 1a (ACE with Cholesky decomposition), the lowest BIC was found in model 5 (two factor common pathway, longitudinal), which better examines longitudinal change on the construct of antisocial behavior. In model 5, variances for the Wave 1 and Wave 3 latent factors were freely estmated while loadings were constrained, allowing for the consideration of change in behavior variance across time. When these variances were constrained (model 5a), the model comparison χ^2 difference was significant (χ^2 =21.45, df=2; P<0.01), suggesting that a freely estimated variance provided a better explanation of the data. When loadings were equated across waves (model 5b), this more parsimonious model provided a better fit to the data (χ^2 =0.94, df=1; P=0.33), and the equated loadings constraint was used in models 5c and 5c*, as well. In model 5c, longitudinal influences on the latent factors were equated; that is, the sources of A, C, and E on the Wave 1 latent factor were constrained to be equal to those that stemmed from the same influences but affected the Wave 3 latent factor. In this manner, the influences that emerged exclusively for Wave 3 can be considered entirely new. While this model was not a better fit to the data as assessed by chi-square-based model comparison, it provided stronger answers to questions about longitudinal change and stability of influences on antisocial behavior. Model 5c* uses model 5c as a basis but drops estimates that emerged as not significant (i.e. included zero in the 95% confidence interval), and was selected as the best explanation of the data. In this model, only A effects were found to be significant for new influences in adolescence.

A (additive genetics) accounted for 41% (0.645^2) of influences on the ASB latent factor in Wave 1, while C (shared environment) accounted for 40% (0.635^2) and E (nonshared environment) accounted for 19% (0.44^2) of influences. The Wave 3 latent factor was 42% (0.65^2) influenced by entirely new factors, all of which were additive genetic factors. The other 58% ($0.33^2 + 0.49^2 + 0.48^2$) of influences on the Wave 3 latent factor were stable – that is, shared with those on the Wave 1 latent factor.

In addition to the influences on the subscales from the latent factor, which comprised 61% (0.78^2) of influences on Wave 1 Aggression, 44% (0.66^2) on Wave 1 Rule-breaking, 79% (0.89^2) on Wave 3 Aggression and 55% (0.74^2) of influences on Wave 3 Rule-breaking, each subscale had additional E (10–24%) or A (11–21%) and E influences specific to it only. Notably, the only shared environmental influence on any subscale was common to both Aggression and Rule-breaking at both waves, as no specific C emerged. Rule-breaking was found to have genetic influences not share with Aggression in both waves. This full model is represented in Figure 1, in which rectangular variables were directly observed and circled variables represented latent variables. Confidence intervals are displayed in Figure 1, with the exception of the longitudinal influences, which are the standardized estimates of values constrained to equal longitudinally.

Discussion

The present study sought to examine the structure and longitudinal stability of influences on two prominent and concerning facets of antisocial behavior problems across childhood and adolescence – aggression and rule-breaking – as well as to investigate sex differences on these influences. Our primary finding is that aggression and rule-breaking are both influenced by a common factor of general antisocial behavior, through which they share genetic and environmental influences. Additionally, at both time points, both aggression and rule-breaking had influences that were unique, and not shared between them or across time. Lastly, it emerged that males and females do not significantly differ in these influences, which is consistent with past findings (Burt, 2009; Rhee & Waldman, 2002).

The latent antisocial behavior factor that emerged is a novel finding, as past research that has not investigated this structure of influences has assumed independent pathways for genetic correlation between the two constructs. Although it is intuitive that different forms of antisocial behavior – both aggressive and rule-breaking – may share etiologies, the structure of these etiologies had never been investigated systematically. The estimates of heritability on the latent factor are in line with past meta-analysis findings of shared heritability between aggressive and rule-breaking (Burt, 2012). Past research found genetic correlation between aggression and rule-breaking concurrently and longitudinally, but this finding suggests an additional layer of complexity: that both sets of behaviors stem from a common origin, influenced by some overlapping genetic and environmental influences, that affects both tendencies in unison. In addition, the subscale-and- time-specific influences on either set of behaviors shapes how tendencies develop, ruling out redundancy between them.

It is important that when constraints were applied to the model in order to discern the influences that were entirely novel, the only significant form of influence that emerged was genetic rather than environmental. This is notable, as it is consistent with past findings that change in overall antisocial behavior is attributable to genetic factors activated during puberty (Jacobson et al 2002). These may represent genetic influences activated at the onset of puberty on the development of ASB. The structure of our findings highlights the significance of adolescent development, biologically and socially, on the etiology of antisocial behavior problems.

In our analyses, both aggression and rule-breaking were found to be highly genetically influenced. This is somewhat counter to past research of rule-breaking that found rule-breaking shows lower levels of heritability and higher levels of shared environment than aggression (Burt, 2009). In our sample, MZ correlations for rule-breaking were very high for both sexes, which accounts for the estimates of heritability. In their meta-analysis, Rhee &Waldman suggested that using parental reports may exaggerate estimates of familial influences (genetics and shared environment) because one reporter is reporting on both twins, and may have specific response bias patterns. While this may be a factor in our estimates, past studies also used parental ratings of rule-breaking. One difference between correlations found here and correlations in past studies is that our DZ estimates were lower than those found previously, at 0.72 (Cloninger & Gottesman, 1987). This may suggest differences in samples between our study and others.

Phenotypically, our findings are consistent with past research, which found that rule-breaking significantly increases during adolescence while aggression significantly decreases. Although males showed high levels of aggression in both waves and rule-breaking in Wave 1, there were not significant interactions between time and sex, suggesting trajectories of phenotypic change in behavior across childhood to adolescence were not different between the sexes. Some theories posit that increases in rule-breaking are typically attributed to peer influence during adolescence, and decreases in aggression to the development of the frontal lobes. Past research has found that rates of impulsivity increase over the course of adolescence (Niv, et al. 2011), which may also account for increases in rule-breaking as manifestation of behavioral disinhibition.

While males showed higher mean levels of aggression in both waves and rule-breaking in Wave 1, the best fitting explanation of genetic and environmental influences on the pattern of observed results suggested equality between the sexes. This finding is consistent with some past literature reviews (Widom & Ames, 1988) but inconsistent with other reviews that found higher heritability of aggression in males (Miles & Carey, 1997). It is important to note the polygenic multiple threshold model, as elucidated by Rhee & Waldman (2002), which posits that the less affected sex needs higher liability in order to develop a phenotype,

but not that the magnitude of influences is necessarily higher. That is, while females may need more liability to express antisocial behavior problems, this does not mean that different or more influences are necessarily involved than in males.

These findings shed light on our understanding of etiology and developmental course of aggression and rule-breaking. While these forms of antisocial behavior problems are considered distinct, correlations exist between them both phenotypically and genetically. However, prior to this study, the structure of these influences had not been elucidated. The finding of a latent pathway supports the hypothesis that there exists a common thread between these different forms of antisocial behaviors, and that they are jointly influenced by a set of genetic and environmental circumstances and develop in unison. Differentiation between these behaviors can be attributed to symptom specific influences. Second, longitudinal change in effects on ASB is entirely attributable to genetic influences, suggesting that potential targets for molecular genetic research may be genes related to pubertal development.

There are a few limitations in this study. First, because parental reports were used, it is possible that underreporting is involved, either due to social desirability or to ignorance of children's true behavior, especially during the adolescent age. Lastly, the twin design relies upon several assumptions, such as lack of assortative mating in the parent generation, which may slightly bias estimates (Plomin, 2001). Assortative mating acts to inflate DZ correlations and thereby increase shared environment (Krueger, et al. 1998), suggesting that is possible that part of the shared environment detected in the current study is due to assortative mating. However, DZ correlations were found to be lower than past studies in our sample.

Future directions may include examinations of specific genes as well as environmental conditions leading to the development of aggression and rule-breaking, for the purposes of intervention. Specific genes involved in the etiology of both aggression and rule-breaking may be investigated in both childhood and adolescence, to better understand which genes affect these behaviors at which age, and especially more so in adolescence, when individuals gain more freedom and ability to become involved in dangerous activity. Our findings also suggest that the environmental conditions that influence ASB of both forms are present prior to adolescence, and may be identified early. By identifying the environmental conditions that most contribute to the development of antisocial behavior problems, appropriate clinical interventions can be directed specifically at these conditions, such as parenting, peer-interactions, exposure to violence, and others.

Conclusions

This study examined parent-reported aggression and rule-breaking at late childhood (age 9–10 years) and mid-adolescence (age 14–15 years) using the highly validated instrument of the Child Behavioral Checklist. These subscales were found to share genetic, shared environmental and nonshared environmental influences through latent common factors at each wave. These common factors were highly correlated longitudinally. In addition, however, exclusively genetic influences on this common factor at the second assessment provide explanation for observed changes across development. Influences on each subscale were 44–79% conveyed through this common factor, and the rest of influences through genetic and nonshared environmental influences on each subscale at each time point specifically, further explaining differentiation and change across age.

Acknowledgments

This study was funded by NIMH (R01 MH58354). Adrian Raine was supported by NIMH (Independent Scientist Award K02 MH01114-08). We thank the Southern California Twin Project staff for their assistance in collecting data, and the twins and their families for their participation.

References

Achenbach, TM. Manual for the youth self-report and 1991 profile. University of Vermont, Department of Psychiatry; Burlington: 1991a.

Achenbach, TM. Manual for the Child Behavior Checklist/4–18 and 1991 Profile. Burlington, VT: University of Vermont Department of Psychiatry; 1991b.

Arsenault L, Moffitt TE, Caspi A, Taylor A, Rijsdijk FV, Jaffee SR, Ablow JC, Measelle JR. Strong genetic effects on cross-situational antisocial behaviour among 5-year-old children according to mothers, teachers, examiner-observers, and twin self-reports. Journal of Child Psychology and Psychiatry. 2003; 44:832–848. [PubMed: 12959492]

Baker LA, Barton M, Lozano DI, Raine A, Fowler JH. The Southern California Twin Register at the University of Southern California: II. Twin Research and Human Genetics. 2006; 9:933–40. [PubMed: 17254433]

Baker LA, Barton M, Raine A. The Southern California Twin Register at the University of Southern California. Twin Research and Human Genetics. 2002; 5(5):456–9.

Baker LA, Tuvblad C, Wang P, Gomez K, Bezdjian S, Niv S, Raine A. The Southern California Twin Register at the University of Southern California: III. Twin Research. 2013

van Beijsterveldt CEM, Bartler M, Hudziak JJ, Boomsma DI. Causes of stability of aggression from early childhood to adolescence: A longitudinal genetic analysis in Dutch twins. Behavior Genetics. 2003; 33:591–605. [PubMed: 14574135]

Blom, G. Statistical estimates and transformed beta-variables. New York: John Wiley and Sons; 1958.

Caspi A, Henry B, McGee RO, Moffitt TE, Silva PA. Temperamental origins of child and adolescent behavior problems: from age three to age fifteen. Child Development. 1995; 66:55–68. [PubMed: 7497829]

Cloninger, CR.; Gottesoman, II. Genetic and environmental factors in antisocial behavior disorders. In: Mednick, SA.; Moffitt, TE.; Stack, SA., editors. The causes of crime: New biological approaches. New York: Cambridge University Press; 1987. p. 92-109.

Edelbrock C, Rende RD, Plomin R, Thompson LA. A twin study of competence and problem behavior in childhood and early adolescence. Journal of Child Psychology and Psychiatry. 1995; 36:775–785. [PubMed: 7559844]

Eley TC, Lichtenstein P, Moffitt TE. A longitudinal behavioral genetic analysis of the etiology of aggressive and nonaggressive antisocial behavior. Development and Psychopathology. 2003; 15:383–402. [PubMed: 12931834]

Eley TC, Lichtenstein P, Stevenson J. The Etiology of aggressive and nonaggressive antisocial behavior: Results from two twin studies. Child Development. 1999; 70:155–168. [PubMed: 10191520]

Gjone H, Stevenson J. The association between internalizing and externalizing behavior in childhood and early adolescence: genetic or environmental influences? Journal of Abnormal Child Psychology. 1997; 25:277–286. [PubMed: 9304444]

Jacobson KC, Prescott CA, Kendlar KS. Sex differences in the genetic and environmental influences on the development of antisocial behavior. Developmental Psychopathology. 2002; 14:395–416.

Krueger RF, Moffitt TE, Caspi A, Bleske A, Silva PA. Assortative mating for antisocial behavior: developmental and methodological implications. Behav Genet. 1998; 28:173–186. [PubMed: 9670593]

Loeber R, Dishion TJ. Early predictors of male rule-breaking: A review. Psychological Bulletin. 1983; 94:68–99. [PubMed: 6353467]

Loeber R, Hay DF. Key issues in the development of aggression and violence from early childhood to early adulthood. Annual Review of Psychology. 1997; 48:371–410.

Miles DR, Carey G. Genetic and environmental architecture of human aggression. Journal of Personality and Social Psychology. 1997; 72:207–217. [PubMed: 9008382]

Moffitt TE. Adolescence-limited and life-course persistent antisocial behavior: A developmental taxonomy. Psychological Review. 1993; 100:674–701. [PubMed: 8255953]

Neale, MC.; Boker, SM.; Xie, G.; Maes, H. Mx: Statistical modeling. Richmond, VA: Department of Psychiatry, Medical College of Virginia; 2003.

Neale, MC.; Cardon, LR. Methodology for genetic studies of twins and families. Dordrecht, The Netherlands: Kluwer Academic Publications; 1992.

Niv S, Tuvblad C, Raine A, Baker LA. Heritability and longitudinal stability of impulsivity in adolescence. Behav Genet. 2011; 42:378–392. [PubMed: 22089391]

Pesenti-Gritti P, Spatola CAM, Fagnani C, Patriarca V, Stazi MA, Battaglia M. The co-occurrence between internalizing and externalizing behaviors. A general population twin study. Journal of European Child and Adolescent Psychiatry. 2005; 17:82–92.

Rhee SH, Waldman ID. Genetic and Environmental Influences on Antisocial Behavior: A Meta-Analysis of Twin and Adoption Studies. Psychological Bulletin. 2002; 128:490–529. [PubMed: 12002699]

Rodgers JL, Buster M, Rowe DC. Genetic and environmental influences on rule-breaking: DF analysis of NLSY kinship data. J Quant Criminology. 2001; 17:145–168.

Rowe DC. Biometrical genetic models of self-reported rule-breaking behavior: A twin study. Behavior Genetics. 1983; 13:473–489. [PubMed: 6686763]

Tuvblad C, Eley TC, Lichtenstein P. The development of antisocial behaviour from childhood to adolescence: A longitudinal twin study. European Child & Adolescent Psychiatry. 2005; 14:216–225. [PubMed: 15981133]

Van der Valk JC, van den Oord E, Verhulst FC, Boomsma DI. Genetic and environmental contributions to stability and change in children's internalizing and antisocial problems. J Am Acad Child Adolesc Psychiatry. 2003; 42:1212–1220. [PubMed: 14560171]

Widow, CS.; Ames, A. Biology and female crime. In: Moffitt, TE.; Mednick, SA., editors. Biological contributions to crime causation. Dordrect, the Netherlands: Martinus Nijhoff; 1988. p. 308-331.

Appendix A. Univariate Model Fit Results for Aggressive and Rule-breaking Behavior in Waves 1 and 3

Table A.1

Model Aggression Wave 1

		Overall fit			Model difference test		
		−2LL	**Df**	**AIC**	**χ^2**	**Δdf**	***P***
1	Saturated (means constrained)	3001.79	1183	786.73			
2	ACE	3031.43	1196	639.43	29.64	13	0.01
3	**ACE M=F**	**3031.72**	**1199**	**633.71**	**29.93**	**16**	**0.02**
4	AE M=F	3039.76	1200	639.76	37.97	17	<0.01
5	AE M≠F	3039.38	1198	643.38	37.59	15	<0.01
6	CE M=F	3038.24	1200	638.24	36.45	17	<0.01
7	CE M≠F	3038.14	1198	642.14	36.35	15	0.02
8	E	3193.76	1201	791.76	191.98	18	<0.01

Estimates: A 0.29 (0.07–0.51); C 0.27 (0.09–0.44); E0.44 (0.37–0.52)

Table A.2

Model Rule-breaking Wave 1

		Overall fit			Model difference test		
		−2LL	Df	AIC	χ^2	Δdf	*P*
1	Saturated (means constrained)	2646.51	1183	280.51			
2	ACE	2661.99	1196	269.99	15.48	13	0.28
3	ACE M=F	2680.19	1199	282.19	33.68	16	0.01
4	AE M=F	2684.34	1200	284.34	37.83	17	<0.01
5	AE M≠F	2674.26	1198	278.26	27.75	15	0.02
6	CE M=F	2700.12	1200	300.12	53.61	17	<0.01
7	CE M≠F	2685.62	1198	289.62	39.11	15	<0.01
8	E	2886.13	1201	484.13	239.62	18	<0.01
9	**Male ACE, female CE**	**2662.40**	**1197**	**268.40**	**15.89**	**14**	**0.32**

Estimates: male - A 0.48 (0.30–0.63); C 0.17 (0.06–0.31); E 0.35 (0.27–0.45); female - C 0.62 (0.54–0.69); E:0.38 (0.31–0.46)

Table A.3

Model Aggression Wave 3

		Overall fit			Model difference test		
		−2LL	Df	AIC	χ^2	Δdf	*P*
1	Saturated (means constrained)	2997.05	1127	743.05			
2	ACE	3008.44	1140	728.44	11.39	13	0.58
3	ACE M=F	3011.94	1143	725.94	14.89	16	0.53
4	**AE M=F**	**3012.13**	**1144**	**724.13**	**15.08**	**17**	**0.59**
5	AE M≠F	3008.65	1142	724.65	11.60	15	0.71
6	CE M=F	3045.21	1144	757.21	48.16	17	<0.01
7	CE M≠F	3043.65	1142	759.65	46.60	15	<0.01
8	E	3230.69	1145	913.69	206.64	18	<0.01

Estimates: A 0.68 (0.61–0.73); E 0.32 (0.27–0.39)

Table A.4

Model Rule-break Wave 3

		Overall fit			Model difference test		
		−2LL	Df	AIC	χ^2	Δdf	*P*
1	Saturated (means constrained)	2708.86	1127	454.86			
2	ACE	2720.05	1140	440.05	11.19	13	0.60
3	**ACE M=F**	**2720.35**	**1143**	**434.35**	**11.49**	**16**	**0.78**
4	AE M=F	2732.66	1144	444.66	23.80	17	0.13
5	AE M≠F	2732.52	1142	448.52	23.66	15	0.07

		Overall fit			Model difference test		
		−2LL	**Df**	**AIC**	**χ^2**	**Δdf**	***P***
6	CE M=F	2759.30	1144	471.30	50.44	17	<0.01
7	CE M≠F	2758.59	1142	474.59	49.73	15	<0.01
8	E	3079.11	1145	789.11	360.25	18	<0.01

Highlights

- Aggression and Rule-breaking are influenced jointly by genetics and environment
- At age 9–10, joint effects are 41% genetic, 40% shared environmental, 19% nonshared environmental
- Novel genetic influences emerge in adolescence, possibly in connection to pubertal development

Aggression and Rule-breaking are distinguished by 21–56% novel genetic and environmental effects

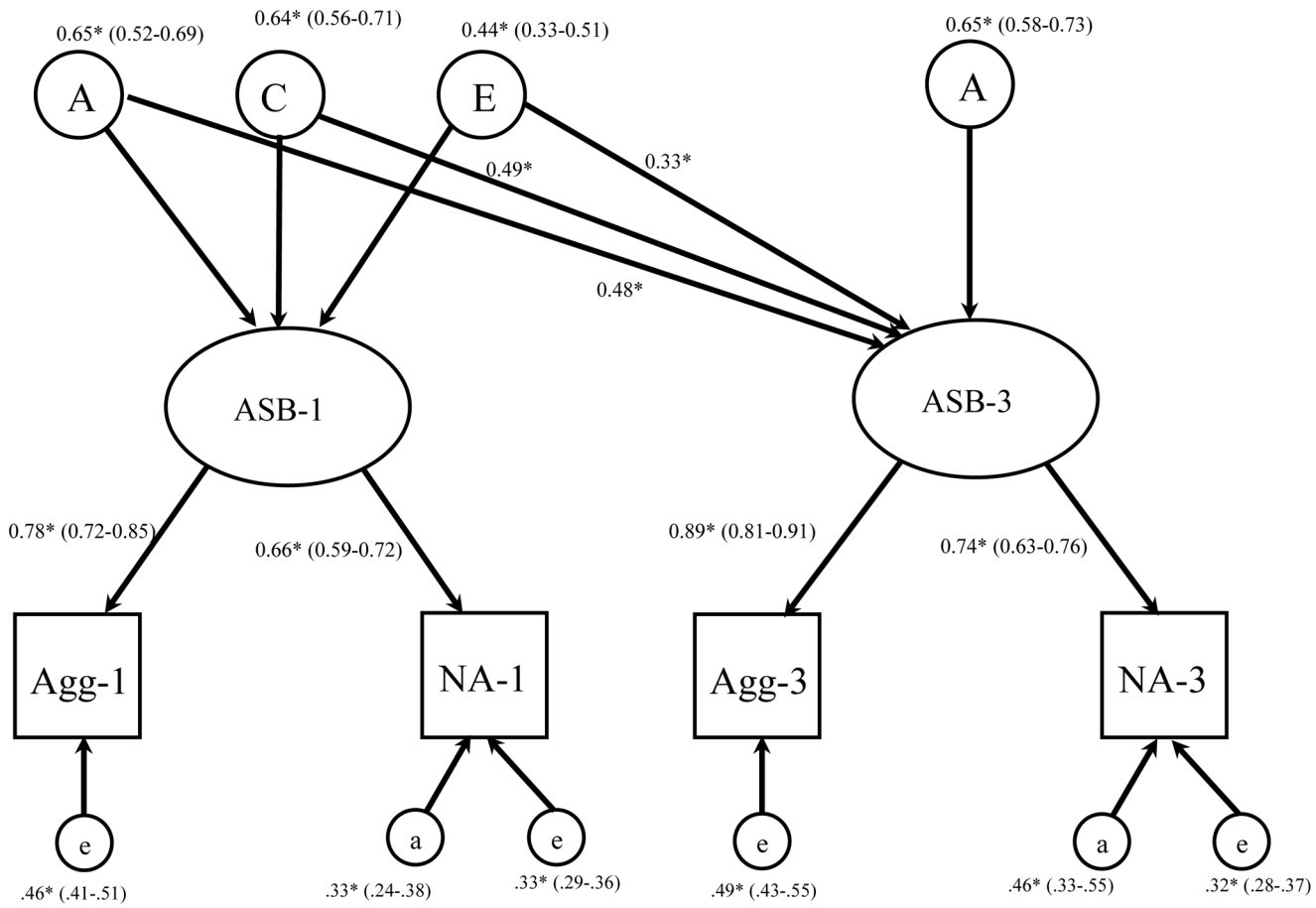

Figure 1. Best-fitting Two-Factor Common Pathway Model of A, C, and E Influences on Aggressive and Rule-breaking Behavior at Ages 9–10 and 14–15 Years

This model shows the latent common factor structure of influences on aggression (Agg) and rule-breaking (RB) behavior problems in Waves 1 and 3. Each Antisocial (AS) latent common factor loads onto the individually measured scales, and is influenced by additive genetic (A), shared environmental (C), and nonshared environmental (E) influences. In addition, each scale shows unique genetic (a) and nonshared environmental (e) influences.

Table 1

Means, Standard Deviations, and Ns for Aggressive and Rule-breaking Behavior by Sex

	Agg-1	**RB-1**	**Agg-3**	**RB-3**
Males	6.36 (5.34) N=586	1.53 (1.85) N=586	5.33 (5.66) N=565	1.69 (2.38) N=565
Females	5.27 (5.19) N=618	1.08 (1.45) N=618	4.60 (5.26) N=583	1.47 (2.52) N=583

Note. Agg-1 = Aggression Wave 1; RB-1 = Rule-breaking Wave 1; Agg-3 = Aggression Wave 3; RB-3 = Rule-breaking Wave 3

Table 2

Phenotypic Correlations among Aggressive and Rule-breaking Behavior Scores by Sex

	Agg-1	**RB-1**	**Agg-3**	**RB-3**
Agg-1	-	.62*	.45*	.35*
RB-1	.62*	-	.29*	.41*
Agg-3	.44*	.40*	-	.69*
RB-3	.38*	.59*	.66*	-

Note. Females are above the diagonal. Agg-1 = Aggression Wave 1; RB-1 = Rule-breaking Wave 1; Agg-3 = Aggression Wave 3; RB-3 = Rule-breaking Wave 3; Starred* values indicated $p<0.05$.

Table 3

Intraclass Correlations for Aggressive and Rule-breaking Behavior, at Ages 9–10 and 14–15 Years

	MZ male	DZ male	MZ female	DZ female	DZ - OS
Agg-1	0.62*	0.38*	0.53*	0.43*	0.41*
RB-1	0.63*	0.42*	0.63*	0.51*	0.30*
Agg-3	0.74*	0.11	0.72*	0.42*	0.52*
RB-3	0.85*	0.39*	0.88*	0.46*	0.56*

Note.

MZ: monozygotic, DZ: dizygotic, DZ-OS: dizygotic opposite sex,

* $p < 0.05$

Table 4

Multivariate Model Fit Indices for Aggressive and Rule-breaking Behavior in Waves 1 (9–10 years) and 3 (14–15 years) Combined

		Overall fit				Compared to model	Model difference test		
Model		**−2LL**	**Df**	**AIC**	**BIC**		**χ^2**	**df**	***P***
0	Full Saturated	10116.16	4516	1084.16	−9958.25				
0a	Equate within sexes	10116.16	4516	1084.16	−9958.25				
1	ACE Cholesky M ≠ F	10297.37	4636	1018.37	−10270.16				
1a	ACE Cholesky M = F	10323.00	4654	1015.00	−10313.70	1	25.63	18	0.11
2	1-Factor IP model M = F	10405.75	4672	1061.75	−10332.18	1a	82.75	18	<0.01
3	1 CP M = F	10537.50	4678	1181.50	−10286.25	1a	206.06	24	<0.01
4	2 CP Agg & Del M = F	10560.02	4675	1210.02	−10265.00	1a	237.02	21	<0.01
5	2 CP Ext1 & Ext3 M=F Variances Freed	10397.55	4673	1051.55	−10339.60	1a	96.00	21	<0.01
5a	2 CP Ext1 & Ext3 M = F variances constrained	10419.00	4675	1069.00	−10335.53	5	21.45	2	<0.01
5b	2 CP Ext1 & Ext3 M=F Variances Freed, loadings equated	10398.49	4674	1050.49	−10342.46	5	0.94	1	0.33
5c	2 CP Ext1 & Ext3 M=F Variances Freed, loadings equated, Equate longitudinal influences on latent factors	10422.15	4677	1068.15	−10340.60	5b	23.66	3	<0.01
5c*	5c with nonsignificant estimates dropped	10437.38	4685	1067.38	−10359.59	5c	15.15	8	0.06