

The Etiology of the Association Between Child Antisocial Behavior and Maternal Negativity Varies Across Aggressive and Non-Aggressive Rule-Breaking Forms of Antisocial Behavior

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Published online: 7 June 2014
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Abstract There is a robust association between negative parenting and child antisocial behavior problems. However, the etiology of this association remains unclear. Extant literature has reported strikingly different conclusions across studies, with some highlighting genetic mediation and others highlighting environmental mediation. One possible reason for these discrepancies across studies may be the failure to differentiate between aggressive and non-aggressive (rule-breaking) dimensions of childhood antisocial behavior, given their notably different etiologies and developmental trajectories (Burt, 2012). The current study sought to examine the phenotypic and etiologic associations of maternal negativity with aggressive and rule-breaking antisocial behavior, respectively. Participants included 824 mothers and their twin children between the ages of 6 and 10. Our results highlighted clear etiologic distinctions in the associations of aggression and rule-breaking with maternal negativity. Aggression was associated with maternal negativity via both genetic and environmental factors, whereas the association between non-aggressive rule-breaking and maternal negativity was entirely environmental in origin. These findings provide additional support for the presence of meaningful distinctions between aggressive and non-aggressive forms of antisocial behavior, and highlight the complex relationship between parenting and child outcome.

Keywords Negative parenting · Antisocial behavior · Aggression · Rule-breaking · Gene-environment correlation

Antisocial behavior in childhood is a costly public health problem that can have serious consequences (prison,

professional underachievement) into adulthood (Alink and Egeland, 2013). Understanding the factors that contribute to antisocial behavior problems in childhood is thus an important goal, in order to inform targeted prevention and intervention strategies. Negative parenting, including verbal criticism, parent–child conflict, and harsh discipline, has been robustly associated with antisocial behavior problems in children and youth (Deater-Deckard and Dodge, 1997; Dishion, Patterson, Stoolmiller, and Skinner, 1991; Kerr, Lopez, Olson, and Sameroff, 2004; Patterson and Fisher, 2002). A recent meta-analysis confirmed the association between negative parenting and delinquency, reporting medium effect sizes for both parental hostility and rejection. In addition, the authors indicated that 67 % of children who experience high levels of negative parenting demonstrate high levels of antisocial behavior, compared to only 33 % of youth who experience low levels of parental negativity (Hoeve et al., 2009). Critically, however, we have relatively little insight into the genetic and/or environmental factors underlying their association (Lysenko, Barker, and Jaffee, 2013). Multiple etiologic pathways have been proposed, with varying degrees of empirical support. These pathways are outlined in more detail below.

Pathway 1: Environmental Mediation

The first possibility is that negative parenting is associated with child antisocial behavior via environmentally-mediated pathways, such that negative parenting causes an increase in children’s antisocial behaviors. Nix and colleagues, for example, found that mother’s harsh discipline of their preschool children predicted children’s externalizing problems up through 3rd grade, even after controlling for the children’s initial level of externalizing problems (Nix et al., 1999). Several twin studies also lend support to the notion that the association between parenting and antisocial behavior is

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shared environmental in origin (Burt, Krueger, McGue, and Iacono, 2003; Burt, McGue, Krueger, and Iacono, 2005). In addition, a handful of adoption studies have implicated environmentally-driven effects (an important set of constructive replications given that the confound of shared genes is entirely circumvented in non-biologically related families; Burt, McGue, Krueger, and Iacono, 2007; Elam et al., 2013; Klahr et al., 2011a; Klahr et al., 2011b). Klahr and colleagues, for example, examined the association between parent–child conflict and conduct problems over time in a longitudinal sample of adoptive families and found that parent–child conflict predicted conduct problems 4 years later, but not the reverse. Such findings are clearly suggestive of an environmental or “causal” association between parenting and child antisocial behavior (Klahr et al., 2011a).

Pathway 2: Genetic Mediation

Prior research has confirmed that both childhood antisocial behavior (Burt, 2009) and negative parenting (Klahr and Burt, 2014) are heritable. Given this, it is possible that common genes account for their association. This genetic overlap could take one (or more) forms. The first of these is passive gene-environment correlation (passive rGE; Plomin, DeFries, and Loehlin, 1977; Scarr and McCartney, 1983), whereby a given set of genes predispose individuals to both negative parenting behaviors and to externalizing problems. More specifically, to the extent that negative parenting is a function of the parent’s own tendency towards externalizing problems, a tendency passed on to the child, the association between negative parenting and child antisocial behavior problems could be a reflection of common genes. Available data has provided some support for passive rGE effects in the association between overall family functioning and child antisocial behavior (Braungart-Rieker et al., 1995; McGue, Sharma, and Benson, 1996). Namely, McGue, et al. (1996) found that the association between family functioning and adolescent externalizing problems was greater in biological than adoptive families. Importantly, however, this study examined overall family functioning rather than parenting; it is thus unclear whether these findings apply to parenting more specifically. Indeed, available studies examining this question indicate that the effects of passive rGE on the association between parenting and child antisocial behavior are small to non-existent (Harold et al., 2013; Marceau et al., 2013; Narusyte et al., 2011b). For example, Marceau and colleagues (2013) modeled passive rGE effects (along with evocative rGE and environmental pathways) underlying the association between negative parenting and adolescent externalizing problems using the Extended Children of Twins model (Narusyte et al., 2008). They found no evidence for passive rGE effects. Rather, their results highlighted the role of evocative rGE, as described below.

Another possible form of genetic mediation is evocative rGE, in which an individual’s genetically-influenced characteristics evoke particular environmental experiences or responses (Plomin et al., 1977; Scarr and McCartney, 1983). In this case, children exhibiting externalizing behavior would elicit negative parenting consistent with their genetic proclivities towards externalizing (Klahr et al., 2013). Consistent with this possibility, research suggests that the characteristics of children are powerful drivers of parenting behavior (Anderson, Lytton, and Romney, 1986; Cunningham and Barkley, 1979; Karraker and Coleman, 2005). Moreover, a number of studies have demonstrated that genetic influences on child behavior problems and parenting at least partially overlap (Alemany, Rijdsdijk, Haworth, Fañanás, and Plomin, 2013; Button, Lau, Maughan, and Eley, 2008; Marceau et al., 2013; Narusyte et al., 2011b), results that are typically interpreted as evidence of evocative rGE. For example, Pike and colleagues examined the etiology of the association between maternal and paternal negativity and child antisocial behavior in a sample of over 700 adolescent sibling pairs (including twins, full siblings, half siblings, and unrelated siblings). Their results suggested that the overlap between parenting and antisocial behavior was primarily mediated by genetic (i.e., evocative rGE) factors (Pike, McGuire, Hetherington, Reiss, and Plomin, 1996). In addition, research with adoptees has found that children (O’Connor, Deater-Deckard, Fulker, Rutter, and Plomin, 1998) and adolescents (Ge et al., 1996) at genetic risk for antisocial behavior (as indexed by birth mother self-reported antisocial behavior) were more likely to receive negative parenting from their adoptive parents. Finally, genetically informed cross-lagged longitudinal studies have highlighted the role of both evocative and environmental effects (Larsson, Viding, Rijdsdijk, and Plomin, 2008; Tuvblad, Bezdjian, Raine, and Baker, 2013).

In sum, available studies have most strongly supported evocative rGE and/or shared environmental origins to the association between negative parenting and antisocial behavior. These processes are not necessarily mutually exclusive, particularly as there is clear evidence for bidirectional effects between child behavior and parenting from a large number of longitudinal examinations (e.g., Belsky, Pasco Fearon, and Bell, 2007; Lysenko et al., 2013; Miner and Clarke-Stewart, 2008; Stice and Barrera, 1994). Nevertheless, studies do differ rather dramatically on the extent to which they support evocative rGE or environmental mediation. What might explain these differences across studies? Possibilities include specific methodological factors (e.g., twin vs. adoption study, informant-reports), developmental processes (e.g., differences across early childhood, middle childhood, and adolescence), and/or sex differences (e.g., mothers vs. fathers or male vs. female children; Leve, Kim, and Pears, 2005; Meier et al., 2009; Narusyte et al., 2011a).

Critically, however, prior studies have also failed to distinguish between different dimensions of antisocial behavior. As reviewed in Burt (2012), there is now substantial evidence that aggression (e.g., fighting, hitting, threatening) and rule-breaking (i.e., nonaggressive delinquent acts such as stealing, vandalism, truancy, lying) are distinguishable but related dimensions of antisocial behavior characterized by distinct etiologies, developmental trajectories, and phenotypic correlates. For example, aggression demonstrates higher rank-order stability than does rule-breaking (Stanger, Achenbach, and Verhulst, 1997; Tremblay, 2003) and appears to be a particularly heritable form of antisocial behavior (65 % of the variance in aggression is genetic in origin, as compared to 48 % in rule-breaking; Burt, 2009). Moreover, only 38 % of the genetic factors influencing aggression also influence rule-breaking (Burt, 2013), collectively indicating that the genetic architecture of aggression differs to a large extent from that of rule-breaking. Genetic influences on aggression also appear to remain stable across childhood and adolescence while genetic influences on rule-breaking increase substantially over adolescence (Burt and Klump, 2009; Burt and Neiderhiser, 2009). Aggression is also more strongly associated with hostile perceptions of others (Burt, Mikolajewski, and Larson, 2009) and the tendency to experience negative emotions (Burt and Donnellan, 2008; Burt and Larson, 2007). Rule-breaking, by contrast, is more strongly associated with impulsivity (Burt and Donnellan, 2008).

Given the many developmental and etiologic differences between the aggressive and rule-breaking dimensions of antisocial behavior, it is quite possible that the two dimensions are differentially associated with negative parenting, at least at an etiologic level. To our knowledge, however, no study to date has examined this possibility. In order to address this question, the current study examined the association of maternal negativity with aggression and rule-breaking, respectively, in a sample of over 800 families with school-aged twins. This design allowed us to simultaneously examine the roles of evocative rGE and environmental mediation in these associations. Because of the child-based nature of this research design, etiological influences are inferred based on differences in the genetic relatedness at the level of the child (i.e., those who are being parented, rather than those who are parenting). Estimates of genetic influences within this design are thus estimates of the effect of the child's genetic makeup on the behavior of their mother, an effect that is presumably driven via evocative rGE processes (as the child's genes cannot directly influence the behavior of others; Klahr and Burt, 2014). A significant association between genetic influences on parenting and genetic influences on aggression or rule-breaking therefore suggests an evocative rGE effect of child antisocial behavior on parenting.

By contrast, shared environmental influences within a child-based design should capture those influences on

parenting behavior that are shared between the twin children and which act to increase similarity in the parenting that they receive, regardless of their degree of genetic relatedness (Klahr and Burt, 2014). These shared environmental influences are thought to include such factors as family socioeconomic status, neighborhood characteristics, and importantly, characteristics of the parent (i.e., personality and beliefs about parenting). Significant overlap of shared environmental influences between parenting and child antisocial behavior may thus index a causal effect of parenting (a between-family effect, such that higher levels of negative parenting are associated with higher levels of child antisocial behavior, regardless of the genetic relatedness of the twins). Finally, significant non-shared environmental overlap is consistent with a potentially causal association between *differential* negative parenting and child antisocial behavior (a within-family effect; e.g., one twin is treated more negatively than the other and demonstrates higher levels of antisocial behavior as a result). However, it may also index other non-shared factors that influence both parenting and child behavior (e.g., one child is part of a more deviant peer group that influences the child's antisocial behavior and evokes differential negativity). Table 1 summarizes the possible processes underlying the association between parenting and child antisocial behavior and how these possibilities are indexed within our current design.

Methods

Participants

Participants for this study were drawn from the population-based Michigan State University Twin Registry (MSUTR), which includes several independent twin projects (Burt and Klump, 2013; Klump and Burt, 2006). The current study included 824 twin families who were assessed as part of the on-going Twin Study of Behavioral and Emotional Development in Children (TBED-C) within the MSUTR. The TBED-C includes both a completed population-based sample ($n=500$ families; 497 biological mothers and one biological grandmother) and an independent at-risk sample for which inclusion criteria also specified that participating twin families lived in moderately to severely disadvantaged neighborhoods (current $n=324$ families; 297 biological mothers, one step-mother, and one biological grandmother). Conclusions are essentially unchanged with and without the at-risk sample and thus these families were retained for analysis. Assessments took place either in our laboratory at Michigan State University or in participant's homes (in the event that families were unable to travel to the university; $n=38$ families from the population-based sample and 62 at-risk families). Children provided informed assent, while parents provided informed consent for themselves and their children.

Table 1 Potential etiologic processes underlying the association between parenting and child antisocial behavior

Association type	Explanation	In our analyses
Environmental	Negative parenting increases child antisocial behavior or other environmental factors (e.g., community violence) impact both parenting and child behavior.	Significant shared environmental correlation between negative parenting and antisocial behavior.
Passive rGE	The same genes that contribute to negative parenting also contribute to parent and child antisocial behavior.	Because all children, including MZ twins, share 50 % of their genes with each biological parent, passive rGE cannot be examined with any certainty in child-based designs using biologically related family members (including those in our sample).
Evocative rGE	Genetically-influenced child antisocial behavior evokes negative parenting.	Significant genetic correlation between parenting and antisocial behavior.

The twins ranged in age from 6 to 10 years ($M=8.16$, $SD=1.46$; although a few twins had turned 11 by the time they participated) and were 47.6 % female. To be eligible for participation in the TBED-C, neither twin could have a cognitive or physical condition (as assessed via parental screen; e.g., a significant developmental delay) that would preclude completion of the assessment.

Families for both samples were recruited via State of Michigan birth records in collaboration with the Michigan Department of Community Health (MDCH). The MDCH manages birth records in Michigan, and is therefore able to identify all twin births in the state. MDCH identified twins living within 120 miles of East Lansing who were between the ages of 6 and 10 years. Twins were identified either directly from birth records or via the Michigan Twins Project, a large-scale population-based registry of twins in lower Michigan that were themselves recruited via birth records. MDCH then utilized the Michigan Bureau of Integration, Information, and Planning Services database to locate current addresses through parent drivers' license information. Using these addresses, MDCH mailed pre-made recruitment packets to parents of twins. A reply postcard was included for parents to indicate their interest in participating. Interested families were then contacted directly by project staff. Parents who did not respond to the first mailing were sent additional mailings approximately 1 month apart until either a reply was received or up to four letters had been mailed.

This recruitment strategy yielded an overall response rate of 62 % for the population-based sample and 56 % for the at-risk sample, which is similar to or better than those of other twin registries that use anonymous recruitment mailings (Baker, Barton, and Raine, 2002; Hay, McStephen, Levy, and Pearsall-Jones, 2002). Importantly, as detailed in Table 1 of Burt and Klump (2013), the final population-based sample was broadly representative of the area population and of recruited families more specifically (as assessed via a brief questionnaire screen administered to 70 % of non-participating families). Participating twins did not differ from non-participating twins in their average levels of conduct

problems, emotional symptoms, and hyperactivity as assessed via the Strength and Difficulties Questionnaire (Goodman and Scott, 1999; Cohen's d standardized effect sizes = -0.05 , 0.01 , and -0.08 , respectively; all $p \geq 0.29$). Participating families also did not differ from non-participating families on most demographic characteristics (as detailed in Table 1 of Burt and Klump, 2013).

Participating population-based families endorsed ethnic group memberships at rates comparable to area inhabitants (e.g., Caucasian: 86.4 % and 85.5 %, African-American: 5.4 % and 6.3 % for the participating families and the local census, respectively). As anticipated, however, the at-risk sample was significantly more racially diverse (15 % African American, 75 % Caucasian) than was the population-based sample. The at-risk sample also reported lower household incomes ($d=0.30$, $p<0.01$), lower availability of neighborhood resources ($d=0.20$, $p<0.05$), and a higher number of neighborhood problems ($d=0.37$, $p<0.01$) compared to the population-based sample. Moreover, they reported higher levels of maternal negative parenting ($d=0.15$, $p<0.01$), maternal rule-breaking ($d=0.17$, $p<0.05$), paternal rule-breaking ($d=0.16$, $p<0.05$) and child rule-breaking ($d=0.11$, $p<0.05$), although maternal, paternal, and child aggression did not differ across the two samples.

Zygosity was established using physical similarity questionnaires administered to the twins' primary caregiver (Peeters et al., 1998), a common method for establishing zygosity in large twin samples. On average, the physical similarity questionnaires used by the MSUTR have accuracy rates of 95 % or better (Peeters et al., 1998). The current study included 351 MZ twin pairs and 473 DZ twin pairs.

Measures

Aggression (AGG) Both mothers and fathers reported on each of their twins' behavior during the past 6 months using the Child Behavior Checklist (CBCL), a commonly used measure of children's adaptive and maladaptive behaviors (Achenbach, 1991). We focused here on the 18-item aggression scale (e.g.,

“threatens others”, “attacks others”; $\alpha=0.88$ for mother and 0.86 for fathers). Both parents also completed the Sub-types of Antisocial Behavior (STAB) questionnaire on each of their twins behavior during the past year (Burt and Donnellan, 2009). For the current study, we focused on the 10-item aggression (e.g., “he/she got into physical fights”, “he/she swore or yelled at others”; $\alpha=0.89$ for mother and 0.87 for fathers) subscale. Maternal and paternal reports of their children’s levels of aggression were moderately correlated (CBCL: $r=0.54$; STAB: $r=0.51$). Correlations within informants and across scales for aggression were also moderate-to-large in magnitude (Mothers: $r=0.67$, Fathers: $r=0.60$). We thus created a composite by standardizing scores from mother and father reports on the CBCL and the STAB and computing the overall mean. The AGG composite evidenced adequate internal consistency reliability for both mother and father reports ($\alpha=0.89$ and 0.86, respectively).

Rule-Breaking (RB) For our measure of rule-breaking, we focused on the 17-item rule-breaking scale on the CBCL (e.g., “breaks rules”, “cheats”) and the 11-item rule-breaking on the STAB (e.g., “he/she littered public areas”, “he/she stole property from school”), as completed by both mothers and fathers. For both scales, it is worth noting that some behaviors were virtually non-existent (e.g., smokes tobacco, uses illicit drugs) or very rare in this sample (e.g., drinks alcohol, thinks about sex too much), not surprising given the age range of our participants (6–10 years). In order to create a more developmentally sensitive measure of RB, we submitted the CBCL and STAB rule-breaking items to a principle-axis exploratory factor analysis, separately for mother and father reports. The scree plot suggested a clear one-factor solution for both informants. We then selected the items with a principle factor loading greater than 0.5 for either informant. This resulted in a 13-item measure of rule-breaking behaviors (6 items from the STAB and 7 items from the CBCL; α for modified RB scale=0.75 for mothers and 0.68 for fathers). Final items are presented in Table 2.

Negative Mothering Child and Parent Reports The Parental Environment Questionnaire (PEQ) was administered to measure various qualities of the parent–child relationship using parallel parent and child forms. The current study focused on the 12-item parent–child conflict scale. Items included “I often criticize my child”, “I often lose my temper with my child”, and “Once in a while my child has been really scared of me”, with alterations in wording appropriate for parents and children. Each informant rated items on a 4-point scale (1=*definitely true*; 2=*somewhat true*; 3=*somewhat false*; 4=*definitely false*). Mothers individually rated their relationship with each of their twin children. Children reported on their relationship with their mother. Consistent with prior studies (Burt et al., 2003, 2005, 2007), child and maternal ratings of

Table 2 Items included in the Final 13-item rule-breaking scale

Scale	Item
CBCL	Breaks rules at home, school, or elsewhere
CBCL	Hangs around with others who get in trouble
CBCL	Lying or cheating
CBCL	Steals at home
CBCL	Steals outside of home
CBCL	Swearing or obscene language
CBCL	Vandalism
STAB	He/she shoplifted things
STAB	He/she littered public areas by smashing bottles, tipping trash cans, etc.
STAB	He/she stole a bicycle
STAB	He/she stole property from school or work
STAB	He/she left home for an extended period of time without telling family/friends
STAB	He/she was suspended or expelled from school

CBCL = Child Behavior Checklist (includes behaviors within the past 6 months); STAB = Sub-types of Antisocial Behavior (includes behaviors within the past year)

conflict were significantly if modestly correlated ($r=0.18$), and were thus averaged to create a composite of reported mother-child conflict.

Observer Ratings Parenting was also observed during an 8 min video-taped interaction between each mother-child dyad (i.e., there were two such interactions per family, one with each twin). The on-campus interactions took place in laboratory offices that were set-up to resemble living rooms, with cameras inconspicuously installed in the ceiling. For those assessments occurring in participants’ homes, interactions took place in a family living space with a video camera placed on a tripod in the room. Each mother-child dyad was asked to use an Etch-a Sketch and work together to draw specific pictures, with the mother and child only using one dial each (a mildly frustrating task). The interactions were then scored by trained undergraduate research assistants using the Parent–child Interaction System or PARCHISY (Deater-Deckard, Pylas, and Petrill, 1997).

For the current study, we focused on two measures of maternal negativity: negative content (e.g., use of criticism, physical control of the dials, and physical control of the child’s hand or body) and negative affect with child (e.g., rejection, frowning, cold/harsh tone). Observer reliability was assessed by randomly assigning 10 % of all tapes to be rated by a second observer, and then comparing the primary and secondary ratings using intraclass correlations. Intraclass correlations between coders were $r \geq 0.96$ for both scales. Mother-child dyad ratings were available for 96.2 % of twins (3.8 % of videos were not codeable; e.g., there was no sound, the file

was corrupted). In order to capture overall levels of maternal negativity, observer-ratings were combined with parent and child-reports by standardizing each variable and computing the overall mean. As found in numerous independent studies using observer and informant-reports of parenting and behavior (Arseneault et al., 2003; Burt et al., 2011; Burt and Klump, 2014), observed and reported negativity were significantly, if modestly, correlated ($r=0.12$).

Statistical Analyses

Twin methodology uses the difference in the proportion of segregating genes shared between monozygotic (MZ) and dizygotic (DZ) twin pairs to estimate genetic and environmental contributions to variance in observed behaviors or characteristics (phenotypes). MZ or identical twins result from a single fertilized zygote splitting and hence share 100 % of their segregating genes. DZ or fraternal twins are the result of two independent conceptions and so, like all full siblings, share an average of 50 % of their segregating genes. Utilizing these differences, the variance within observed behaviors is partitioned into three components: additive genetic effects (A), shared environmental effects (C), and non-shared environmental effects plus measurement error (E). The additive genetic component (A) is the effect of individual genes summed over loci. Additive genetics effects, if acting alone, would result in MZ correlations that are double those of DZ correlations. The shared environment (C) is that part of the environment that acts to make the twins within a pair similar to each other regardless of the proportion of genes shared. The non-shared environment (E) includes environmental factors unique to each twin within a pair along with measurement error. The non-shared environment differentiates each twin within a pair, making them less similar.

The Equal Environments Assumption is foundational to these analyses, as it assumes that MZ twin pairs are no more likely to share the environmental factors that are etiologically relevant to the phenotype under study than are DZ twin pairs. Under this assumption, any differences in the similarity of MZ and DZ twins are due to differences in their genetic similarity. The Equal Environments Assumption has been repeatedly tested and found to be valid for numerous phenotypes (Hettema, Neale, and Kendler, 1995; Kendler et al., 1993; Morris-Yates et al., 1990; Scarr and Carter-Saltzman, 1979), but it remains an assumption for any particular phenotype until subjected to empirical testing. Parenting does appear to differ somewhat across zygosity (i.e., parents treat MZ twins more similarly than DZ twins). However, because twin similarities are associated with actual rather than perceived zygosity (Kendler et al., 1994), these differences are likely a function of evocative rGE rather than differences in parental treatment.

For our primary analyses, we made use of a bivariate correlated factors model (see Fig. 1) in which the variance within and the covariance between child behavior and parenting were decomposed into their genetic and environmental components. This enabled us to obtain estimates of etiological influences on child behavior and parenting, as well as on their overlap. In particular, genetic and environmental correlations index the proportion of genetic and environmental influences on child behavior that are shared with parenting behavior. A genetic correlation of 1.0 would indicate that the genetic influences on child behavior overlap entirely with those on maternal negativity (i.e., the same genes influence both phenotypes). A correlation of zero would indicate no genetic overlap. These models thus enabled us to explicitly estimate the extent to which genetic and environmental influences on child aggression and rule-breaking, respectively, overlap with those on maternal negativity.

Because there was a small amount of missing data, we made use of Full-Information Maximum-Likelihood raw data techniques (FIML), which produce less biased and more efficient and consistent estimates than techniques like pairwise or listwise deletion in the face of missing data (Little and Rubin, 1987). Mx (Neale et al., 2003) was used to fit models to the raw data. When fitting models to raw data, variances, covariances, and means of those data are first freely estimated by minimizing minus twice the log-likelihood ($-2\ln L$). The minimized value of $-2\ln L$ in the baseline model was then compared with the $-2\ln L$ obtained in the biometric models to yield a likelihood-ratio chi-square test. The chi-square was then converted to the Akaike's Information Criterion (AIC; $AIC = \chi^2 - (2 * df)$; Akaike, 1987), so as to measure model fit relative to parsimony. Negative AIC values are generally thought to indicate that the biometric model provides a good fit to the data. To control for any effect of child age, sex, or ethnicity, these variables were regressed out of the data prior to analysis (McGue and Bouchard, 1984).

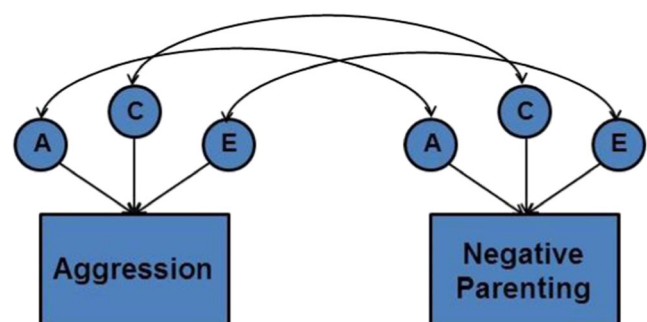


Fig. 1 Bivariate correlated factors model. Path diagram of a full ACE Correlated Factors model for child aggression and maternal negative parenting. The variance in each phenotype is parsed into additive genetic effects (A), shared environmental effects (C), and nonshared environmental effects (E). Genetic and environmental correlations are indicated via the double-headed arrows. Paths (indicated via single-headed arrows) are squared to estimate the percentage of variance accounted for

Results

Descriptive Statistics

Descriptive statistics are presented in Table 3. Boys exhibited higher levels of aggression and rule-breaking than girls (all $p < 0.05$; Cohen’s d ranged from 0.13 - 0.41). Mothers also exhibited higher levels of negative parenting with their sons than with their daughters, across all informants (all $p < 0.05$; Cohen’s d ranged from 0.09 - 0.27). As expected, aggression and rule-breaking were positively associated with each other ($r = 0.60, p < 0.01$) and with maternal negativity (r 's = 0.33 and 0.27 with aggression and rule-breaking, respectively; both $p < 0.01$). 8.4 % of boys and 5.2 % of girls scored above the borderline clinically significant cut-off for aggression, as defined by the Achenbach scoring system ($T > 65$; Achenbach and Rescorla, 2001). For rule-breaking, 6.1 % of boys and 2.0 % of girls scored above the cut-off.

Intraclass Correlations

Prior to model fitting analyses, intraclass and cross-twin, cross-trait correlations were computed for MZ and DZ twin pairs on the negative parenting, aggression, and rule-breaking composites. MZ intraclass correlations that are double those of DZ intraclass correlations are indicative of genetic effects, whereas MZ correlations that are less than double but still greater than DZ correlations suggest the importance of genetic

and shared environmental effects. As seen in Table 4, intraclass correlations suggest both genetic and shared environmental effects may be important for aggression and rule-breaking, as well as for negative parenting. Cross-twin, cross-trait correlations are also presented in Table 4. These correlations give a preliminary indication of the degree of genetic and environmental overlap between parenting and child behavior. Cross-twin, cross trait correlations for aggression and negative parenting suggest the possibility of genetically- and shared environmentally-mediated overlap ($MZr = 0.32, DZr = 0.19$). The pattern differs somewhat for rule-breaking, pointing instead to predominantly shared environmentally-mediated overlap ($MZr = 0.23, DZr = 0.18$).

Bivariate Model-Fitting Analyses

Fit indices for both models indicated a reasonable fit to the data (Aggression: baseline model: $-2lnL = 6265.12$ on 3,268 df ; full model: $-2lnL = 6297.72$ on 3,285 $df, AIC = -35.39$; Rule-breaking: baseline model: $-2lnL = 6656.38$ on 3,268 df ; full model: $-2lnL = 6681.62$ on 3,285 $df, AIC = -42.76$). Parameter estimates for genetic and environmental influences on aggression, rule-breaking, and negative parenting are presented in Table 5. Genetic, shared, and non-shared environmental estimates were significant for both aggression and rule-breaking. For maternal negativity, there was evidence of evocative genetic (22 %), shared environmental (37 %), and non-shared environmental (42 %) influences.

Table 3 Mean levels of aggression, rule-breaking, and negative parenting separately by child sex

	Males					Females				
	Mean	SD	Min	Max	N	Mean	SD	Min	Max	N
Aggression										
CBCL-Mother report	5.16**	5.37	0	31	855	4.01	4.54	0	28	783
CBCL- Father report	5.01**	4.52	0	25	709	4.14	4.45	0	25	659
STAB-Mother report	19.59**	6.18	10	47	842	17.26	5.27	10	35	775
STAB-Father report	18.79**	5.35	10	46	697	16.79	4.85	10	37	653
Rule-breaking										
CBCL-Mother report	1.89**	2.23	0	14	855	1.20	1.62	0	14	783
CBCL- Father report	1.78**	1.99	0	17	709	1.27	1.53	0	8	659
STAB-Mother report	11.26**	0.88	11	20	842	11.11	0.56	11	21	775
STAB-Father report	11.25*	1.78	11	25	697	11.08	0.35	11	15	653
Negative Parenting										
Observed Negative Content	1.67	0.78	1	6	831	1.60	0.72	1	4	751
Observed Negative Affect	1.35*	0.66	1	6	831	1.28	0.56	1	4	754
Conflict- Mother report	21.21**	6.08	12	42	847	20.31	5.76	12	41	776
Conflict- Twin report	21.33**	6.21	12	46	837	19.71	5.64	12	45	784

CBCL = Child Behavior Checklist. STAB = Sub-types of Antisocial Behavior

*Mean are significantly different between males and females, $p < 0.05$

** Means are significantly different between males and females, $p < 0.01$

Table 4 Intraclass and cross-twin cross-trait correlations for aggression, rule-breaking, and negative parenting

	1	2	3	4	5	6
1. Aggression- a	-	0.62	0.30	0.42	0.33	<i>0.19</i>
2. Rule-breaking- a	0.57	-	0.25	0.33	0.44	<i>0.18</i>
3. Negative parenting- a	0.37	0.30	-	0.19	0.18	0.47
4. Aggression- b	0.68	0.46	0.32	-	0.62	0.30
5. Rule-breaking - b	0.46	0.68	0.22	0.57	-	0.25
6. Negative parenting- b	<i>0.32</i>	<i>0.23</i>	0.58	0.38	0.30	-

A = twin a; b = twin b; MZ correlations are below the diagonal, DZ correlations are above. Intraclass correlations are in bold. Cross-twin cross-trait correlations are in italics

We also examined the genetic and environmental correlations between child behavior and negative parenting (see Fig. 2). For aggression and maternal negativity, the overlap was both genetic ($r_A=0.40$; 95 % CI [0.05, 0.95]) and shared environmental ($r_C=0.62$; 95 % CI [0.18, 1.00]) in origin. By contrast, the association between negative parenting and rule-breaking was largely shared environmental in origin ($r_C=0.82$; 95 % CI [0.39, 1.00]). There was no significant genetic overlap ($r_A=0.05$; 95 % CI [-0.37, 0.39]). Notably, a comparison of the genetic correlations using fisher’s r to Z transformation indicated that the genetic correlation between child rule-breaking and maternal negativity was significantly smaller than the genetic correlation between child aggression and maternal negativity ($p<0.01$), while the shared environmental correlation was significantly larger for rule-breaking and negativity ($p<0.01$). Lastly, there was evidence of small but significant non-shared environmental overlap between maternal negativity and aggression ($r_E=0.14$; 95 % CI [0.04, 0.24]) and rule-breaking ($r_E=0.19$; 95 % CI [0.09, 0.29]).

Discussion

The current study examined the etiology of the association between two dimensions of antisocial behavior in children, aggression and rule-breaking, and the presence of maternal

Table 5 Genetic and environmental etiology of aggression, rule-breaking, and negative parenting

	A	C	E
Aggression	55 % (0.40, 0.71)	14 % (0.01, 0.28)	30 % (0.26, 0.36)
Rule-breaking	58 % (0.42, 0.71)	14 % (0.03, 0.27)	29 % (0.24, 0.34)
Maternal negativity	22 % (0.04, 0.39)	37 % (0.22, 0.50)	42 % (0.36, 0.49)

A = genetic variance; C = shared environmental variance; E = non-shared environmental variance. 95 % confidence intervals are presented in parentheses beside their respective correlations. Significant estimates are in bold

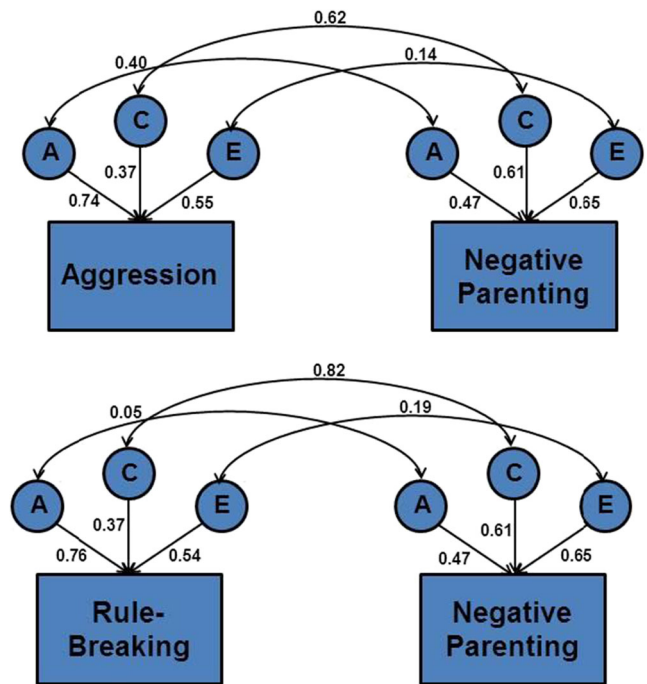


Fig. 2 Results from model-fitting analyses

negativity. The results highlighted an important difference across aggression and rule-breaking. Although the association between aggression and maternal negativity was partially genetic in origin, the association between rule-breaking and maternal negativity was primarily shared environmental in origin. Such findings suggest that evocative rGE processes are specific to the relationship between maternal negativity and child aggression, and do not contribute to the association with non-aggressive rule-breaking. Put differently, mothers appear to be responding to their child’s genetically-influenced aggressive behavior with higher levels of negativity towards their child. Genetic influences on child rule-breaking, by contrast, do not appear to elicit this reaction. That said, less than half of the evocative genetic influences on maternal negativity overlapped with genetic influences on child aggression, suggesting other genetically-influenced child characteristics are also important for evoking negative parenting. Although it remains unclear what these other characteristics might be (temperament, internalizing problems, and/or ADHD symptoms are all possible candidates as these characteristics are both genetically-influenced and associated with parenting), they do not appear to include non-aggressive rule-breaking antisocial behavior.

There was also evidence for small but significant non-shared environmental overlap with maternal negativity, although the shared environment overlap was far more prominent. Indeed, shared environmental overlap was significant and moderate-to-large in magnitude for both aggression and rule-breaking. This environmental mediation was particularly important for rule-breaking, both because of the absence of

genetic overlap, but also because the shared environmental correlation with maternal negativity was significantly stronger for rule-breaking as compared with aggression.

There are several limitations to the existing study. First, our assessment of child aggression and rule-breaking relied solely upon parent report. Although we were able to utilize reports from both mothers and fathers, additional informants would be beneficial. Fortunately, our measures of parenting included parent-reports, child-reports, and observer-ratings and so we were able to minimize shared-informant effects between our measures of antisocial behavior and parenting. Relying solely upon parent reports of child aggression and rule-breaking is also less of a concern given the age range of our participants (6–10 years), both because of concerns regarding child reliability, but also because parents are relatively well-informed on their child's acting out behaviors during middle childhood (at least as compared to adolescence; Dishion and McMahon, 1998; Patterson and Stouthamer-Loeber, 1984). This points to a second limitation, namely that the results of this study are only applicable during middle childhood and not during other developmental stages. Prior research has highlighted substantial developmental shifts in the etiology and phenotypic expression of aggression and rule-breaking across development (Burt and Klump, 2009; Burt and Neiderhiser, 2009; Burt, 2014; Stanger et al., 1997; Tremblay, 2003). Additional research would therefore be needed before making any conclusions regarding the etiology of the relationships among aggression, rule-breaking, and maternal negativity in the preschool and adolescent periods. Also, given that most of our sample exhibited low levels of aggression and rule-breaking, the current findings are most applicable for aggressive and rule-breaking behaviors within the normal range. Additional research should examine these associations in a clinic-based sample.

Next, our analyses focused solely on maternal negativity, to the exclusion of paternal negativity. Behavioral genetic research suggests the presence of etiological differences between maternal and paternal negativity (Klahr and Burt, 2014). Given these differences, we plan to explicitly compare etiological differences in the associations between maternal and paternal parenting and child outcomes in future analyses. In addition, other aspects of the parent–child relationship are also associated with antisocial behavior (e.g., positivity/warmth, control/limit setting, and attachment; Marcus and Kramer, 2001; Pettit et al., 2001; Stormshak et al., 2000; Troy and Sroufe, 1987) and may exhibit differential associations with aggression vs. rule-breaking. Sub-types of negative parenting (e.g., verbal criticism vs. physical punishment) may also demonstrate differential associations. Additional research in this domain is needed. Finally, the current study is unable to disambiguate the effects of passive rGE from environmentally-mediated effects. Because we examined biological families, we cannot rule-out the possibility that shared genes may account for what appears to be an

environmental association. That said, existing empirical literature (Marceau et al., 2013; Narusyte et al., 2011b) has not identified passive rGE effects in the association between parenting and child antisocial behavior, implying that passive rGE is unlikely to account for the association between parenting and offspring behavior problems in this study. Building on these results, several adoption studies have found evidence of a shared environmentally-driven association between parenting and antisocial behavior in general (Burt et al., 2007; Klahr et al., 2011a, b), results that again argue against passive rGE (because passive rGE is entirely circumvented in non-biologically related family members).

In spite of these limitations, the current study has several important implications. The results highlight a possible explanation for differing conclusions about genetic vs. environmental mediation in the existing behavior genetic literature (e.g., Klahr et al., 2011a; Marceau et al., 2013) and for differing conclusions among longitudinal investigations of parenting and behavior problems that implicate parent-driven effects (Gardner et al., 2003) vs. child-driven effects (Reitz et al., 2006). These results also add to the large body of literature supporting a distinction between aggressive and rule-breaking dimensions of antisocial behavior in children. For example, aggression is more strongly associated with negative emotion and affective dysfunction (Burt and Donnellan, 2008, 2009; Burt, 2012; Moffitt, 1993, 2003; Tackett, 2010; Verona, Patrick, and Lang 2002) while rule-breaking is more strongly associated with impulsivity (Burt and Donnellan, 2008, 2009; Burt, Donnellan, and Tackett, 2012; Hopwood et al., 2009; Moffitt, 2003; Tackett, 2010). In addition, aggression is uniquely associated with executive dysfunction (Barker et al., 2007; Barker et al., 2011; Hancock, Tapscott, and Hoaken, 2010; Miura, 2009). The current study adds to such work by suggesting that even those correlates that are associated with both aggression and rule-breaking have different etiologic underpinnings. In particular, evocative rGE effects on maternal negativity are in part a function of child aggression, but are entirely unrelated to non-aggressive rule-breaking in those same children.

Next, the shared environmentally-mediated associations between maternal negativity and child aggression and rule-breaking, respectively, are consistent with a potentially causal effect of parenting on these behaviors. Such results dovetail nicely with the well-established role of parenting in the initiation and maintenance of antisocial behaviors in the treatment literature (Barkley, 1997; Klahr and Burt, 2014; McMahon, Forehand, and Foster, 2005). Because our study is cross-sectional, it is worth noting that other third variable shared environmental factors may also be at play, in addition to parenting. For example, neighborhood disadvantage and financial strain are associated with negative parenting (Bradley et al., 2001; Klebanov et al., 1994; McLeod and Shanahan, 1993) and with antisocial behavior in children (Costello, Foley, and Angold, 2006; Côté et al., 2006). As both neighborhood disadvantage and financial strain

are considered family-wide variables, they may well contribute to the common shared environmental variance between parenting and child antisocial behavior.

Finally, the current results have implications for understanding both the etiology of negative parenting and the transactional nature of parenting and child behavior problems. A given adult's parenting behaviors are not just a reflection of the parent, but are also dependent on the characteristics of the child being parented. Our results further suggest that these evocative effects are more prominent for aggressive behavior problems than they are for rule-breaking behaviors. Although most of our sample exhibited behavior problems and negative parenting in the normative range, parental reactions to normative acting out behaviors in children can serve to either promote normative development or foster a trajectory of worsening behavior problems (i.e., the coercive cycle; Patterson, 1982). As a result, many intervention programs attempt to teach parents how to constructively respond to normative behavior problems before these problems reach clinical severity (e.g., The Chicago Parent Program; Gross et al., 2009). The evocative association between aggression and maternal negativity identified within the current population-based sample thus has implications related to tailoring prevention and treatment for primarily aggressive vs. primarily rule-breaking subtypes of antisocial behavior (Klahr and Burt, *in press*). In particular, although behavioral parent-management training is considered the gold-standard treatment for antisocial behavior (Brestan and Eyberg, 1998), the early age of onset and high levels of rank-order stability associated with aggressive behavior problems (Tremblay, 2003) suggests the importance of early intervention for the most aggressive preschool children. This early intervention might focus on how parents respond to aggressive behaviors in their children and subsequently help to curtail the maintenance and escalation of negative parenting practices for children who are likely to continue to be more aggressive than their peers across development.

Acknowledgments This project was supported by R01-MH081813 from the National Institute of Mental Health (NIMH) and by R01-HD066040 from the Eunice Kennedy Shriver National Institute for Child Health and Human Development (NICHD), awarded to Drs. Burt and Klump. The content is solely the responsibility of the authors and does not necessarily represent the official views of the NIMH, the NICHD, or the National Institutes of Health. The authors thank all participating twins and their families for making this work possible.

Conflict of Interest The authors declare that they have no conflict of interest.

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