Published in final edited form as:

Clin Psychol Sci. 2016 May ; 4(3): 511–526. doi:10.1177/2167702615618164.

Neighborhood Disadvantage Alters the Origins of Children's Nonaggressive Conduct Problems

S. Alexandra Burt, PhD¹, Kelly L. Klump, PhD¹, Deborah Gorman-Smith, PhD², and Jenae M. Neiderhiser, PhD³

¹ Department of Psychology, Michigan State University, East Lansing, MI

² School of Social Service Administration, University of Chicago, Chicago, IL

³ Department of Psychology, The Pennsylvania State University, University Park, PA

Abstract

Neighborhood disadvantage plays a pivotal role in child mental health, including child antisocial behavior (e.g., lying, theft, vandalism; assault, cruelty). Prior studies have indicated that shared environmental influences on youth antisocial behavior increase with increasing disadvantage, but have been unable to confirm that these findings persist once various selection confounds are considered. The current study sought to fill this gap in the literature, examining whether and how neighborhood disadvantage alters the genetic and environmental origins of child antisocial behavior. Our sample consisted of 2,054 child twins participating in the Michigan State University Twin Registry, half of whom were oversampled to reside in modestly-to-severely impoverished neighborhoods. We made use of an innovative set of nuclear twin family models, thereby allowing us to disambiguate between, and simultaneously estimate, multiple elements of the shared environment as well as genetic influences. Although there was no evidence that the etiology of aggressive antisocial behavior was moderated by neighborhood disadvantage, the etiology of non-aggressive antisocial behavior shifted dramatically with increasing neighborhood disadvantage. Sibling-level shared environmental influences were estimated to be near zero in the wealthiest neighborhoods, and increased dramatically in the most impoverished neighborhoods. By contrast, both genetic risk and family-level shared environmental transmission were significantly more influential in middle- and upper-class neighborhoods than in impoverished neighborhoods. Such results collectively highlight the profound role that pervasive neighborhood poverty plays in shaping the etiology of child non-aggressive antisocial behavior. Implications are discussed.

Keywords

antisocial behavior; conduct problems; neighborhood poverty; genetic

There is now considerable evidence that neighborhood disadvantage predicts child antisocial behavior (Brooks-Gunn, Duncan, Klebanov, & Sealand, 1993; Jencks & Mayer, 1990;

Correspondence: Address correspondence to Alex Burt, Department of Psychology, Michigan State University, 107D Psychology Building, East Lansing, MI 48824. Electronic mail may be sent to burt@msu.edu. Fax number is (517) 432-2476.

None of the authors report any conflicts of interest.

Leventhal & Brooks-Gunn, 2000; Sampson, Raudenbush, & Earls, 1997). Moreover, this effect appears to be causal, at least to an extent. Experimental studies (Damm & Dustmann, 2014; Ludwig, Duncan, & Hirschfield, 2001) leveraging quasi-randomized neighborhood assignment (i.e., refugee immigrants to Denmark assigned to neighborhoods) or randomized neighborhood assignment (i.e., housing vouchers in Baltimore) have generally indicated that the neighborhood structural characteristics of poverty and crime causally increase risk for youth antisocial behavior (although for an excellent debate of these and related findings, see Ludwig et al., 2008; Sampson, 2008). A quasi-experimental comparison of cousins residing in neighborhoods with different levels of neighborhood disadvantage (Goodnight et al., 2012) further supported this conclusion.

Although such work brings much needed attention to the economic and related conditions that contribute to child antisocial behavior, studies of poverty, crime, and more general disadvantage per se tell us little about how neighborhood structural characteristics influence child outcomes. Several theoretical frameworks for understanding the effects of neighborhood on child antisocial behavior have been developed, all of which focus on social processes within the neighborhood. Jencks & Mayer (1990) highlight two such models: the ‘collective socialization model’ proposes that the neighborhood influences children via community social organization and social control, including supervision and monitoring by adult neighbors. The ‘epidemic (or contagion) model’ focuses on the ways in which problematic behavior in neighborhood residents (and particularly neighborhood peers) can influence or spread to children. The ‘collective efficacy model’ described by Leventhal and Brooks-Gunn (2000) synthesizes the above two models but limits the mechanisms of influence to community-level (as opposed to family- or individual-level) regulatory processes and institutions. The ‘developmental-ecological model’ (Coie, Miller-Johnson, & Bagwell, 2000; Gorman-Smith, Tolan, & Henry, 2000) builds on Bronfenbrenner’s social ecological model of development (Bronfenbrenner, 1979, 1988) to additionally incorporate family- and community-level influences on child development.

Consideration of the individual: A key missing ingredient

Critically, however, the role of individual genetic and biologic risk has not been incorporated in the above models in any meaningful way. The sole (albeit brief) exception to this can be found in Jencks and Mayer (1990): “epidemic models must allow for individual differences in susceptibility to neighborhood or school influences. Epidemic model of antisocial or self-destructive behavior usually impute differential susceptibility differences in upbringing, but the model works the same way if we impute individual differences to heredity or to chance” (p.114). One way to incorporate individual differences in susceptibility into all of the above models is via the gene-environment interaction (GxE). GxE is defined as differential responsiveness to environmental risk as a function of genetic variability (Plomin, DeFries, & Loehlin, 1977; Rutter, Silberg, O’Connor, & Simonoff, 1999a, 1999b), and is thought to constitute a fundamental mechanism through which genes influence human behavior and mental health (Johnson, in press; Moffitt, Caspi, & Rutter, 2006; Rutter, Moffitt, & Caspi, 2006).

There is provocative, if limited, support for possible GxE between youth antisocial behavior and structural characteristics of the neighborhood (Cleveland, 2003; Tuvblad, Grann, & Lichtenstein, 2006). Cleveland (2003) examined the heritability of adolescent aggression by neighborhood in more than 2,000 sibling pairs from the National Longitudinal Study of Adolescent Health. In classifying neighborhoods, he created a composite of neighborhood disadvantage (i.e., proportion of single-parent homes, proportion of households with annual incomes of less than \$15,000, and the unemployment rate). Neighborhoods were then dichotomized into disadvantaged (defined as the 25% most disadvantaged neighborhoods) and adequate (the remaining 75% of neighborhoods). Heritability estimates for aggression were calculated for each of the two neighborhood types. Results revealed that although aggression was genetically influenced regardless of neighborhood type, shared environmental influences (i.e., those that create similarities across family members regardless of the proportion of genes shared) were significant only for those residing in disadvantaged neighborhoods. Similarly, Tuvblad and colleagues (2006) examined how contextual and familial risk (defined via parental education and occupation, and socioeconomic conditions in the neighborhood) moderated the heritability of general antisocial behavior in a population-based Swedish study of 1,133 adolescent twin pairs. As with Cleveland (2003), Tuvblad et al. (2006) found that shared environmental influences on antisocial behavior were more important for adolescents residing in disadvantaged environments. Genetic influences, by contrast, were more important in advantaged environments.

The above findings thus collectively suggest that shared environmental influences on antisocial behavior are more influential for those living in disadvantaged neighborhoods. The specific processes underlying this pattern of moderation remain unclear, but one possibility is that some experiences are so risky that they can elicit psychopathological outcomes even in the absence of genetic risk, a phenomenon referred to as a bioecological GxE. The bioecological model of GxE (Bronfenbrenner & Ceci, 1994; Pennington et al., 2009) harkens back to early notions that genetic influences may sometimes be most strongly expressed in 'average, expectable environments' (Scarr, 1992), while deleterious environments amplify environmental influences (Lewontin, 1995; Pennington et al., 2009; Raine, 2002). The logic of this model was best illustrated by Lewontin (1995) through his analogy of genetically variable seeds that are planted in either a nutrient-rich or a nutrient-deprived field (Lewontin, 1995). The environmental adversity conferred by the deprived soil should eventuate in field populated largely by short plants. By contrast, because all plants received adequate nutrition in the nutrient-rich soil, the plants would be able to fully express their genetic endowment for height, making height more heritable in this environment. Put differently, some adverse experiences provide such a strong 'social push' for a given outcome that the importance of genetic factors in these environments is diminished (Raine, 2002; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). Only in the absence of these risks can genetically-mediated individual differences fully manifest. If true, such findings would have key implications for the treatment and prevention of child antisocial behavior, as well as future GWAS studies of antisocial behavior, as they would suggest that antisocial behavior may be more or less genetic in origin across various contexts.

Unfortunately, these findings of shared environmental (and possibly genetic) moderation by neighborhood disadvantage are less conclusive than one would like, for two key reasons. First, although Cleveland (2003) examined aggression as the outcome variable, neither the Cleveland nor the Tuvblad study differentiated between or considered both aggressive and non-aggressive rule-breaking forms of antisocial behavior. This is potentially problematic since there is converging evidence that, although aggression (e.g., assaulting others, bullying; AGG) and non-aggressive rule-breaking (e.g., lying, stealing, vandalism; RB) are moderately-to-strongly correlated, they nevertheless constitute meaningfully distinct dimensions of antisocial behavior. As reviewed previously (Burt, 2012; Tremblay, 2010), AGG and RB evidence distinctive developmental trajectories, demographic correlates, personological underpinnings, and importantly, etiologic differences. In particular, AGG is a highly heritable (65%) behavioral dimension that emerges in early childhood and exhibits specific ties to negative emotionality and executive dysfunction. Although the frequency of aggressive behavior decreases after early childhood, those who are most aggressive early in life typically continue to aggress at relatively high rates across the lifespan. By contrast, RB demonstrates particularly strong associations with impulsivity, is most frequent during adolescence, and evidences more moderate levels of genetic influences (48%) and stronger shared environmental influences as compared to AGG (18% versus 5%, respectively). It is thus entirely possible that neighborhood poverty differentially moderates AGG and RB.

Second, and more importantly, neither the Cleveland nor the Tuvblad study evaluated whether the increase in shared environmental influences with neighborhood disadvantage actually reflected the increasing importance of environmental experiences on adolescent antisocial behavior or whether it instead reflected an increasing role for passive gene-environment correlation (rGE) in antisocial behavior. Passive rGE refers to the fact that the environment parents provide to their biological children likely reflects the genetically influenced preferences/ tendencies of the parent. And because parents also share genes with their biological children, the child's genes are then correlated with her environmental experiences (thereby mimicking shared environmental influences; Neiderhiser et al., 2004). In this case, what appears to be an increasing effect of the shared environment on youth antisocial behavior with increasing neighborhood disadvantage could actually reflect an increasing role for passive rGE, such that parents with a tendency towards antisocial behavior themselves are both selecting into more disadvantaged neighborhoods and passing on genes of risk for antisocial behavior to their children. In short, it is as yet entirely unclear whether the increase in shared environmental influences with increasing neighborhood disadvantage does in fact reflect the increasing influence of actual environmental experiences in youth antisocial behavior.

Assortative mating can also inflate estimates of shared environmental influences. Assortative mating is thought to reflect a largely active rGE process in which individuals seek out and mate with others similar to themselves. To the extent that these phenotypic similarities between spouses reflect genetic similarities, assortative mating in the parents would increase the proportion of genes shared by DZ twins (but not MZ twins, who are already genetically-identical). By doing so, assortative mating can serve to artifactually inflate shared environmental estimates. This point is critically important here, since it is now well known that there are at least modest levels of assortative mating for antisocial behavior (Krueger,

Moffitt, Caspi, Bleske, & Silva, 1998). Should assortative mating for antisocial behavior varies with neighborhood (which is at yet unknown), it could be possible that prior findings of shared environmental moderation actually reflect increases in assortative mating.

How might we disambiguate actual shared environmental influences from passive rGE and assortative mating? The answer lies in the methodologic design. Both Tuvblad et al. (2006) and Cleveland (2003) made use of the classical twin design, evaluating the extent to which twin similarity varied by zygosity in advantaged versus disadvantaged neighborhoods, respectively. Although useful in many ways, this design is unable to disambiguate passive rGE from estimates of the shared environment, and is thus considered to be a less optimal design for the study of shared environmental influences (Burt, 2014). Fortunately, there is a straightforward solution to this dilemma: namely, we could also include data on the twins' parents. This extension of the classical twin design is referred to as the nuclear twin family design. The nuclear twin family model provides two additional pieces of information, over and above the covariance between the twins, on which to base parameter estimates: the covariance between parents and the covariance between parents and children. This additional information allows the nuclear twin family model to, among other things, disambiguate shared environmental influences shared only by siblings (S or sibling-level) from those shared by parents and children (F or family-level; see Table 1). The model then capitalizes on the newfound individuation of the family-level environment by further modeling its covariance with genetic influences, thereby allowing researchers to both explicitly estimate passive rGE and to disambiguate it from sibling-level shared environmental influences. Comparing these various estimates across advantaged and disadvantaged neighborhoods, respectively, thus allows us to explicitly evaluate which specific components of the shared environment vary by neighborhood status. In other words, we would be able to more definitively evaluate whether the increase in shared environmental influences reflects actual increases in the importance of the environment on child antisocial behavior.

The current study sought to do just this, making use of the nuclear twin family model to examine whether and how neighborhood disadvantage moderates the etiology of aggressive and non-aggressive antisocial behavior, respectively. Our sample consisted of 1,027 child twin pairs, half of whom were oversampled to reside in modestly-to-severely impoverished neighborhoods. Consistent with the results of existing twin studies (Cleveland, 2003; Tuvblad et al., 2006), we expected to find evidence of shared environmental moderation of child antisocial behavior by neighborhood disadvantage. We further expected this moderation to be more pronounced for RB, given meta-analytic and nuclear twin family studies indicating that shared environmental influences are far more salient for RB than for aggression (Burt, 2009; Burt & Klump, 2012), as well as evidence that RB may be particularly affected by broader societal processes (Breslau et al., 2011). We further anticipated that this shared environmental moderation would be a function of actual shared environmental experiences rather than passive rGE or assortative mating, given the important role of shared environmental influences on youth RB noted in other studies (Burt & Klump 2012).

METHODS

Participants were recruited as part of the Twin Study of Behavioral and Emotional Development in Children (TBED-C), a study within the population-based Michigan State University Twin Registry (MSUTR) (Burt & Klump, 2013; Klump & Burt, 2006). The TBED-C includes two independent samples: a population-based sample of 1,054 twins from 527 families recruited from across lower Michigan, and an “at-risk” sample of 1,000 twins from 500 families residing in modestly-to-severely disadvantaged neighborhoods in the same recruitment area. These two samples were combined for our primary analyses, thereby allowing us to capture both severity and variability in neighborhood poverty, and to maximize our sample size. To be eligible for participation in the TBED-C, neither twin could have a cognitive or physical condition that would preclude completion of the assessment (as assessed via parental screen; e.g., a significant developmental delay). Children provided informed assent, while parents provided informed consent for themselves and their children. The twins were 48.7% female and ranged in age from 6 to 10 years (mean = 8.03, SD = 1.49; although 30 of the 1,027 pairs had turned 11 by the time the family participated).

Although virtually all mothers participated with their twins during the in-person assessment, roughly 5% of fathers completed their questionnaires via the mail. In keeping with the parameterization of the nuclear twin family model (described below), parental self-report data were omitted for those parent figures who did not share 50% of their genes with the twins (i.e., grandmothers and stepfathers), although their reports of the twins were retained. The self-reports of divorced or separated biological parents with joint custody arrangements or who were otherwise involved in their twins’ lives were retained for analysis (note that their exclusion did not alter our conclusions). Our final sample thus included self-reports from 992 biological mothers and 822 biological fathers.

Recruitment procedures are detailed in prior work (Burt & Klump, 2013). In brief, families were recruited directly from birth records, or from a population-based registry that was itself recruited via birth records, via anonymous recruitment mailings in conjunction with the Michigan Department of Health and Human Services. Recruitment procedures for the at-risk sample were identical except that mailings were restricted to those families residing in neighborhoods with Census-level poverty data above the 2008 mean of 10.5% (additional information on neighborhood poverty rates is provided below). This recruitment strategy yielded overall response rates of 62% for the population-based sample and 57% for the at-risk sample. Families participating in the population-based sample endorsed ethnic group memberships at rates comparable to area inhabitants (e.g., White: 86.4%, Black: 5.4%) (Burt & Klump, 2013). Compared to the population-based sample, the at-risk sample was significantly more racially diverse (15% Black, 75% White), reported lower family incomes (the means were \$72,027 and \$57,281, respectively; Cohen’s $d = -.38$), higher paternal felony convictions ($d = .30$), and higher rates of twin conduct problems and hyperactivity ($d = .34$ and $.27$, respectively).

Importantly, both samples appear representative of recruited families, as indexed via a brief questionnaire administered to ~85% of non-participating families (Burt & Klump, 2013). As compared to non-participating twins, participating twins were experiencing equivalent levels

of conduct problems and hyperactivity (d ranged from $-.08$ to $.01$ in the population-based sample and $.01$ to $.09$ in the at-risk sample; all ns). Participating families also did not differ from non-participating families in paternal felony convictions ($d = -.01$ and $.13$ for the population-based and the at-risk samples, respectively), rate of single parent homes ($d = .10$ and $-.01$ for the population-based and the at-risk samples, respectively), paternal years of education (both $d = .12$), or maternal and paternal alcohol problems (d ranged from $.03$ to $.05$ across the two samples). However, participating mothers reported slightly more years of education ($d = .17$ and $.26$ in the population-based and at-risk samples, respectively) than non-participating mothers. Maternal felony convictions were also more common in participating than in non-participating families, but only in the population-based sample ($d = -.20$ in the population-based sample and $.02$ in the at-risk sample).

Zygosity was established using physical similarity questionnaires administered to the twins' primary caregiver (Peeters, Van Gestel, Vlietinck, Derom, & Derom, 1998). On average, the physical similarity questionnaires used by the MSUTR have accuracy rates of 95% or better. The population-based study included 259 monozygotic or MZ pairs (137 male-male and 122 female-female) and 268 dizygotic or DZ pairs (125 male-male, 111 female-female, and 32 opposite-sex pairs). The at-risk study included 165 MZ pairs (86 male-male and 79 female-female) and 335 DZ pairs (85 male-male, 95 female-female, and 155 opposite-sex pairs).

MEASURES

Neighborhood poverty—We collected information on the proportion of neighborhood residents living below the poverty line in each family's census tract from www.Census.gov. Given that all families were recruited from 2008 onwards, we focused here on the 2008-2012 census data. In the population-based sample, 2008-2012 neighborhood poverty rates ranged from 0 to 81%, with a mean of 11.4% (see Supplemental Figure 1). In the at-risk sample, 2008-2012 neighborhood poverty rates ranged up to 93%, with a mean of 23.4% (see Supplemental Figure 1). Note that 16% of the families participating in the at-risk sample ($n=80$) resided in neighborhoods that appear to have 'gentrified' somewhat over the intake recruitment period (e.g., the neighborhood was above the poverty cut-point of 10.5% according to the 2005-2009 Census data available at the time of recruitment, but not according to the 2008-2012 data). In most cases, however, neighborhood poverty rates were higher in the 2008-2012 data than in prior years.

Child Antisocial Behavior—To avoid shared informant variance with parent self-reports of their own antisocial behavior (as described below), teacher-reports of child antisocial behavior served as our primary outcome variable. The twins' teacher(s) completed the Achenbach Teacher Report Form (TRF; Achenbach & Rescorla, 2001), one of the most commonly used instruments for assessing antisocial behaviors prior to adulthood. Teachers rated the extent to which a series of statements described the child's behavior over the past six months using a three point scale (0=never to 2=often/mostly true). In the current study, we focused specifically on the Rule-breaking Behavior (RB) scale (e.g., lies, breaks rules, steals, truant; 12 items; $\alpha = .70$) and the Aggressive Behavior (AGG) scale (e.g., destroys others' things, fights, threatens others, argues, suspicious, temper; 20 items; $\alpha = .93$). The teachers of 115 participants were not available for assessment (because the twins were

home-schooled or because parental consents to contact the teachers were completed incorrectly, etc.). Data collection/data entry with the remaining teacher reports is on-going. As of now, however, our teacher participation rate across the two samples is 79.6%, with teacher reports available for 1,543 participants. Consistent with manual recommendations (Achenbach & Rescorla, 2001), analyses were conducted on the raw scale scores. To adjust for positive skew, data were log-transformed prior to analysis to better approximate normality.

Parental Antisocial Behavior—Parents each completed the Adult Self-Report (ASR; Achenbach & Rescorla, 2003), which includes a fifteen-item AGG scale ($\alpha = .82$) and a fourteen-item RB scale ($\alpha = .69$). Participants were asked to rate the extent to which a series of statements described their behavior over the past six months using a three point scale (0=never to 2=often/mostly true). Consistent with recommendations in the manual (Achenbach & Rescorla, 2003), analyses were conducted on the raw scale scores. To adjust for positive skew, both scales were log-transformed prior to analysis to better approximate normality.

Of note, the ASR AGG and RB scales appear to tap roughly the same constructs as their counterparts on the TRF. In part, this similarity reflects overlapping item content: more than 50% of the items on the TRF AGG and RB scales directly overlap with those on the ASR. The remaining items were often conceptually similar across the two measures (e.g., “truant” on the TRF, “cannot keep job” and on the ASR). Perhaps more importantly, however, validation studies revealed that TRF reports of children’s behavior predict ASR self-reports by those same children as adults. Visser and colleagues (2000), for example, examined a referred sample of 789 young adults participating in a Time 2 assessment after a mean of 10.5 years (Visser, Van der Ende, Koot, & Verhulst, 2000). Results revealed that self-reports of AGG and RB at time 2 (obtained via the ASR) were correlated at least .24 with teacher reports of AGG and RB obtained more than 10 years earlier. Although small, correlations of this magnitude are in fact rather remarkable, in that they are as high as cross-informant correlations obtained concurrently (Achenbach, McConaughy, & Howell, 1987). In short, our primary measures of parental and child antisocial behavior appear to be tapping quite similar constructs.

ANALYSES

Twin studies leverage the difference in the proportion of genes shared between MZ twins (who share 100% of their genes) and DZ twins (who share an average of 50% of their segregating genes) to estimate the relative contributions of genetic and environmental influences (as defined in Table 1) to the variance within observed behaviors or characteristics (phenotypes). More information on twin studies is provided elsewhere (Neale & Cardon, 1992).

GxE models—Prior to our primary nuclear twin family analyses, we first sought to directly replicate the shared environmental moderation reported in the Cleveland and Tuvblad studies. To do so, we fitted the ‘univariate GxE’ classical twin model (Purcell, 2002), separately for child AGG and RB. The univariate GxE model is well-suited for data

in which the twins are perfectly concordant on the moderator (van der Sluis, Posthuma, & Dolan, 2012) and is robust to the identifiability and misspecification issues reported for the bivariate GxE model (Rathouz, Van Hulle, Rodgers, Waldman, & Lahey, 2008). We were not able to directly examine rGE confounds in this model, both because the twins reside in the same neighborhoods (and parent data are not included in this analysis), but also because moderation was modeled specifically on the variance in child antisocial behavior that did not overlap with neighborhood poverty (i.e., the moderator values for each pair are entered in a means model of child antisocial behavior; moderation is then modeled on the residual variance). Poverty was coded as either “low” (0-19.9%; $n=690$ families) or “high” (20+%; $n=$). This cut-point was chosen in accordance with recent work indicating that 20% neighborhood poverty appears to be something of a tipping point, such that the effects of neighborhood poverty on youth outcomes are very small until poverty reaches 20% (Galster, 2010).

Mx (Neale, Boker, Xie, & Maes, 2003) was used to fit the GxE models to the data using Full-Information Maximum-Likelihood (FIML) techniques. When fitting models to raw data, variances, covariances, and means are first freely estimated to get a baseline index of fit (minus twice the log-likelihood; $-2\ln L$). The $-2\ln L$ in the least restrictive GxE model was then compared those that in more restricted GxE models to compute the chi-square index of fit. Non-significant changes in chi-square indicate that the more restrictive model provides a better fit to the data. Model fit was also evaluated using four information theoretic indices that balance overall fit with model parsimony: the Akaike’s Information Criterion (AIC; Akaike, 1987), the Bayesian Information Criteria (BIC; Raftery, 1995), the sample-size adjusted Bayesian Information Criterion (SABIC; Sclove, 1987), and the Deviance Information Criterion (DIC; Spiegelhalter, Best, Carlin, & Van Der Linde, 2002). The lowest or most negative AIC, BIC, SABIC, and DIC among a series of nested models is considered best. Because fit indices do not always agree (they place different values on parsimony, among other things), we reasoned that the best fitting model should yield lower or more negative values for at least 3 of the 5 fit indices. To facilitate interpretation of the unstandardized values (Purcell, 2002), we standardized our log-transformed child AGG and RB scores to have a mean of zero and a standard deviation of one prior to analysis.

Nuclear Twin Family Constraint Models—For our primary analyses, we made use of nuclear twin family models to more fully evaluate how the etiology of child RB varies with the level of neighborhood poverty. By incorporating data on the parents of the twins as well as the twins themselves, the nuclear twin family model (see Supplemental Figure 2) provides four pieces of information on which to base parameter estimates: the covariance between MZ twins, the covariance between DZ twins, the covariance between parents, and the covariance between parents and children. This additional information allows us to estimate several parameters on top of additive genetic and non-shared environmental influences (as defined in Table 1). First, we are able to disambiguate two general types of shared environmental influences: 1) those that create similarity between siblings, but not between parents and their children (termed S; e.g., exposure to common peers, school, and experiences of similar parenting across siblings), and 2) those that are passed via vertical “cultural transmission” between parents and their offspring (termed F; e.g., socioeconomic

status, social mores). The model then allows us to capitalize on this newfound individuation of the various types of shared environmental influences by directly estimating the covariance between F and genetic influences, otherwise known as passive rGE effects (see *w* in Supplemental Figure 2). Finally, the nuclear twin family model allows researchers to directly model and account for the effects of assortative mating on parameter estimates.

The nuclear twin family model thus allows us to more definitively evaluate whether the previously identified shifts in the magnitude of shared environmental influences reflect shifts in the importance of actual environmental experiences rather than shifts in the importance of passive rGE or assortative mating. We specifically fitted the nuclear twin family model separately for families experiencing lower and higher levels of neighborhood poverty, and examined which model (ASFE, ASE, or AFE) provided the best fit to the data at each level of neighborhood poverty. We also ran a series of constraint models to directly evaluate whether we were able to constrain parameter estimates to be equal across the two poverty groups, and evaluated the change in model fit. Significant changes in fit indicated that the parameter could not be constrained to be equal across advantaged and disadvantaged neighborhoods.

Mx, a structural-equation modeling program (Neale et al., 2003), was used to perform the nuclear twin family constraint analyses. Model fit was again evaluated using the χ^2 , the AIC, the BIC, the SABIC, and the DIC, as described above. To address missing data, we made use of FIML raw data techniques. Of note, FIML raw data analyses assume that missing data are missing at random (MAR; i.e., the probability that data are missing is unrelated to their value after controlling for other variables in the data). In essence, MAR allows missingness to depend on other variables in the dataset, but not on variables that are not observed (Allison, 2003; Croy & Novins, 2005). Although the missing mother and child data did appear to be MAR, the missing father did not. Maternal-reports of paternal felony convictions varied with father missingness (4.3% and 26.6% in participating versus non-participating fathers, respectively; $p < .001$). Importantly, however, controlling for the other variables in our various analyses (i.e., maternal and twin antisocial behavior, twin ethnicity, twin age, twin sex, neighborhood disadvantage) appeared to reduce this effect. In a regression of father missingness, the Beta for paternal felony convictions dropped from .32 ($p < .001$) when analyzed alone to .15 ($p = .023$) when analyzed with the other variables. In short, our missing father data may not be fully MAR.

There are several assumptions undergirding the nuclear twin family model. First, although the model accommodates the possibility of assortative mating, it assumes that assortative mating stems from primary phenotypic assortment, in which mates choose each other based on phenotypic similarity, and does not allow for other forms of assortative mating (e.g., social homogamy, in which mates choose each other due to environmental similarity). Second, additive genetic (A) and non-shared environmental (E) estimates are assumed to influence all traits to some extent. However, there is not enough information in the data to simultaneously estimate dominant genetic (D), S, and F effects (in addition to A and E). We are thus required to fix one of these estimates to zero. Given our specific questions, we focused on the ASFE model herein.

RESULTS

Descriptive statistics for AGG and RB are presented in Supplemental Table 1. Paired samples t-tests indicated that fathers were engaging in higher levels of RB relative to mothers ($p < .001$), and somewhat higher levels of AGG ($p = .077$). Independent samples t-tests similarly indicated that boys evidenced higher rates of RB and AGG than did girls (Cohen's $d = .29$ and $.31$, respectively; both $p < .001$). Twin RB and AGG also varied by ethnicity, such that they were less common in White participants than in non-White participants ($d = -.39$ and $-.35$, respectively; both $p < .001$). RB also decreased slightly with age ($r = -.10$, $p < .001$). Ethnicity, sex, and age were regressed out of the twin data prior to analysis (McGue & Bouchard, 1984). Neighborhood poverty (as measured continuously) was modestly correlated with maternal and paternal self-reports of their own RB ($r = .08$ and $.10$, respectively; both $p < .01$), but not with their AGG (both $r = .00$). Neighborhood poverty was also associated with teacher-reports of child RB and AGG ($r = .17$ and $.10$, respectively, both $p < .001$).

Correlations

A preliminary indication of etiologic moderation can be gleaned from the twin intraclass correlations. For child AGG, there was relatively little evidence that MZ and DZ twin similarity shifted with increasing levels of neighborhood poverty (low poverty: $r_{MZ} = .54$, $r_{DZ} = .34$; high poverty $r_{MZ} = .67$, $r_{DZ} = .31$). In both cases, the MZ correlation was significantly larger than the DZ correlation, indicating strong genetic influences. For child RB, however, the MZ and DZ correlations did appear to shift with increasing neighborhood poverty (low poverty: $r_{MZ} = .51$, $r_{DZ} = .23$; high poverty $r_{MZ} = .61$, $r_{DZ} = .46$). The MZ correlation was roughly double that of the DZ correlation in wealthy and middle-class neighborhoods (a significant difference at $p < .05$), indicating that RB may be largely genetic in origin in more advantaged neighborhoods. In impoverished neighborhoods (20+%), by contrast, shared environmental influences appeared prominent, as evidenced by statistically equivalent MZ and DZ correlations ($z = 1.46$, $p = .14$).

GxE model results

We confirmed these impressions via formal tests of etiologic moderation (Purcell, 2002). Model fit statistics are reported in Table 2. The no moderation model provided a better fit to the AGG data by 3 of the 5 fit indices, arguing against the presence of significant etiologic moderation. Moreover, even when estimated, the pattern of (non-significant) moderation pointed to increasing genetic influences and decreasing shared environmental influences with increasing poverty (the genetic and shared environmental moderators were estimated at $.25$ and $-.26$, respectively), results that are not in keeping with those of prior studies.

By contrast, the linear ACE moderation model provided a better fit than the no moderation to child RB data model using 4 of the 5 fit indices. We then examined specific submodels of the linear moderation model. The C moderation only model provided the best fit to the child RB data, both relative to the full ACE moderation model, and the A and E moderation only models, by all five fit indices. Parameter estimates for the best-fitting C-moderation model indicated that genetic and non-shared environmental parameter estimates were moderate-to-

large in magnitude regardless of neighborhood type (the unstandardized genetic and non-shared environmental variances were .50 and .41, respectively; both $p < .05$). Moreover, as their moderators were constrained to be zero, these genetic and non-shared environmental influences did not shift with the level of neighborhood poverty (note that even when estimated, their moderators were very small, .04 and $-.02$, respectively). Shared environmental influences, by contrast, were estimated at .00 (ns) in wealthy and middle-class neighborhoods and increased dramatically in impoverished neighborhoods (the shared environmental moderator was estimated to be .56 (95% CI = .24, .99), indicating an unstandardized shared environmental variance estimate of .31). This rather large C moderator was observed even in the full ACE moderation model, although it was not significant in that less restrictive model (the moderator was .53).

Nuclear twin family model results

For our primary analyses, we sought to better understand the shared environmental moderation observed for child RB via a series of nuclear twin family models. Parameter estimates for the full ASFE models are presented in Table 3. Genetic influences on child RB were observed to be moderate-to-large in magnitude regardless of the level of neighborhood poverty. Assortative mating also appeared to be present regardless of the level of neighborhood poverty. Shared sibling environmental influences (i.e., S) were small and non-significant in wealthy and middle-class neighborhoods but significant and moderate in magnitude in impoverished neighborhoods. Alternately, family environmental (i.e., F) and passive rGE effects contributed significantly to RB in wealthy and middle-class neighborhoods. Interestingly, the passive rGE effect in in wealthy and middle-class neighborhoods was negatively-signed, indicating that increases in the genetic variance (A) in child RB are associated with decreases in the importance of vertical cultural transmission (F).

To clarify whether the above differences in the etiology of child RB across level of neighborhood poverty were statistically significant, we ran two sets of analyses. We first evaluated which nuclear twin family model (i.e., ASFE, AFE, or ASE) provided the best fit to the RB data at low and high levels of neighborhood poverty, respectively. Model fit results are presented in Table 4. As seen there, the AFE model provided the best fit to the data in wealthy and middle-class neighborhoods (i.e., 0-19.9% poverty) by all 5 fit indices. In sharp contrast, the ASE model provided the best fit to the data in impoverished (20+% poverty) neighborhoods, again by all 5 fit indices. Such results confirmed our overall impressions from Table 3, namely that the etiology of child RB varies significantly with level of neighborhood poverty.

We also ran a series of constraint models (see Supplemental Table 2). As seen there, the fully unconstrained ASFE model provided a better fit to the data than the fully constrained ASFE model, in which all parameter estimates were constrained across neighborhood type, by all 5 fit indices. Such results again point to significant etiologic differences in child RB with level of neighborhood poverty. Additional constraint analyses further revealed that neither F nor S could be individually constrained across neighborhood type without a significant decrement in fit, consistent with the model fit results presented above. Passive

rGE, A, and E, by contrast, could each be constrained across level of neighborhood poverty without a significant decrement in fit. Interestingly, however, visual inspection of the parameter estimates for the best-fitting ASE/AFE models revealed the presence of non-overlapping confidence intervals for the genetic estimates (i.e., .72 (.56, .88) versus .30 (.13, .46)). Such results indicate that genetic influences on RB may in fact be more important in wealthy and middle-class neighborhoods than in impoverished neighborhoods.

Confirmatory analyses

In an effort to evaluate the robustness of our NTFM results, we conducted a constructive replication using community resource availability (i.e., the extent to which physical and social resources such as schools, parks, recreational facilities, and volunteer work are available in the broader community). Although resource availability is moderately correlated with neighborhood poverty, it is also thought to be a more proximal and highly tangible form of neighborhood disadvantage (Henry, Gorman-Smith, Schoeny, & Tolan, 2014). Consistent with this, our measure of resource availability centered on more proximal and nuanced assessments (i.e., neighbor informant-reports) in place of Census-level macro data.

In the current study, neighbor reports were assessed as follows: following the participation of a given at-risk twin family, we sent mailings to 10 randomly-chosen addresses in that family's Census tract, inviting one adult resident per household to complete a survey. When a particular randomly-chosen address was no longer inhabited (i.e., the letter was returned as undeliverable), one attempt was made to find a replacement address. When more than one participating twin family resided in a given Census tract, we continued to recruit only 10 neighborhood informants. The informant-report data were thus identical for all families residing in that neighborhood. This approach resulted in a current sample of 1,804 neighbors (63.2% women; 80.4% White, 11.7% Black, 7.9% other ethnic group membership; average age of 52.4 with a range of 18-95 years). Our response rate was 70%, of which 70% agreed to participate (for a final participation rate of 49%). The average number of informant-reports per neighborhood was 4.39 (SD = 1.64), and at least one is currently available for 492 of the 500 families (only one of the 8 represents actual missing data, as assessments have only just begun for the final 7 neighborhoods). Of note, these data are available only for the at-risk sample (neighbor informant-reports were not collected in the population-based sample).

Informant-reports of neighborhood structural characteristics were assessed via the 13-items Community Resources scale ($\alpha = .74$) on the well-validated Neighborhood Matters questionnaire (Henry et al., 2014). Informant-reports were averaged within neighborhoods to create an overall neighborhood-level index of community resources. Average informant-reports of resources were correlated $-.21$ with Census-reports of neighborhood poverty ($p < .001$). The resources variable was dichotomized at the 50% mark for our NTFM analyses.

Given the much smaller sample size ($N=500$ families versus 1,027 families) and the confirmatory nature of these analyses, we focus here on the overall pattern of parameter estimates and the model fit comparisons of the ASFE, ASE, and AFE models (see Tables 3 and 4)¹. ASFE model parameter estimates again pointed to prominent sibling-level shared environmental influences in neighborhoods with low levels of resources, and significant

family-level environmental influences in neighborhoods with more resources. Consistent with this observation, the AFE model provided the best fit to the data in neighborhoods with high levels of resources (by all 5 fit indices), while the ASE model provided the best fit to the data in neighborhoods with low levels of community resources (again by all 5 fit indices). Genetic influences were again observed to decrease with decreasing resource availability (as indexed by non-overlapping confidence intervals), albeit only in the ASE/AFE models. In short, the examination of neighbor informant-reports of community resources generally confirmed our neighborhood poverty results, indicating that sibling-level shared environmental influences on child RB are important primarily in neighborhoods with higher levels of disadvantage, whereas genetic, familial environmental, and passive rGE influences are particularly important in neighborhoods with higher levels of disadvantage.

Comment

The current study evaluated whether and how neighborhood disadvantage shaped the etiology of child AGG and RB. Although we did not find evidence that neighborhood poverty moderated the etiology of child AGG, we observed consistent differences in the etiology of RB by level of neighborhood poverty and lack of community resources. Familial environmental influences (i.e., cultural transmission from parents to children) were observed almost exclusively in wealthy and middle class neighborhoods, as were passive gene-environment correlations. There was also some evidence that genetic influences on child RB are particularly prominent in wealthy and middle class neighborhoods. In sharp contrast, sibling-level shared environmental influences were estimated to be near zero in the wealthiest neighborhoods, and increased several fold with increasing levels of neighborhood poverty. Critically, this pattern of results persisted even when examining neighbor informant-reports of community resource availability as our measure of neighborhood disadvantage, findings which not only serve to bolster our results for neighborhood poverty but also suggest that our conclusions may extend to neighborhood disadvantage more broadly. Such findings collectively indicate that pervasive neighborhood disadvantage substantively alters the etiology of child RB, serving both to enhance sibling-level environmental influences, but also to suppress the otherwise important roles of genetic influences and vertical cultural transmission on these outcomes.

These results constructively replicate, and meaningfully extend, those of prior studies. Cleveland (2003) and Tuvblad et al. (2006) both reported that shared environmental influences on adolescent antisocial behavior increased with increasing neighborhood disadvantage, results that are fully compatible with those reported here despite the fact that both Tuvblad et al. (2006) and Cleveland (2003) examined samples of adolescents whereas the current study examined participants in middle childhood. Similarly, Tuvblad et al. (2006) found evidence that genetic influences on antisocial behavior decrease with increasing neighborhood disadvantage, results that were partially replicated in the current study. That said, Cleveland (2003) reported shared environmental moderation of AGG in particular, for which we did not find evidence of moderation. Although it remains unclear what might

¹Constraint models did not reveal any significant differences across level of resource availability (results available upon request), likely reflecting the smaller sample sizes in those analyses (n~250 families per cell).

account for this discrepancy, the developmental stage of our respective samples is one possibility (middle childhood versus adolescence). That said, Cleveland (2003) did not examine RB, and thus we cannot know how those results might look.

The current study also substantively adds to our understanding of the way in which neighborhood disadvantage moderates the etiology of child antisocial behavior. Neither the Tuvblad nor the Cleveland studies made use of nuclear twin family models, and thus were unable to disambiguate passive rGE from shared environmental influences. Because of this, it was possible that the increase in C observed in those studies actually reflected the increasing importance of passive rGE. The results of the current study strongly argue against this possibility. Sibling-level shared environmental influences were found to increase rather dramatically with increasing levels of neighborhood poverty and decreasing levels of community resources. Moreover, passive rGE and familial shared environmental influences were all but exclusive to wealthy and middle-class neighborhoods, further arguing against the notion that increasing levels of passive rGE underlay the increase in shared environmental influences observed in prior work. The negative direction of the passive rGE we observed in wealthy and middle-class neighborhoods also represents a novel result: namely, our results indicate that in wealthy and middle-class neighborhoods, children with a lower genetic loading for RB have a higher cultural propensity to engage in RB (and vice versa). Although it remains unclear what that cultural propensity might be, one possibility is that familial SES and social mores exert more of an effect on child RB in the absence of genetic risk for RB. Future work should examine this possibility directly.

Finally, the results of the current study dovetail nicely with prior suggestions that AGG and RB constitute meaningfully different, if correlated, dimensions of youth antisocial behavior (Burt, 2012). Only child RB was consistently moderated by neighborhood disadvantage, whereas etiologic influences on child AGG were relatively unaffected. Such results clearly imply that the etiologic differences between AGG and RB extend beyond previously-reported differences in their basic genetic and environmental architectures (Burt, 2009) to also include differential responsiveness to particular environmental experiences. Given this, we would argue that researchers interested in the causal processes underlying antisocial behavior should be disambiguating the two dimensions whenever possible.

LIMITATIONS

There are some limitations to be considered. First, given the role of development in the etiology of conduct problems (Burt & Neiderhiser, 2009), the current results should be considered specific to middle childhood, and should not be applied to other developmental periods. That said, similar results were reported for adolescent samples in both Cleveland (2003) and Tuvblad et al. (2006), suggesting a general environmental effect of neighborhood disadvantage on youth antisocial behavior. Of note, the finding of a consistent pattern of moderation across child and adolescent studies of antisocial behavior has not been found for other environmental risk factors (Burt, in press), suggesting that neighborhood may represent a more potent, or at least longer term, moderator. Similarly, it remains unclear how child sex might further moderate these associations, although extant work has not found evidence of etiologic differences in antisocial behavior across sex (Burt, 2009a).

Nonetheless, future work should seek to confirm the absence of additional moderation by sex. Next, although the phenotypic associations between parent and child RB and neighborhood poverty were in the expected direction, they were small. However, the presence of a small phenotypic association between the moderator and the outcome has no bearing on the extent of etiologic moderation, with the exception of reducing concerns about possible rGE confounds (van der Sluis et al., 2012).

Finally, although the results presented here highlight distinctions within the overarching construct of antisocial behavior, it is worth noting that AGG and RB demonstrate considerable overlap as well. Previous studies have found that, of those with childhood-onset AGG, roughly 50% also exhibit clinically-significant RB (Hudziak et al., 2003), results that were replicated here ($r = .64$ for teacher report of twin; when squared to index the coefficient of determination, this corresponds to 41% overlap across AGG and RB, respectively). Although this level of overlap may appear incompatible with meaningful differences between these two dimensions of antisocial behavior, AGG and RB are in fact differentially predictive of a wide variety of youth outcomes (as reviewed in prior work; see Burt, 2012). Moreover, we would argue that the presence of significant overlap between AGG and RB is in fact to be expected. The comorbidity of mental disorders, once thought to be the exception, now seems to be the rule (Clark, Watson, & Reynolds, 1995), and has been conceptualized as evidence that core psychopathological processes link separate mental disorders (Kendler, Prescott, Myers, & Neale, 2003; Krueger et al., 2002). In addition to these common processes however, there is also evidence of causal processes that are disorder-specific (see especially Krueger et al., 2002). Accordingly, although our results highlight etiological distinctions between AGG and RB, they are not inconsistent with common etiological influences contributing both to their covariation with each other and to the comorbidity between antisocial behavior and other externalizing spectrum disorders.

CONCLUSIONS

Our findings have several important implications for our understanding of the etiology of antisocial behavior in advantaged and disadvantaged neighborhoods specifically, as well as for our understanding of GxE processes more generally. Indeed, although much has been made of the moderation of genetic influences by measured aspects of the environment, the current study indicates that this process of etiological moderation is not unique to genetic influences. And although the moderation of environmental influences is somewhat less straightforward to interpret than the moderation of genetic influences, prior literature points to two possible routes for these effects. One possibility is that effects of parenting on child outcomes are accentuated in disadvantaged neighborhoods (Gorman-Smith, Tolan, & Henry, 1999; Gorman-Smith, Tolan, Zelli, & Huesmann, 1996), as exposure to a common parenting style is thought to load on S or the sibling-level shared environment rather than on F (since parents are not themselves being parented anymore; see Table 1). This effect of parenting may take the form of protection, in which the parent-child relationship is able to buffer children from the consequences of neighborhood poverty (e.g., increased crime and joblessness), or increased risk, in which the parent-child relationship mirrors and accentuates the experiences in the broader community.

Another possibility (which may or may not co-occur with the first) involves early exposure to environmental contaminants, which are experienced disproportionately by those living in impoverished environments (Perera et al., 2002). Decades of research have highlighted the damaging effects of prenatal and early childhood exposure to common environmental toxicants (e.g., lead, cigarette smoke) on later health outcomes, including youth antisocial behavior (DiFranza, Aligne, & Weitzman, 2004; Mansi et al., 2007; Needleman, Schell, Bellinger, Leviton, & Allred, 1990). Fetuses and children are particularly sensitive to such exposure, both because early disruptions in development can have long-lasting effects (Rice & Barone Jr, 2000), but also because many neurotoxicants are transferred across the blood brain barrier (Neubert & Tapken, 1988; Rodier, 2004). These exposures could contribute to the increase in RB in disadvantaged neighborhoods and may exert powerful enough main effects to obviate the contributions of individual genes. Moreover, we would apriori expect such early exposure to create similarities between siblings (e.g., fetal exposure would be experienced by both twins), but not necessarily between children and their parents, and thus load on S.

The current results also speak to the unique etiology of child RB in wealthy and middle-class neighborhoods, and in those with high levels of community resources. Namely, genetic influences appear to be particularly important for the etiology of RB in advantaged neighborhoods, as are familial environmental influences. Moreover, these genetic and family-level shared environmental influences are themselves negatively-correlated. Such findings point to a distinctive etiology of child RB in advantaged neighborhoods – namely, RB appears to be a function of either high levels of genetic risk, or high levels of family-level C, but not both. Future work should seek to identify the environmental experiences that contribute to RB in advantaged neighborhoods in particular, and to evaluate their negative correlation with genetic risk for RB.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

This project was supported by R01-MH081813 from the National Institute of Mental Health (NIMH) and R01-HD066040 from the Eunice Kennedy Shriver National Institute for Child Health and Human Development (NICHD). The content is solely the responsibility of the authors and does not necessarily represent the official views of the NIMH, NICHD, or the National Institutes of Health. The primary author had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. The authors thank all participating twins and their families for making this work possible.

References

- Achenbach TM, McConaughy SH, Howell CT. Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin*. 1987; 101:213–232. [PubMed: 3562706]
- Achenbach, TM.; Rescorla, LA. Manual for ASEBA School-Age Forms & Profiles. University of Vermont, Research Center for Children, Youth, & Families; Burlington, VT: 2001.
- Achenbach, TM.; Rescorla, LA. Manual for ASEBA Adult Forms & Profiles. University of Vermont, Research Center for Children, Youth, & Families; Burlington, VT: 2003.
- Akaike H. Factor analysis and AIC. *Psychometrika*. 1987; 52:317–332.

- Allison PD. Missing data techniques in structural equation modeling. *Journal of Abnormal Psychology*. 2003; 112:545–557. [PubMed: 14674868]
- Breslau J, Borges G, Saito N, Tancredi DJ, Benjet C, Hinton L, Medina-Mora ME. Migration From Mexico to the United States and Conduct Disorder: A cross-national study. *Archives of General Psychiatry*. 2011; 68:1284–1293. [PubMed: 22147845]
- Bronfenbrenner, U. *The ecology of human development: Experiments by nature and design*. Harvard University Press; Cambridge, MA: 1979.
- Bronfenbrenner, U. Interacting systems in human development. Research paradigms: Present and future. In: Bolger, N.; Caspi, A.; Downey, G.; Moorhouse, M., editors. *Persons in Context: Developmental Processes*. Cambridge University Press; New York, NY: 1988. (pp. 25-49)
- Bronfenbrenner U, Ceci SJ. Nature-nurture reconceptualized in developmental perspective: A bioecological model. *Psychological Review*. 1994; 101:568–586. [PubMed: 7984707]
- Brooks-Gunn J, Duncan GJ, Klebanov PK, Sealander N. Do neighborhoods influence child and adolescent development? *American Journal of Sociology*. 1993; 99:353–395.
- Burt SA. Are there meaningful etiological differences within antisocial behavior? Results of a meta-analysis. *Clinical Psychology Review*. 2009; 29:163–178. [PubMed: 19193479]
- Burt SA. How do we optimally conceptualize the heterogeneity within antisocial behavior? An argument for aggressive versus non-aggressive behavioral dimensions. *Clinical Psychology Review*. 2012; 32:263–279. [PubMed: 22459789]
- Burt SA. The shared environment as a key source of variability in child and adolescent psychiatry. *Journal of Child Psychology and Psychiatry*. 2014; 55:304–312. [PubMed: 24261560]
- Burt, SA. *Child Development Perspectives*. Evidence that the GxE underlying youth conduct problems vary across development. in press
- Burt SA, Klump KL. Etiological distinctions between aggressive and non-aggressive antisocial behavior: Results from a Nuclear Twin Family Model. *Journal of Abnormal Child Psychology*. 2012; 40:1059–1071. [PubMed: 22466619]
- Burt SA, Klump KL. The Michigan State University Twin Registry (MSUTR): An update. *Twin Research and Human Genetics*. 2013; 16:344–350. [PubMed: 23101567]
- Burt SA, Neiderhiser JM. Aggressive versus non-aggressive antisocial behavior: Distinctive etiological moderation by age. *Developmental Psychology*. 2009; 45:1164–1176. [PubMed: 19586186]
- Clark LA, Watson D, Reynolds S. Diagnosis and classification of psychopathology: Challenges to the current system and future directions. *Annual Review of Psychology*. 1995; 46:121–153.
- Cleveland HH. Disadvantaged neighborhoods and adolescent aggression: Behavioral genetic evidence of contextual effects. *Journal of Research on Adolescence*. 2003; 13:211–238.
- Coie, JD.; Miller-Johnson, S.; Bagwell, C. Prevention science. In: Sameroff, AJ.; Lewis, M.; Miller, S., editors. *Handbook of developmental psychopathology*. 2. Kluwer Academic Publishers; Dordrecht, Netherlands: 2000.
- Croy CD, Novins DK. Methods for addressing missing data in psychiatric and developmental research. *Journal of the American Academy of Child & Adolescent Psychiatry*. 2005; 44:1230–1240. [PubMed: 16292114]
- Damm AP, Dustmann C. Does growing up in a high crime neighborhood affect youth criminal behavior? *The American Economic Review*. 2014; 104(6):1806–1832.
- DiFranza JR, Aligne CA, Weitzman M. Prenatal and postnatal environmental tobacco smoke exposure and children's health. *Pediatrics*. 2004; 113(Supplement 3):1007–1015. [PubMed: 15060193]
- Galster, GC. The mechanism(s) of neighborhood effects: Theory, evidence, and policy implications; Paper presented at the ESRC Seminar: Neighborhood Effects: Theory and Evidence"; Scotland, UK. 2010. St. Andrews University
- Goodnight JA, Lahey BB, Van Hulle CA, Rodgers JL, Rathouz PJ, Waldman ID, D'Onofrio BM. A quasi-experimental analysis of the influence of neighborhood disadvantage on child and adolescent conduct problems. *Journal of Abnormal Psychology*. 2012; 121(1):95. [PubMed: 21942334]
- Gorman-Smith, D.; Tolan, PH.; Henry, D. The relation of community and family to risk among urban poor adolescents. In: Cohen, P.; Robins, L.; Slomkowski, C., editors. *Where and When: Influence of Historical Time and Place on Aspects of Psychopathology*. Lawrence Erlbaum Associates; Hillsdale, NJ: 1999. pp. 349-367

- Gorman-Smith D, Tolan PH, Henry D. A developmental-ecological model of the relation of family functioning to patterns of delinquency. *Journal of Quantitative Criminology*. 2000; 16(2):169–198.
- Gorman-Smith D, Tolan PH, Zelli A, Huesmann LR. The relation of family functioning to violence among inner-city minority youths. *Journal of Family Psychology*. 1996; 10:115–129.
- Henry DB, Gorman-Smith D, Schoeny ME, Tolan PH. “Neighborhood Matters”: Assessment of Neighborhood Social Processes. *American journal of community psychology*. 2014; 54(3-4):187–204. [PubMed: 25287739]
- Jencks, C.; Mayer, E. The social consequences of growing up in a poor neighborhood. In: Lynn, L.; McGeary, M., editors. *Inner-city poverty in the United States*. National Academy Press; Washington, D.C: 1990. pp. 111-186
- Johnson W. Genetic and environmental influences on behavior: Capturing all the interplay. *Psychological Review*. in press.
- Kendler KS, Prescott CA, Myers J, Neale MC. The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. *Archives of General Psychiatry*. 2003; 60:929–937. [PubMed: 12963675]
- Klump KL, Burt SA. The Michigan State University Twin Registry (MSUTR): Genetic, environmental, and neurobiological influences on behavior across development. *Twin Research and Human Genetics*. 2006; 9:971–977. [PubMed: 17254439]
- Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M. Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology*. 2002; 111:411–424. [PubMed: 12150417]
- Krueger RF, Moffitt TE, Caspi A, Bleske A, Silva PA. Assortative mating for antisocial behavior: Developmental and methodological implications. *Behavior Genetics*. 1998; 28:173–186. [PubMed: 9670593]
- Leventhal T, Brooks-Gunn J. The neighborhoods they live in: The effects of neighborhood residence on child and adolescent outcomes. *Psychological Bulletin*. 2000; 126:309–337. [PubMed: 10748645]
- Lewontin, R. *Scientific American Library*; New York: 1995. *Human Diversity*
- Ludwig J, Duncan GJ, Hirschfield P. Urban poverty and juvenile crime: Evidence from a randomized housing-mobility experiment. *QUARTERLY JOURNAL OF ECONOMICS*. 2001; 116(2):655–680.
- Ludwig J, Liebman JB, Kling JR, Duncan GJ, Katz LF, Kessler RC, Sanbonmatsu L. What can we learn about neighborhood effects from the Moving to Opportunity experiment. *American Journal of Sociology*. 2008; 114(1):144–188.
- Mansi G, Raimondi F, Pichini S, Capasso L, Sarno M, Zuccaro P, Paludetto R. Neonatal urinary cotinine correlates with behavioral alterations in newborns prenatally exposed to tobacco smoke. *Pediatric Research*. 2007; 61(2):257–261. [PubMed: 17237732]
- McGue M, Bouchard TJJ. Adjustment of twin data for the effects of age and sex. *Behavior Genetics*. 1984; 14:325–343. [PubMed: 6542356]
- Moffitt TE, Caspi A, Rutter M. Measured gene-environment interactions in psychopathology. *Perspectives on Psychological Science*. 2006; 1:5–27. [PubMed: 26151183]
- Neale, MC.; Boker, SM.; Xie, G.; Maes, HH. *Mx: Statistical Modeling*. 6th. Department of Psychiatry; VCU Box 900126, Richmond, VA 23298: 2003.
- Neale, MC.; Cardon, LR. *Methodology for Genetic Studies of Twins and Families*. Kluwer Academic Publishers; Boston, MA: 1992.
- Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN. The long-term effects of exposure to low doses of lead in childhood: an 11-year follow-up report. *New England Journal of Medicine*. 1990; 322(2):83–88. [PubMed: 2294437]
- Neiderhiser JM, Reiss D, Pedersen N, Lichtenstein P, Spotts EL, Hansson K, Ellhammer O. Genetic and environmental influences on mothering of adolescents: A comparison of two samples. *Developmental Psychology*. 2004; 40:335–351. [PubMed: 15122961]
- Neubert D, Tapken S. Transfer of benzo(a)pyrene into mouse embryos and fetuses. *Archives of Toxicology*. 1988; 62:236–239. [PubMed: 3196162]

- Peeters H, Van Gestel S, Vlietinck R, Derom C, Derom R. Validation of a telephone zygosity questionnaire in twins of known zygosity. *Behavior Genetics*. 1998; 28(3):159–161. [PubMed: 9670591]
- Pennington BF, McGrath LM, Rosenberg J, Barnard H, Smith SD, Willcutt EG, Olson RK. Gene X environment interactions in reading disability and Attention-Deficit/Hyperactivity Disorder. *Developmental Psychology*. 2009; 45:77–89. [PubMed: 19209992]
- Perera FP, Illman SM, Kinney PL, Whyatt EM, Kelvin EA, Shephard P, Rauh VA. The challenge of preventing environmentally related disease in young children: community-based research in New York City. *Environmental Health Perspectives*. 2002; 110:197–204. [PubMed: 11836150]
- Plomin R, DeFries JC, Loehlin JC. Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin*. 1977; 84:309–322. [PubMed: 557211]
- Purcell S. Variance components model for gene-environment interaction in twin analysis. *Twin Research*. 2002; 5:554–571. [PubMed: 12573187]
- Raftery AE. Bayesian model selection in social research. *Sociological Methodology*. 1995; 25:111–163.
- Raine A. Biosocial studies of antisocial and violent behavior in children and adolescents: A review. *Journal of Abnormal Child Psychology*. 2002; 30:311–326. [PubMed: 12108763]
- Rathouz PJ, Van Hulle CA, Rodgers JL, Waldman ID, Lahey BB. Specification, testing, and interpretation of gene-by-measured-environment interaction models in the presence of gene-environment correlation. *Behavior Genetics*. 2008; 38:301–315. [PubMed: 18293078]
- Rice D, Barone S Jr. Critical periods of vulnerability for the developing nervous system: evidence from humans and animal models. *Environmental Health Perspectives*. 2000; 108(Suppl 3):511. [PubMed: 10852851]
- Rodier PM. Environmental causes of central nervous system maldevelopment. *Pediatrics*. 2004; 113:1076–1083. [PubMed: 15060202]
- Rutter M, Moffitt TE, Caspi A. Gene-environment interplay and psychopathology: Multiple varieties but real effects. *Journal of Child Psychology and Psychiatry*. 2006; 47:226–261. [PubMed: 16492258]
- Rutter M, Silberg J, O'Connor TJ, Simonoff E. Genetics and child psychiatry: I Advances in quantitative and molecular genetics. *Journal of Child Psychology and Psychiatry*. 1999a; 40:3–18. [PubMed: 10102724]
- Rutter M, Silberg J, O'Connor TJ, Simonoff E. Genetics and child psychiatry: II Empirical research findings. *Journal of Child Psychology and Psychiatry*. 1999b; 40:19–55. [PubMed: 10102725]
- Sampson RJ. Moving to inequality: Neighborhood effects and experiments meet structure. *AJS; American journal of sociology*. 2008; 114(11):189.
- Sampson RJ, Raudenbush SW, Earls FJ. Neighborhoods and violent crime: A multilevel study of collective efficacy. *Science*. Aug.1997 277:918–924. [PubMed: 9252316]
- Scarr S. Developmental theories for the 1990s: Developmental and individual differences. *Child Development*. 1992; 63:1–19. [PubMed: 1343618]
- Sclove LS. Application of model-selection criteria to some problems in multivariate analysis. *Psychometrika*. 1987; 53(3):333–343.
- Spiegelhalter DJ, Best NG, Carlin BP, Van Der Linde A. Bayesian measures of model complexity and fit. *Journal of the Royal Statistical Society: Series B*. 2002; 64(4):583–639.
- Tremblay RE. Developmental origins of disruptive behaviour problems: The 'original sin' hypothesis, epigenetics and their consequences for prevention. *Journal of Child Psychology and Psychiatry*. 2010; 51:341–367. [PubMed: 20146751]
- Turkheimer E, Haley A, Waldron M, D'Onofrio B, Gottesman II. Socioeconomic status modifies heritability of IQ in young children. *Psychological Science*. 2003; 14:623–628. [PubMed: 14629696]
- Tuvblad C, Grann M, Lichtenstein P. Heritability for adolescent antisocial behavior differs with socioeconomic status: Gene-environment interaction. *Journal of Child Psychology and Psychiatry*. 2006; 47:734–743. [PubMed: 16790008]
- van der Sluis S, Posthuma D, Dolan CV. A note on false positives and power in GxE modeling of twin data. *Behavior Genetics*. 2012; 42:170–186. [PubMed: 21748401]

Visser JH, Van der Ende J, Koot HM, Verhulst FC. Predictors of psychopathology in young adults referred to mental health services in childhood or adolescence. *British Journal of Psychiatry*. 2000; 177:59–65. [PubMed: 10945090]

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 1

Definitions of the parameters obtained via twin modeling.

Parameter	Model	Definition
A	CTM NTFM	Additive genetic variance = the effect of individual genes summed over loci; acts to increase familial correlations (between twin siblings and between parents and their biological children) relative to the proportion of genes shared; if acting alone, creates MZ correlations that are twice the magnitude of DZ correlations
C	CTM	Shared environmental variance = environmental influences common to family members that act to make them similar to each other regardless of the proportion of genes shared; if acting alone, creates equivalent MZ and DZ correlations.
S	NTFM	Sibling environmental influences = those shared environmental influences that create similarity between siblings (e.g., exposure to common peers, schools, and parenting styles), but not between parents and their children
F	NTFM	Familial environmental variance = shared environmental influences passed via vertical “cultural transmission” between parents and their offspring (e.g., socioeconomic status, social mores); create similarities between siblings and between parents and their children
E	CTM NTFM	Non-shared environmental variance = environmental influences that serve to differentiate family members regardless of the proportion of genes shared; measurement error is also included here
w	NTFM	Covariance between additive genetic and familial environmental effects = reflects the extent to which the familial environment is a function of the genetically influenced preferences/ tendencies of the parent; also referred to as a passive gene-environment correlation
μ	NTFM	Assortative mating copath = spousal similarity on the trait in question; is assumed to be a function of primary phenotypic assortment, whereby mates choose each other based on phenotypic similarity

Note. Some parameters can be obtained only in the classical twin model (CTM)), others can be obtained only in the nuclear twin family model (NTFM; see Supplemental Figure 2), and others can be obtained in both models.

* Dominant or non-additive genetic influences are not estimated in the current paper, and are thus omitted here

Table 2

GxE Fit Indices

Model	-2lnL	df	χ^2 (df)	AIC	BIC	SABIC	DIC
<u>Moderation of child AGG</u>							
Linear ACE moderation	4155.84	1519	--	1117.84	-3020.57	-608.68	-1624.70
No moderation	4165.64	1522	9.81 (3)*	1121.64	-3025.74	-609.09	-1627.12
<u>Moderation of child RB</u>							
Linear ACE moderation	4164.96	1519	--	1126.96	-3016.01	-604.12	-1620.14
No moderation	4179.99	1522	15.03 (3)*	1135.99	-3018.57	-601.91	-1619.94
Linear A moderation only	4167.89	1521	2.93 (1)	1125.89	-3021.26	-606.19	-1623.55
Linear C moderation only	4165.08	1521	0.12 (1)	1123.08	-3022.66	-607.60	-1624.96
Linear E moderation only	4177.63	1521	12.67 (1)*	1135.63	-3016.39	-601.32	-1618.68

Note. AGG and RB represent aggressive and non-aggressive antisocial behavior, respectively. χ^2 indicates the change in chi-square relative to the full ASFE model (* indicates a significant change at $p < .05$). The best fitting model for a given set of analyses is highlighted in bold font, and is indicated by a non-significant change in χ^2 relative to the linear ACE moderation model and/or the lowest or most negative AIC (Akaike's Information Criterion), BIC (Bayesian Information Criterion), SABIC (sample size adjusted Bayesian Information Criterion), and DIC (Deviance Information Criterion) values for at least 3 fit indices.

Table 3

Unstandardized nuclear twin family design heritability estimates for non-aggressive rule-breaking by level of neighborhood poverty (upper part of the table) and community resource availability (lower part of the table).

Level of Neighborhood Poverty	Model	A	S	F	Passive rGE	Assortative mating	E
Neighborhood Poverty of 0-19.9%	ASFE	.68* (.39, .88)	.02 (.00, .16)	.12* (.03, .20)	-.19* (-.26, -.08)	.19* (.12, .23)	.41* (.34, .50)
	AFE	.72* (.56, .88)	---	.13* (.07, .20)	-.20* (-.26, -.14)	.19* (.12, .27)	.40* (.34, .49)
Neighborhood Poverty of 20+%	ASFE	.55* (.13, .98)	.30* (.10, .52)	.03 (.00, .15)	-.08~ (-.22, .009)	.15~ (-.003, .31)	.39* (.29, .54)
	ASE	.30* (.13, .46)	.40* (.26, .56)	---	---	.15~ (-.006, .31)	.45* (.35, .58)
More availability	ASFE	.82* (.35, 1.24)	.08 (.00, .30)	.17* (.02, .36)	-.24* (-.40, -.06)	.27* (.14, .41)	.31* (.22, .44)
	AFE	.98* (.71, 1.26)	---	.24* (.12, .38)	-.31* (-.40, -.20)	.27* (.14, .41)	.30* (.22, .43)
Less availability	ASFE	.30~ (.00, .87)	.33* (.10, .58)	.00 (.00, .11)	-.02 (-.19, .06)	.21* (.06, .36)	.43* (.29, .64)
	ASE	.23* (.07, .40)	.35* (.21, .51)	---	---	.21* (.06, .36)	.45* (.34, .59)

Note. Additive genetic, environmental influences shared between siblings, family environmental influences, and non-shared environmental influences are represented with A, S, F and E, respectively. 95% confidence intervals are presented below the point estimate in brackets. Because A, S, F, and E are variances, neither their estimates nor their confidence intervals can be negatively-signed. The passive rGE and assortative mating estimates can be either positively- or negatively-signed. * indicates that the parameter is significantly greater than zero at $p < .05$. ~ indicates that the parameter is marginally significant at $p < .10$.

Table 4

NTFM fit Indices for non-aggressive rule-breaking (RB), separately by level of neighborhood poverty (upper part of table) and community resource availability (lower part of table)

Model	−2lnL	df	χ^2 (df)	AIC	BIC	SABIC	DIC
<u>0-19.9% neighborhood poverty</u>							
ASFE model	6255.33	2300	--	1655.33	−4381.17	−729.76	−2267.61
ASE model	6268.59	2301	13.26 (1)*	1666.59	−4377.80	−724.80	−2263.32
AFE model	6255.45	2301	0.12 (1)	1653.45	−4384.37	−731.37	−2269.89
<u>20+% neighborhood poverty</u>							
ASFE model	2948.53	1013	--	922.53	−1453.68	152.88	−522.80
ASE model	2950.20	1014	1.67 (1)	922.20	−1455.74	152.42	−523.93
AFE model	2956.77	1014	8.24 (1)*	928.77	−1452.45	155.70	−520.65
<u>More resource availability</u>							
ASFE model	2098.91	767	--	564.91	−1049.16	166.42	−344.33
ASE model	2106.51	768	7.60 (1)*	570.51	−1048.10	169.06	−342.35
AFE model	2099.46	768	0.55 (1)	563.46	−1051.62	165.54	−345.88
<u>Less resource availability</u>							
ASFE model	2205.45	783	--	639.45	−1038.02	202.90	−318.49
ASE model	2205.53	784	0.08 (1)	637.53	−1040.72	201.79	−320.27
AFE model	2213.91	784	8.46 (1)*	645.91	−1036.52	205.98	−316.08

Note. Additive genetic, environmental influences shared between siblings, family environmental influences, and non-shared environmental influences are represented with A, S, F and E, respectively. Analyses were conducted separately for neighborhood poverty and community resource availability. χ^2 indicates the change in chi-square relative to the full ASFE model (* indicates a significant change at $p < .05$). The best fitting model for a given set of analyses is highlighted in bold font, and is indicated by a nonsignificant change in chi-square relative to the linear ACE moderation model and/or the lowest or most negative AIC (Akaike's Information Criterion), BIC (Bayesian Information Criterion), SABIC (sample size adjusted Bayesian Information Criterion), and DIC (Deviance Information Criterion) values for at least 3 of the 5 fit indices.