HOW DO NEIGHBORHOOD, FAMILIAL, AND SCHOOL DISADVANTAGE ALTER THE ETIOLOGY OF CHILDREN'S ANTISOCIAL BEHAVIOR?

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ABSTRACT

HOW DO NEIGHBORHOOD, FAMILIAL, AND SCHOOL DISADVANTAGE ALTER THE ETIOLOGY OF CHILDREN'S ANTISOCIAL BEHAVIOR?

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Disadvantaged contexts come in myriad forms and are widely known to predict antisocial behavior, including both physical aggression and rule-breaking. These predictions go beyond phenotypic associations, with research indicating that genetic and environmental influences on antisocial behavior also vary as a function of neighborhood disadvantage. These findings are interpreted as evidence of a bioecological genotype-environment interaction, such that environmental influences are amplified in impoverished contexts. It is unclear, however, whether the findings related to neighborhood disadvantage apply to familial and school disadvantage. The current study sought to fill this gap by examining multiple forms of disadvantage as etiologic moderators of antisocial behavior in a sample of 1,030 pairs of school-aged twins enriched for disadvantage. Two factors underlay the indicators of disadvantage. Proximal disadvantage, comprising two familial indicators, moderated the etiology of rule-breaking in a way that was consistent with a diathesis-stress model, amplifying the genetic variance, while contextual disadvantage comprised one school and two neighborhood indicators and augmented the effect of the shared environment on rule-breaking, as predicted by the bioecological model. Nuclear twin family model analyses further indicated that this represented a true environmental effect, rather than an increase in passive gene-environment correlation or assortative mating. The indicators of disadvantage had little effect on the etiology of aggression and did not interact with one another as moderators. Implications and future research directions are discussed.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Disadvantaged contexts have consistently been linked to childhood and adolescent antisocial behavior. Although often conceptualized as a unitary construct (e.g., Brooks-Gunn, 1997; Ross, Mirowsky, & Pribesh, 2001), disadvantage comes in at least three broad categories, including neighborhood (e.g., blight, crime, community problems, neighborhood poverty), school (e.g., high student-teacher ratio, high proportion of children getting free lunches), and familial (e.g., familial poverty and low parental educational attainment) disadvantage. Each of these forms of disadvantage has been shown to predict antisocial behavior (Brooks-Gunn, 1997; Christle, Jolivette, & Nelson, 2005; Kupersmidt, Griesler, DeRosier, Patterson, & Davis, 1995), such that children growing up in one or more of these disadvantaged contexts are more often truant, more likely to be rated as aggressive in peer reports, and more likely to be arrested for both violent and nonviolent crimes (Brooks-Gunn, Duncan, Klebanov, & Sealand, 1993; Leventhal & Brooks-Gunn, 2000). Additionally, a study of 1,271 school-aged children found that neighborhood poverty predicted childhood antisocial behavior even after controlling for familial poverty (Kupersmidt et al., 1995), suggesting that the many forms of disadvantage may independently predict behavioral outcomes in unique ways. This possibility is further bolstered by the fact that typical associations among neighborhood, familial, and school disadvantage are relatively small (*r*s .2-.4; Mode, Evans, & Zonderman, 2016).

Importantly, at least some of these predictions go beyond simple phenotypic associations, with research now indicating that genetic and environmental influences on antisocial behavior also vary as a function of disadvantage, with largely consistent findings across all studies conducted to date (Cleveland, 2003; Tuvblad et al., 2006; Burt et al., 2016). As one representative example, Burt and colleagues examined more than 1,000 twin pairs who had been oversampled for neighborhood disadvantage and found that neighborhood poverty (defined as the proportion of neighborhood residents living below the poverty line in each family's census tract) served as an etiologic moderator of non-aggressive antisocial behavior. More specifically, they found that genetic influences on antisocial behavior predominated in wealthy and middleclass neighborhoods, while the shared environment accounted for the majority of the variance in impoverished contexts (Burt, Klump, Gorman-Smith, & Neiderhiser, 2016). Results were fully replicated when neighborhood disadvantage was defined in terms of access to community resources, as assessed using neighbor-informant reports.

This pattern of findings is typically interpreted as evidence of a bioecological genotype-environment interaction (GxE), which predicts that genetic influences will be most strongly expressed in 'average, expectable environments' and stymied in impoverished contexts (Bronfenbrenner & Ceci, 1994). This model is best illustrated by Lewontin's analogy of genetically variable seeds that are planted in either nutrient-deprived or nutrient-rich soil. The environmental adversity conferred by the deprived soil should eventuate in a field populated largely by short plants, regardless of their genetic predisposition for height. 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). Only in the absence of these risks can genetically mediated individual differences fully manifest.

One key limitation to extant GxE studies, however, is that they have almost always restricted their analyses to only a single form of disadvantage to the exclusion of other, related forms of disadvantage. Specifically, Burt et al. (2016) and Cleveland (2003) focused exclusively on neighborhood disadvantage. Tuvblad et al. (2006) included both neighborhood and familial disadvantage, examining moderation by each measure of disadvantage and confirming findings across all indices. Critically, however, they did not examine which form of disadvantage (if either) may drive these effects, nor did they evaluate the possibility of joint moderation by neighborhood and familial disadvantage. Put another way, prior work has yet to try and identify the ‘active ingredients’ of disadvantage that drive its moderation of the etiology of antisocial behavior. Moreover, no study to date has evaluated the etiologic moderation of antisocial behavior by school disadvantage, as defined by school poverty rate and student-teacher ratio. Given the small-to-moderate correlations across the various forms of disadvantage (.2-.4), along with prior findings indicating that the various forms of disadvantage increment each other in the phenotypic prediction of antisocial behavior (Blanz, Schmidt, & Esser, 1991; Ackerman, Schoff, Levinson, Youngstrom, & Izard, 1999; Dodge & Pettit, 2003), these represent key omissions in the extant literature.

Yet another reason why the multiple forms of disadvantage might independently moderate the etiology of antisocial behavior is that this sort of complexity is fully consistent with Bronfenbrenner’s original socio-ecological model (Bronfenbrenner, 1988). According to the model, individual development is embedded in multiple environmental contexts, including family, school, and neighborhood, among others. The microsystem, which refers to the individual’s immediate context, includes the family and school community. The neighborhood is part of the exosystem, or broader social context. The individual interacts differently within each of these contexts, and the contexts themselves may interact when shaping individual development. In short, prior theoretical work also points to the possibility that the types of processes conducive to the development of antisocial behavior differ across neighborhoods, schools, and homes, such that disadvantage in each domain could independently moderate the etiology of antisocial behavior. Alternately, the three forms of disadvantage may serve as joint etiologic moderators, augmenting each other's effects. For example, familial poverty may be more deleterious to youth outcomes when it is embedded in the context of neighborhood poverty. Antisocial behavior in children experiencing multiple forms of disadvantage may thus be especially environmental in origin. No study to date has tested these alternate hypotheses.

The final reason why the multiple forms of disadvantage might independently moderate the etiology of antisocial behavior is that the moderating effects of neighborhood disadvantage appear to depend on the type of antisocial behavior under study. Namely, Burt et al. (2016) found that GxE effects in question varied across aggressive and non-aggressive antisocial behavior, such that findings of GxE were stronger and more consistent for non-aggressive rule-breaking (RB) than for physical aggression (AGG) (Burt et al., 2016; 2018). Such results are consistent with a larger body of work indicating that, although AGG is highly heritable and emerges in early childhood, RB is subject to greater environmental influence and often limited to adolescence (Burt, 2012). Regardless, the fact that GxE appear to vary across correlated forms of antisocial behavior indirectly bolsters the possibility that these GxE might also vary across correlated types of disadvantage.

Current Study

Most relevant GxE studies examining disadvantage as a moderator of antisocial behavior have focused on neighborhood disadvantage. Only one study has examined familial disadvantage as an etiologic moderator of antisocial behavior (Tuvblad et al., 2006), and none have examined school disadvantage. It thus remains unclear whether prior GxE findings for neighborhood disadvantage extend to other forms of disadvantage, and whether the specificity of GxE for RB will persist to other forms of disadvantage. The goal of this study was to fill these gaps in the literature by examining multiple forms of environmental disadvantage as etiologic moderator(s) of AGG and RB in childhood. Using a sample of more than 1,000 twin pairs enriched for neighborhood disadvantage, we first sought to constructively replicate Burt et al. (2016)'s finding that neighborhood disadvantage serves as an etiologic moderator of RB, but not AGG, using alternate measures of neighborhood disadvantage (specifically, we used a composite index of census tract deprivation as well as neighbor-informant reports of neighborhood problems). We then evaluated whether the same pattern emerged for familial and school disadvantage, and whether the various forms of disadvantage synergistically moderated the etiology of antisocial behavior. Based on prior research, we hypothesized that each measure of disadvantage would independently and synergistically moderate the etiology of RB, such that environmental influences would be amplified in high-risk environments.

METHODS

Participants

Participants were drawn from 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). The TBED-C includes both a population-based sample (N = 528 families) and an independent at-risk sample for which inclusion criteria also specified that participating twin families lived in neighborhoods with neighborhood poverty levels at or above the Census mean at study onset (10.5%) (N = 502 families). 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. The TBED-C was approved by the Michigan State University IRB (#04-887, entitled "Genotype-environment interactions in child conduct problems"). Children provided informed assent, while parents provided informed consent for themselves and their children.

The Department of Vital Records in the Michigan Department of Health and Human Services (formerly the Michigan Department of Community Health) identified twins in our specified age-range via the Michigan Twins Project, a large-scale population-based registry of twins in lower Michigan that were recruited via birth records. The Michigan Bureau of Integration, Information, and Planning Services database was used to locate family addresses no more than 90-120 miles of East Lansing, MI through parent driver's license information. Premade recruitment packets were then mailed on our behalf by the Michigan Department of Health and Human Services to parents. A reply postcard was included for parents to indicate their interest in participating. Interested families were contacted directly by project staff. Parents who did not respond to the first mailing were sent additional mailings approximately one 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 57% for the at-risk sample and 63% for our population-based sample, which are similar to or better than those of population-based twin registries that use anonymous recruitment mailings (Baker, Barton, & Raine, 2002; Hay, McStephen, Levy, & Pearsall-Jones, 2002). A brief questionnaire was completed by families participating in the Michigan Twins Project, from which this sample was recruited, thereby allowing us to not only compare families in the at-risk and population-based samples at the time of recruitment, but perhaps more importantly, to compare families who chose to participate versus those who were recruited but did not participate. Compared to the population-based sample, the at-risk sample reported lower mean family incomes ($72,027 and $57,281, respectively; Cohen's *d* effect size = -.38), higher paternal felony convictions (*d* = .30), and higher rates of youth conduct problems and hyperactivity (*d* = .34 and .27, respectively), although they did not differ in youth emotional problems (*d* = .08, *n.s.*). However, both samples were largely representative of non-participating families. As compared to non-participating twins, participating twins were experiencing similar levels of conduct problems, emotional symptoms, or hyperactivity (*d* ranged from -.08 to .01 in the population-based sample and .01 to .09 in the at-risk sample; all n.s.). 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 $d \leq .12$), or maternal and paternal alcohol problems (*d* ranged from .03 to .05 across the two samples). However, participating mothers in both samples reported slightly more years of education (*d* = .17 and .26, both $p < .05$) than non-participating mothers. Maternal felony convictions differed across participating and non-participating families in the population-based sample ($d = -.20$; $p < .05$) but not in the at-risk sample ($d = .02$). All told, we do not believe these differences significantly compromise the generalizability of these data.

The twins in the TBED-C ranged in age from 6 to 10 years (mean = 7.7, SD = 1.51; although 27 pairs had turned 11 by the time the family participated) and were 48.7% female. Families were somewhat more racially diverse than the local area population (e.g., 10% Black and 82% White versus 5% Black and 85% White). 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 at least 95% when compared to DNA. The current study included 224 monozygotic (MZ) male pairs, 211 dizygotic (DZ) male pairs, 202 MZ female pairs, 206 DZ female pairs, and 187 DZ opposite-sex pairs.

Disadvantage

We examined three indicators of disadvantage: neighborhood, familial, and school. Each is detailed below. Sample sizes for each measure of disadvantage are shown in Table 1.

<u>Neighborhood</u>

Neighborhood disadvantage was assessed in two ways: using a composite index of census tract disadvantage and neighbors' reports of neighborhood problems. Kind & Buckingham (2018) constructed an area deprivation index (ADI) comprising 17 measures of socioeconomic disadvantage, including poverty rate, percentage of single-parent households, and income disparity, among others. We recreated this index in our sample using the same measures of disadvantage, assessed from 2008 to 2012. The measures were weighted according to the factor loadings identified by Kind & Buckingham (2018), and the weighted variables were summed to create a deprivation index score for each census tract. Participating families were assigned a percentile score indicating the level of deprivation in their census tract relative to that of all census tracts in Michigan.

Data were also collected from neighbors of participating families in the at-risk sample. We specifically recruited 10 randomly chosen neighbors residing in each family's census tract to complete a survey regarding their perceptions of structural disadvantage in their community. Neighbors completed the 13-item Extent of Neighborhood Problems scale (α=.95), in which they reported whether issues such as graffiti, drugs, and violent crime were problems in their community using a 5-point Likert scale (1= strongly agree to 5= strongly disagree). Responses were reverse-coded so that higher scores on this scale thus indicated greater disadvantage. We then geocoded and mapped neighbor and twin family addresses using ArcGIS v10.3. Average perceptions of neighborhood disadvantage, based on reports from neighbors residing within a 5km radius of participating families, were calculated for each family (in both the population-based and the at-risk samples). The mean number of neighbors living within 5km of a given twin family was 13.09 (SD = 10.98), with a median of 10 and a range of 1 to 47.

<u>Familial</u>

Assessment of disadvantage at the family-level was based on maternal reports of total annual household income and educational attainment. Household income was measured on a 10point Likert scale (1=less than $10,000 to 10=greater than $50,000). Maternal educational attainment was also measured on a 10-point Likert scale (1=less than seventh grade; 2=junior high school; 3=partial high school; 4=high school graduate; 5=trade school; 6=some college; 7=Associate's degree; 8=Bachelor's degree; 9=Master's degree; 10=Advanced degree).

School

School disadvantage was assessed via publicly available records regarding school performance. The National Center for Education Statistics classifies schools as impoverished based on the percentage of students qualifying for subsidized lunch ("Concentration of Public School Students Eligible for Free or Reduced-Price Lunch", 2018). Because there are relatively few studies using school indicators of disadvantage, we used the NCES standard when selecting these indicators. We are defining school disadvantage here via a combination of subsidized lunch rate and student-teacher ratio.

Antisocial Behavior

Twins

To maximize the number of participants with available data, we used a combination of teacher and maternal reports of child antisocial behavior 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 in children and adolescents. 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; α=.70) and the Aggressive Behavior (AGG) scale (e.g., destroys others' things, fights, threatens others, argues, suspicious, temper; 20 items; α=.93). The twins' mothers completed the Child Behavior Checklist, which also includes separate scales for rule-breaking (17 items; α=.85) and aggressive (18 items; α=.94) conduct problems (Achenbach & Rescorla, 2001). Behaviors during the preceding six months were rated using the three-point scale described above.

Maternal informant-reports were available for 99% of the twins. 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.). Our teacher participation rate across the two samples was 86%, with teacher reports available for 1,551 participants. When maternal and teacher reports were combined, data were available for 2,053 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. Additionally, sex and age were regressed out of the data, consistent with prior recommendations (McGue & Bouchard, 1984).

Parents

Biological parents each completed the Achenbach 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. Data were available for 987 biological mothers and 830 biological fathers.

Of note, the ASR scales appear to tap roughly the same constructs as their counterparts on the TRF and CBCL. In part, this similarity reflects overlapping item content: more than 50% of the items on the TRF and CBCL AGG and RB scales directly overlap with those on the ASR. The remaining items are conceptually similar across the two measures (e.g., “truant” on the TRF and CBCL, “cannot keep job” 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, 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 measures of parental and child antisocial behavior appear to be tapping quite similar constructs.

Data Analyses

Using M*plus* 8.0 (Muthén & Muthén, 2019), we first conducted a confirmatory factor analysis (CFA) to clarify the underlying relationship among the indicators of disadvantage. Fit was evaluated with four indices: the Akaike information criterion (AIC; Akaike, 1987), Bayesian information criterion (BIC; Raftery, 1995), sample-size adjusted Bayesian information criterion (SABIC; Sclove, 1987), and root mean square error of approximation (RMSEA; Hooper, Coughlan, & Mullen, 2008). For all indices, lower values indicate better model fit. The best-fitting model was indicated by the lowest or most negative AIC, BIC, SABIC, and RMSEA values for at least three of the four fit indices.

The genetically informed GxE analyses leveraged the differing degrees of genetic similarity between identical (MZ) and fraternal (DZ) twin pairs to determine the genetic and environmental contributions to childhood antisocial behavior, as well as the extent to which these values shift with increasing levels of disadvantage. Using the factor scores derived above as continuous indicators of disadvantage, we then fitted the "univariate GxE" classical twin model (Purcell, 2002) for AGG and RB, respectively, consistent with prior research indicating distinct etiologies for the two primary dimensions of youth antisocial behavior (Burt, 2012). Continuous moderator values were floored to 0, with a maximum of 1. These analyses yielded estimates of the additive genetic (A), shared environmental (C), and nonshared environmental (E) contributions to antisocial behavior. The best-fitting moderation model, based on the AIC, BIC, and SABIC statistics, indicated which of the three parameters, if any, shifted with increasing disadvantage.

Because parent data were not included in these models, however, we could not account for the potentially confounding effects of assortative mating or passive gene-environment correlation (rGE). Assortative mating is the tendency to partner with one who is phenotypically similar to oneself, while passive rGE refers to the fact that the environment that parents provide for their biological children may reflect the parents' genetically influenced tendencies. These are key confounds for studies of GxE, and ones that cannot be ruled out when using the classical twin model. They are particularly problematic in GxE studies of disadvantage, because either of these phenomena may inflate estimates of shared environmental influences, limiting our ability to make causal inferences regarding the bioecological model of GxE.

To address these potential confounds, we dichotomized each indicator of disadvantage, coding the most disadvantaged quartile as 1 and the remaining families as 0, and applied a nuclear twin family model (NTFM), which accounts for confounding factors in a way that the classical twin model cannot. By incorporating data on the parents of the twins as well as the twins themselves, the nuclear twin family model 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 the standard additive genetic (A) and non-shared environmental (E) influences. First, we can directly estimate assortative mating in the model and account for its effects accordingly. Second, 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, or passive rGE effects (Keller, Medland, & Duncan, 2010).

There are several assumptions undergirding the NTFM. 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, A and E 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 the need to estimate passive rGE in our analyses, we focused on the ASFE model herein.

At each level of disadvantage, we determined the nuclear twin family model (ASFE, ASE, or AFE) that best fit the data. Fit was assessed using four indices: the AIC, BIC, SABIC, and RMSEA. The best-fitting model was indicated by the lowest or most negative values for at least three of the four fit indices. If the best-fitting model differed at low and high levels of disadvantage for a given index, this would indicate etiologic moderation. We also ran a series of constraint models to determine whether parameter estimates in the ASFE model could be constrained to be equal at low and high levels of disadvantage. Significant changes in fit, using the indices described above, would indicate that the parameters could not be constrained to be equal and therefore, that etiology shifts with increasing disadvantage.

Finally, to evaluate whether the different forms of disadvantage synergistically moderate the etiology of antisocial behavior, we fit an extension of the univariate GxE classical twin model (Purcell, 2002), which allows multiple variables to simultaneously moderate a given outcome and to interact with one another when doing so. (It is not yet possible to fit this model within the nuclear twin family design). Significant decrements in fit when the synergistic moderation terms are constrained to zero indicate joint etiologic moderation. Such a finding would suggest that the moderating effect of one form of disadvantage on etiology is either accentuated or dampened in the presence of an additional form of disadvantage.

RESULTS

Confirmatory Factor Analysis

Correlations were significantly greater than zero among five of the six indicators of disadvantage (see Table 1). Specifically, ADI percentile, neighborhood problems score, and subsidized lunch rate were highly correlated, while income and education were moderately correlated. Small-to-moderate correlations were observed between each of the familial indicators of disadvantage and ADI percentile, neighborhood problems score, and subsidized lunch rate. Student-teacher ratio, by contrast, was not correlated with any of the other indicators, and will thus not be considered further.

Given this pattern of results, we fitted a CFA to the disadvantage data to compare the respective fit of a two-factor and one-factor solution underlying the indicators of disadvantage. The two-factor solution was superior to the one-factor solution by all fit indices (AIC=-1811.57 versus -1697.62, BIC=-1732.59 versus -1623.57, SABIC=-1783.40 versus -1671.22, and RMSEA=.05 versus .16). Results from the better-fitting model are presented in Figure 1. Subsidized lunch rate, ADI percentile, and neighborhood problems score loaded onto one factor, dubbed contextual disadvantage. Income and education loaded onto the other factor, which we subsequently refer to as proximal disadvantage. The contextual and proximal disadvantage factors were correlated .69. Note that this is higher than the observed correlations in Table 1, and reflects the fact that factors are necessarily error-free.

Classical Twin GxE Models

<u>Correlations</u>

To preliminarily evaluate the presence of etiologic moderation, we dichotomized both proximal and contextual disadvantage, coding the most disadvantaged quartile as 1 and the remaining families as 0, and compared the twin intraclass correlations at low and high levels of disadvantage. Correlations are shown in Table 2. For AGG, there was little evidence that MZ or DZ twin similarity shifted with increasing proximal or contextual disadvantage. Regardless of the type or level of disadvantage, the MZ correlation for AGG was substantially larger than the corresponding DZ correlation, indicating prominent genetic influences. For RB, by contrast, twin similarity appeared to shift with increasing levels of each disadvantage indicator. Specifically, MZ twins were more similar to one another when proximal disadvantage was high, while DZ twin similarity was roughly equivalent at low and high proximal disadvantage. For contextual disadvantage, MZ twin similarity increased slightly with greater disadvantage, while DZ twin similarity increased markedly, indicating a possible increase in shared environmental effects.

It is worth noting, however, that the two disadvantage factors did not increment one another in the phenotypic prediction of either RB or AGG. Proximal and contextual disadvantage each independently predicted the occurrence of RB (ß=1.13 and .84, respectively, both $p<.05$) and AGG (ß=.78 and .60, respectively, both $p<.05$), but contextual disadvantage did not explain any of the variance in either RB (ß=.14, *n.s.*) or AGG (ß=.13, *n.s.*) beyond that explained by proximal disadvantage. However, several of the individual disadvantage indicators, which index aspects of disadvantage that are both shared across indices and unique to each index, did evidence joint phenotypic prediction of RB and AGG. Maternal education incremented each contextual indicator in the prediction of RB (ß range: .10-.12; $p<.05$), and each contextual indicator incremented maternal education (all ß were .11; $p<.05$). Moreover, income incremented each of the three contextual indicators in the prediction of RB (ß range: .17-.21; $p<.05$), and each contextual indicator incremented income (ß range: .07-.08; $p<.05$). For AGG, maternal education incremented ADI percentile and neighborhood problems score (ß range: .06-.07; $p<.05$), but not subsidized lunch rate, and each contextual indicator incremented maternal education (ß range: .09-.10; p<.05). Income also incremented each contextual indicator (ß range: .14-.15; p<.05), and neighborhood problems score and subsidized lunch rate incremented income (ß range: .06-.07; p<.05), although ADI percentile did not.

GxE Model Results

We confirmed these impressions through formal tests of etiologic moderation (Purcell, 2002), using participants' factor scores as continuous measures of proximal and contextual disadvantage, respectively. Table 3 contains the model fit statistics for these analyses, and Table 4 contains the parameter estimates for the full and best-fitting linear moderation models. Results for each separate indicator of disadvantage are presented in Supplementary Tables 1 through 4.

Aggression

Because the full ACE moderation model provided a better fit to AGG than the no moderation model did on all three fit indices, we subsequently examined specific submodels, evaluating the effect of proximal disadvantage on each parameter separately and in pairs. The E moderation only model provided the best fit to the data, indicating an increase in nonshared environmental contributions to AGG with increasing proximal disadvantage (moderator=.30, p<.001; Figure 2a). Neither genetic nor shared environmental contributions to AGG significantly shifted with the level of proximal disadvantage, as both moderators could be constrained to zero.

For contextual disadvantage, by contrast, the A moderation only model provided the best fit to the data, according to all fit indices (moderator=.346, p=.002). As seen in Figure 2b, such findings suggest that, as contextual disadvantage increased, so did the additive genetic contribution to AGG. Shared and nonshared environmental influences did not significantly shift with increasing contextual disadvantage as their moderators were constrained to be zero.

Rule-Breaking

In contrast to the above, proximal disadvantage appeared to moderate additive genetic contributions to RB, such that the A only moderation model provided the best fit to the RB data, as indicated by all measures of fit (moderator=.763, p<.001). As shown in Figure 3a, increases in proximal disadvantage appeared to accentuate genetic influences on RB. Neither shared nor nonshared environmental effects varied with the level of proximal disadvantage (both moderators could be constrained to zero).

Contextual disadvantage also moderated the etiology of RB but did so in very different ways. According to all fit indices, shared and nonshared environmental contributions to RB increased with contextual disadvantage (C moderator=1.237, E moderator=.211; both p<.01; Figure 3b). Additive genetic influences on RB were large regardless of the level of disadvantage, and the A moderator could be constrained to zero.

Nuclear Twin Family Models

To confirm that the increase in shared environmental influences on RB with increasing contextual disadvantage represented a true environmental effect, rather than the effect of confounding factors, we next made use of a series of nuclear twin family models. Table 5 contains parameter estimates for the full ASFE models at low and high contextual disadvantage, dichotomized at the 75th percentile. Genetic and nonshared environmental influences were moderate to large in magnitude regardless of the level of contextual disadvantage, and assortative mating was also present at both low and high disadvantage. Although significantly different than zero at both low and high disadvantage, sibling-shared environmental influences (S) were larger in impoverished contexts. By contrast, family environmental (F) and passive rGE effects were significantly different than zero in wealthy and middle-class contexts but were not present when contextual disadvantage was high.

We also ran a series of constraint models to determine whether the parameter estimates could be constrained to be equal at low and high levels of contextual disadvantage. As shown in Table 6, the fully unconstrained ASFE model provided a better fit to the data than did the fully constrained model, in which all parameter estimates were constrained to be equal across low and high disadvantage, by all four fit indices. These results further indicate that the etiology of RB shifts with increasing contextual disadvantage. Additional constraint analyses revealed that S, F, and E could not be individually constrained across level of contextual disadvantage without a decrement in model fit, while A and passive rGE could be, consistent with our earlier results indicating a moderating effect of contextual disadvantage on environmental, but not genetic, contributions to RB.

Joint Moderation Model

As the final step in our analyses, we evaluated whether proximal and contextual disadvantage, operationalized dichotomously, interacted with one another as etiologic moderators of antisocial behavior, using a series of two-moderator models. Results indicated that for both RB and AGG, the synergistic moderation terms could be fixed to 0 without any decrement in fit. Specifically, for RB, the AIC, BIC, and SABIC values for the full moderation model were 5334.18, 5418.08, and 5364.09, respectively, while the values were 5329.22, 5398.32, and 5353.85, respectively, for the model that did not allow for joint moderation. For AGG, the respective AIC, BIC, and SABIC values were 5489.94, 5573.84, and 5519.85 for the full moderation model, and were 5484.13, 5553.22, and 5508.76 for the model without joint moderation. Such results collectively indicate that the effects of proximal and contextual disadvantage on the etiology of antisocial behavior are neither accentuated nor suppressed in the presence of the other; rather, they independently moderate the etiology of antisocial behavior.

DISCUSSION

The aim of the present study was to evaluate the respective and synergistic roles of multiple, correlated forms of environmental disadvantage as etiologic moderators of children's antisocial behavior. We hypothesized that each indicator of disadvantage would independently and synergistically moderate the etiology of RB, consistent with the predictions of the bioecological model, such that RB would be particularly environmental in origin among children living in highly disadvantaged proximal and contextual environments. We further speculated that these changes would be specific to RB and would not extend to AGG.

Underlying the five indicators of disadvantage included in the study were two distinct, yet substantially correlated (r=.69), higher-order factors. (The low correlations between student-teacher ratio and all other measures, including subsidized lunch rate, indicate that it may not be a valid index of disadvantage.) One factor, comprising household income and maternal educational attainment, represented disadvantage within the family, while the other represented disadvantage in the broader context, with moderate-to-high factor loadings for ADI percentile, neighborhood problems score, and subsidized lunch rate. In particular, the high correlation between subsidized lunch rate and ADI percentile (r=.71) suggests that, rather than existing as separate constructs, disadvantage at the school and neighborhood levels may overlap considerably as indicators of concentrated poverty. We labelled the two underlying factors proximal and contextual disadvantage, respectively.

In findings that were partially consistent with our hypotheses, the etiology of AGG remained relatively constant with increasing proximal and contextual disadvantage. For proximal disadvantage, there was a small-to-moderate increase in nonshared environmental variance with increasing disadvantage which, although significant, was substantially smaller than the moderating effect of proximal disadvantage on the etiology of RB, as discussed later. Likewise, contextual disadvantage had only a small moderating effect on the etiology of AGG, in which the additive genetic contribution increased slightly with increasing disadvantage. The parameter estimates for the full ACE moderation model, however, appear to be more consistent with a weak C moderation effect, given that the estimated C moderator was more than three times larger than the estimated A moderator. The detection of an A, rather than C, moderation effect may be related to power, with biometric GxE models underpowered to distinguish between moderation of the genetic and shared environmental components of variance (Hanscombe et al., 2012). Regardless, these results are consistent with prior work identifying AGG as a highly heritable phenotype (Burt, 2012), but one subject to limited etiologic moderation in the context of disadvantage (Burt et al., 2016), and they further support the division of antisocial behavior into two subtypes that evidence distinct developmental patterns and etiologies (Burt, 2009).

For RB, by contrast, proximal and contextual disadvantage each had a substantial effect on etiology, albeit in different directions. As proximal disadvantage increased, so did the additive genetic contribution to RB, a pattern consistent with the predictions of a diathesis-stress model

(Ingram & Luxton, 2005). In this model, which is frequently discussed in studies of psychopathology and disease, environmental stressors are thought to activate individual genetic vulnerabilities to psychological symptoms, such that genetic effects are more pronounced in disadvantaged environments. This is in direct contrast to the predictions of the bioecological model, in which genetic influences are most strongly expressed in ‘average, expectable’ environments and environmental influences amplified in high-risk contexts (Bronfenbrenner & Ceci, 1994). Only one study of GxE prior to this one included familial indicators of environmental disadvantage as moderators of youth antisocial behavior. Tuvblad et al. (2006) evaluated the moderating effects of parental occupational status and educational attainment on antisocial behavior and obtained results that were largely in line with the predictions of the bioecological model.

Importantly, however, participants in the Tuvblad et al. (2006) sample were adolescents aged 16-17. A meta-analysis found a moderate, statistically significant increase per year in the heritability of externalizing behaviors, encompassing antisocial behavior, aggression, conduct disorder, oppositional defiant disorder, and problem behavior, from age 10 until young adulthood (Bergen, Gardner, & Kendler, 2007). Antisocial behavior in our middle-childhood sample may thus be subject to an alternate form of etiologic moderation than it was in Tuvblad et al.'s study, and our participants may be particularly susceptible to the detrimental effects of disadvantage within the family environment. Put another way, it is possible that, in this particular age group, familial disadvantage facilitates the manifestation of genetic tendencies for problem behaviors. Subsequent studies of GxE should examine indicators of familial disadvantage in samples spanning a broad range of ages, as well as employ longitudinal designs to clarify the trajectory of GxE throughout childhood and adolescence.

Contextual disadvantage, by contrast, altered the etiology of RB in a way that was fully consistent with the predictions of the bioecological model. Specifically, as contextual disadvantage increased, so did shared and nonshared environmental contributions to the variance in RB, with a particularly pronounced moderation of the shared environment. To confirm that the increase in shared environmental variance was not the result of confounding by passive rGE and/or assortative mating, we incorporated parents' reports of their own RB and used a nuclear twin family design, the results of which further indicated that the etiology of child RB varied by level of contextual disadvantage. We specifically found evidence of significant genetic, sibling-level shared environmental, and nonshared environmental effects at both low and high disadvantage, and assortative mating was present across contexts. By contrast, familial environmental influences (i.e., vertical cultural transmission from parents to children) and passive rGE were present in wealthy and middle-class contexts (where contextual disadvantage was low) but absent from impoverished ones. Because passive rGE was not present at high contextual disadvantage and assortative mating did not increase in magnitude, our results appear to represent a true environmental effect rather than an increase in confounding, such that environmental factors that increase sibling resemblance for RB do in fact become more pronounced in impoverished contexts. Results from the constraint models further support this conclusion, as additive genetic influences and passive rGE could be constrained to be equal across low and high disadvantage without a significant decrement in model fit, while sibling environmental, family environmental, and nonshared environmental influences could not.

These results both constructively replicate and meaningfully extend prior GxE studies of disadvantage. Cleveland (2003), Tuvblad et al. (2006), and Burt et al. (2016) each reported an increase in shared environmental variance with increasing neighborhood disadvantage, and Burt et al. employed a nuclear twin family design to confirm that results were not due to potential confounding factors. The effect of contextual disadvantage on the etiology of RB identified in the present study, which used the same sample of school-aged twins as used in Burt et al. (but evaluated different indicators of contextual disadvantage), further suggests that these findings extend beyond neighborhood measures to indicators of school disadvantage (e.g., subsidized lunch rate). In other words, disadvantage at the school level may act in much the same way as neighborhood disadvantage in altering the etiology of youth behavioral outcomes in line with the bioecological model, although further GxE research is needed to examine the effects of school indicators beyond subsidized lunch rate.

While Cleveland reported shared environmental moderation of AGG, Burt et al. (2016) found no evidence of etiologic moderation when AGG was the outcome under study. In this study, we found evidence of weak etiologic moderation of child AGG, and our overall pattern of results was consistent with prior findings that RB was subject to stronger, more consistent etiologic moderation than was AGG. The discrepancy between these results and Cleveland's may be related to participant age (middle childhood versus adolescence), although Cleveland did not examine RB, and it is thus unknown whether it would have been subject to etiologic moderation in his sample.

The current study also adds to our understanding of the moderating role of multiple forms of disadvantage considered simultaneously. Contrary to our hypotheses, proximal and contextual disadvantage did not interact as etiologic moderators of either RB or AGG. In other words, the moderating effect of proximal disadvantage was neither amplified nor reduced in the presence of contextual disadvantage (and vice versa). Because the two underlying forms of disadvantage have opposing effects on etiology, particularly with regard to RB, it appears that they act independently of one another as moderators. Moreover, proximal and contextual disadvantage did not increment one another in the phenotypic prediction of AGG or RB, contrary to prior research indicating that neighborhood disadvantage predicted antisocial behavior even after controlling for familial poverty (Kupersmidt et al., 1995). While it remains unclear what might account for these findings, it is noteworthy that proximal disadvantage was a more robust phenotypic predictor of both RB and AGG than was contextual disadvantage in our sample, while contextual disadvantage was a more robust etiologic moderator.

There are several limitations to the present study. First, we used data collected at one time point from a sample of twins in middle childhood, meaning our results may not generalize to other age groups. This is particularly relevant in light of prior meta-analytic work reporting substantial changes in the etiologies of externalizing behaviors from middle childhood through adolescence (Bergen et al., 2007). However, the role of contextual disadvantage in middle childhood identified in this study was consistent with that of neighborhood disadvantage in studies of adolescents (Cleveland, 2003; Tuvblad et al., 2006), suggesting a rather robust moderating effect of concentrated disadvantage throughout development. The moderating role of proximal disadvantage, on the other hand, was quite different than that reported by the only other GxE study to include familial indicators (Tuvblad et al., 2006). Because no other study of GxE has examined familial indicators of disadvantage in a childhood sample, it is unclear to what extent our finding of a diathesis-stress effect is robust both to other samples and to alternate conceptualizations of disadvantage within the home. Future research should seek to clarify the moderating role of familial disadvantage throughout childhood and adolescence.

Next, child gender was regressed out of the data prior to analysis. Prior research has not identified etiologic differences in antisocial behavior by sex (Burt, 2009), and a recent study found that sex and disadvantage, defined as a combination of family poverty, neighborhood poverty, and exposure to community violence, did not serve as joint etiologic moderators (Burt et al., 2018). Because no prior study of GxE has included school indicators of disadvantage, however, it is unclear whether there is in fact joint etiologic moderation of antisocial behavior by school disadvantage and sex. Future work should seek to confirm the absence of joint moderation by sex and alternate forms of disadvantage.

Lastly, although our results emphasize distinctions between two types of antisocial behavior (AGG and RB), it is worth noting the considerable overlap between them. Specifically, child AGG and RB were correlated .70 in our sample. Although this degree of overlap may seem incompatible with meaningful differences, a large body of research supports the existence of AGG and RB as meaningful subtypes of antisocial behavior with distinct etiologies, developmental trajectories, and interpersonal correlates (Burt, 2009; Burt, 2012; Burt et al., 2016). As such, the relatively high correlation between AGG and RB is not inconsistent with our finding that the moderating effects of disadvantage differed across the two domains.

Despite these limitations, the current study advances our understanding of the moderating effects of multiple correlated forms of environmental disadvantage on the etiology of children's antisocial behavior. Our study constructively replicated prior work indicating that environmental influences on child RB are amplified in impoverished neighborhoods and also identified, for the first time, both a moderating effect of school disadvantage that followed the predictions of the bioecological model and a moderating role of familial disadvantage consistent with a diathesis-stress effect. These results underscore the importance of considering disadvantage as a multifaceted, rather than unitary, construct, with each domain exerting specific effects on children's behavioral outcomes. Future studies of GxE should further examine the moderating role of familial and school indicators of disadvantage, as well as seek to clarify the 'active ingredients' of neighborhood disadvantage that underlie the robust moderating effect identified in this study and in prior research.

APPENDICES

APPENDIX A: Tables

Table 1: Sample sizes and correlations among measures of disadvantage.

Measures	Number of twin families			Correlations					
	Total	**MZ**	**DZ**	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. ADI	972	404	568	--					
2. Neighborhood problems	845	332	513	**.55***	--				
3. Income	979	408	571	**.37***	**.32***	--			
4. Maternal education	980	408	572	**.26***	**.15***	**.43***	--		
5. Subsidized lunch rate	815	332	483	**.71***	**.55***	**.32***	**.27***	--	
6. Student-teacher ratio	880	363	517	.03	.03	.00	.06	.00	--

Note. Bold font and an asterisk indicate that the correlation was significant at $p<.05$.

Table 2: Twin intraclass correlations at low and high disadvantage.

		Low proximal disadvantage	High proximal disadvantage	Low contextual disadvantage	High contextual disadvantage
AGG	*r*MZ	.62	.62	.63	.57
	*r*DZ	.37	.36	.36	.40
RB	*r*MZ	.63	.71	.65	.70
	*r*DZ	.42	.44	.38	.51

Table 3: Biometric GxE fit indices.

Type of disadvantage	Type of antisocial behavior	Model	AIC	BIC	SABIC
Proximal	**AGG**	Linear ACE moderation	5470.13	5509.62	5484.21
		No moderation	5482.50	5507.18	5491.30
		Linear A moderation only	5470.63	5500.24	5481.19
		Linear C moderation only	5476.26	5505.88	5486.82
		Linear E moderation only	**5469.82**	**5499.43**	**5480.37**
Contextual	**AGG**	Linear ACE moderation	5490.43	5529.92	5504.51
		No moderation	5496.97	5521.64	5505.76
		Linear A moderation only	**5488.56**	**5518.17**	**5499.12**
		Linear C moderation only	5490.70	5520.31	5501.26
		Linear E moderation only	5491.55	5521.16	5502.10
Proximal	**RB**	Linear ACE moderation	5308.60	5348.09	5322.68
		No moderation	5357.41	5382.09	5366.21
		Linear A moderation only	**5305.30**	**5334.91**	**5315.86**
		Linear C moderation only	5321.01	5350.62	5331.56
		Linear E moderation only	5333.22	5362.83	5343.78
Contextual	**RB**	Linear ACE moderation	5360.26	5399.74	5374.33
		No moderation	5390.05	5414.73	5398.85
		Linear A moderation only	5364.38	5394.00	5374.94
		Linear C moderation only	5364.29	5393.90	5374.85
		Linear E moderation only	5374.74	5404.36	5385.30
		Linear C and E moderation	**5358.42**	**5392.97**	**5370.74**

Note. The best-fitting model for a given set of analyses is highlighted in bold font, and is indicated by the lowest AIC, BIC, and SABIC values for at least 2 of the 3 fit indices.

Table 4: Unstandardized path and moderation parameter estimates for the full linear moderation and best-fitting moderation models.

Type of disadvantage	Type of antisocial behavior	Model	Paths			Linear Moderators		
			a	c	e	A_1	C_1	E_1
Proximal	**AGG**	Linear ACE moderation	**.67***	.25	**.54***	.20	.16	.20
		Linear E moderation only	**.71***	**.31***	**.51***	-	-	**.30***
Contextual	**AGG**	Linear ACE moderation	**.69***	.14	**.55***	.11	.38	.13
		Linear A moderation only	**.60***	**.29***	**.59***	**.35***	-	-
Proximal	**RB**	Linear ACE moderation	**.55***	.25	**.52***	**.61***	.25	.09
		Linear A moderation only	**.49***	**.32***	**.54***	**.76***	-	-
Contextual	**RB**	Linear ACE moderation	**.73***	-.31	**.46***	.09	**1.25***	**.19***
		Linear C and E moderation	**.75***	-.27	**.46***	-	**1.24***	**.21***

Note. Bold font and an asterisk indicate that the parameter estimate was significantly different than zero at $p<.05$.

Table 5: Unstandardized NTFM variance estimates for RB at low and high levels of contextual disadvantage.

Contextual disadvantage	Model	A	S	F	Passive rGE	Assortative mating	E
Low	ASFE	**.66***	**.14***	**.08***	**-.16***	**.24***	**.26***
		[.42, .88]	[.04, .25]	[.01, .16]	[-.24, -.06]	[.21, .26]	[.20, .34]
High	ASFE	**.53***	**.36***	.01	-.04	**.13***	**.39***
		[.06, 1.0]	[.15, .6]	[.0, .11]	[-.19, .06]	[.11, .15]	[.23, .55]

Note. 95% confidence intervals are below the point estimates in brackets. A, S, F, and E represent the additive genetic influences, environmental influences shared by siblings, family environmental influences, and nonshared environmental influences, respectively. Because A, S, F, and E are variances, neither their estimates nor their confidence intervals can be negatively signed. The passive rGE (gene-environment correlation) and assortative mating estimates can be either positively or negatively signed. Bold font and an asterisk indicate that the parameter is significantly different than zero at $p<.05$.

Table 6: Fit indices for NTFM constraint models.

ASFE Model	AIC	BIC	SABIC	RMSEA
Fully unconstrained	10510.07	10559.43	10527.67	.095
Fully constrained	10551.76	10581.38	10562.32	.109
A constrained	10508.56	10552.99	10524.41	.093
S constrained	***10514.18***	***10558.61***	***10530.02***	***.096***
F constrained	***10514.01***	***10553.50***	***10528.09***	***.095***
E constrained	***10513.58***	***10558.00***	***10529.42***	***.095***
Passive rGE constrained	10511.69	10556.11	10527.53	.095

Note. Bold and italicized font indicates that the parameter value could not be constrained to be equal at low and high levels of contextual disadvantage.

Supplementary Table 1: Biometric GxE fit indices: AGG.

Type of disadvantage	Model	AIC	BIC	SABIC
ADI	Linear ACE moderation	5205.74	5244.78	5219.37
	No moderation	5210.30	5234.70	5218.82
	Linear A moderation only	**5204.16**	**5233.43**	**5214.38**
	Linear C moderation only	5207.84	5237.12	5218.06
	Linear E moderation only	5204.58	5233.86	5214.80
Neighborhood Problems	Linear ACE moderation	4559.85	4597.76	4572.36
	No moderation	4559.32	4583.02	4567.14
	Linear A moderation only	4560.62	4589.05	4570.00
	Linear C moderation only	4560.19	4588.63	4569.57
	Linear E moderation only	**4556.91**	**4585.35**	**4566.29**
Income	Linear ACE moderation	5208.76	5247.85	5222.45
	No moderation	5223.43	5247.86	5231.98
	Linear A moderation only	5212.23	5241.55	5222.49
	Linear C moderation only	5217.89	5247.21	5228.15
	Linear E moderation only	**5207.33**	**5236.64**	**5217.59**
Maternal Education	Linear ACE moderation	5259.49	5298.59	5273.18
	No moderation	**5255.63**	**5280.07**	**5264.19**
	Linear A moderation only	5255.63	5284.96	5265.90
	Linear C moderation only	5256.42	5285.74	5266.69
	Linear E moderation only	5256.72	5286.04	5266.99
Subsidized Lunch Rate	Linear ACE moderation	4385.12	4422.75	4397.34
	No moderation	4389.29	4412.81	4396.93
	Linear A moderation only	4383.35	4411.57	4392.51
	Linear C moderation only	**4381.28**	**4409.50**	**4390.45**
	Linear E moderation only	4389.66	4417.88	4398.82

Note. The best-fitting model for a given set of analyses is highlighted in bold font, and is indicated by the lowest AIC, BIC, and SABIC values for at least 2 of the 3 fit indices.

Supplementary Table 2: Biometric GxE fit indices: RB.

Type of disadvantage	Model	AIC	BIC	SABIC
ADI	Linear ACE moderation	5127.31	5166.34	5140.94
	No moderation	5144.29	5168.68	5152.80
	Linear A moderation only	5134.32	5163.60	5144.54
	Linear C moderation only	**5126.80**	**5156.07**	**5137.02**
	Linear E moderation only	5138.27	5167.55	5148.49
Neighborhood Problems	Linear ACE moderation	4425.15	4463.06	4437.66
	No moderation	4441.61	4465.31	4449.43
	Linear A moderation only	4431.25	4459.69	4440.63
	Linear C moderation only	4433.19	4461.63	4442.57
	Linear E moderation only	4426.66	4455.10	4436.04
	Linear C and E moderation	**4423.15**	**4456.33**	**4434.10**
Income	Linear ACE moderation	5067.24	5106.34	5080.93
	No moderation	5107.89	5132.32	5116.44
	Linear A moderation only	**5063.65**	**5092.97**	**5073.91**
	Linear C moderation only	5081.50	5110.82	5091.77
	Linear E moderation only	5087.17	5116.49	5097.43
Maternal Education	Linear ACE moderation	5140.42	5179.52	5154.11
	No moderation	5151.78	5176.22	5160.34
	Linear A moderation only	5139.03	5168.36	5149.30
	Linear C moderation only	**5137.55**	**5166.88**	**5147.82**
	Linear E moderation only	5151.82	5181.15	5162.09
Subsidized Lunch Rate	Linear ACE moderation	4292.13	4329.76	4304.35
	No moderation	4312.15	4335.67	4319.79
	Linear A moderation only	**4291.03**	**4319.25**	**4300.20**
	Linear C moderation only	4293.34	4321.55	4302.50
	Linear E moderation only	4302.56	4330.77	4311.72

Note. The best-fitting model for a given set of analyses is highlighted in bold font, and is indicated by the lowest AIC, BIC, and SABIC values for at least 2 of the 3 fit indices.

Supplementary Table 3: Unstandardized path and moderation parameter estimates for the full linear moderation and best-fitting moderation models for AGG.

Type of disadvantage	Model	Paths			Linear Moderators		
		a	c	e	A_1	C_1	E_1
ADI	Linear ACE moderation	**.60***	**.41***	**.56***	.23	-.08	.11
	Linear A moderation only	**.58***	**.38***	**.60***	**.28***	**-**	**-**
Neighborhood Problems	Linear ACE moderation	**.84***	.11	**.50***	-.23	.51	**.27***
	Linear E moderation only	**.80***	.22	**.51***	**-**	-	**.23***
Income	Linear ACE moderation	**.71***	**.29***	**.55***	.07	.18	**.19***
	Linear E moderation only	**.72***	**.32***	**.55***	-	-	**.24***
Maternal Education	Linear ACE moderation	**.65***	.39	**.58***	.22	-.14	.02
	No moderation	**.73***	**.34***	**.59***	-	-	-
Subsidized Lunch Rate	Linear ACE moderation	**.70***	.04	**.60***	.03	.66	.02
	Linear C moderation only	**.71***	.02	**.61***	-	**.72***	-

Note. Bold font and an asterisk indicate that the parameter estimate was significantly different than zero at $p<.05$.

Supplementary Table 4: Unstandardized path and moderation parameter estimates for the full linear moderation and best-fitting moderation models for RB.

Type of disadvantage	Model	Paths			Linear Moderators		
		a	c	e	A_1	C_1	E_1
ADI	Linear ACE moderation	**.76***	**-.42***	**.50***	-.02	**1.31***	.13
	Linear C moderation only	**.76***	**-.39***	**.55***	-	**1.30***	-
Neighborhood Problems	Linear ACE moderation	**.79***	.09	**.40***	.01	.76	**.36***
	Linear C and E moderation	**.79***	.09	**.40***	-	**.77***	**.36***
Income	Linear ACE moderation	**.65***	**.31***	**.53***	**.47***	.04	.04
	Linear A moderation only	**.64***	**.32***	**.54***	**.51***	-	-
Maternal Education	Linear ACE moderation	**.70***	.04	**.54***	.17	.76	0
	Linear C moderation only	**.74***	-.01	**.54***	-	**.94***	-
Subsidized Lunch Rate	Linear ACE moderation	**.63***	.14	**.51***	.16	.58	.14
	Linear A moderation only	**.45***	**.39***	**.57***	**.57***	-	-

Note. Bold font and an asterisk indicate that the parameter estimate was significantly different than zero at p<.05.

APPENDIX B: Figures

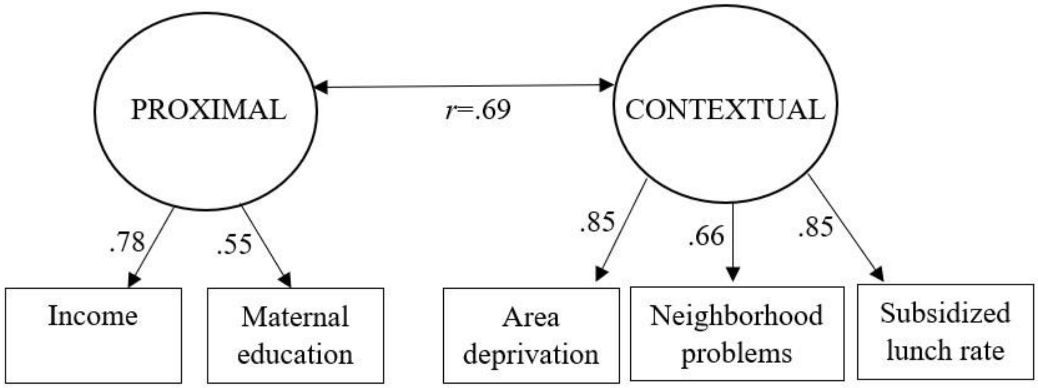
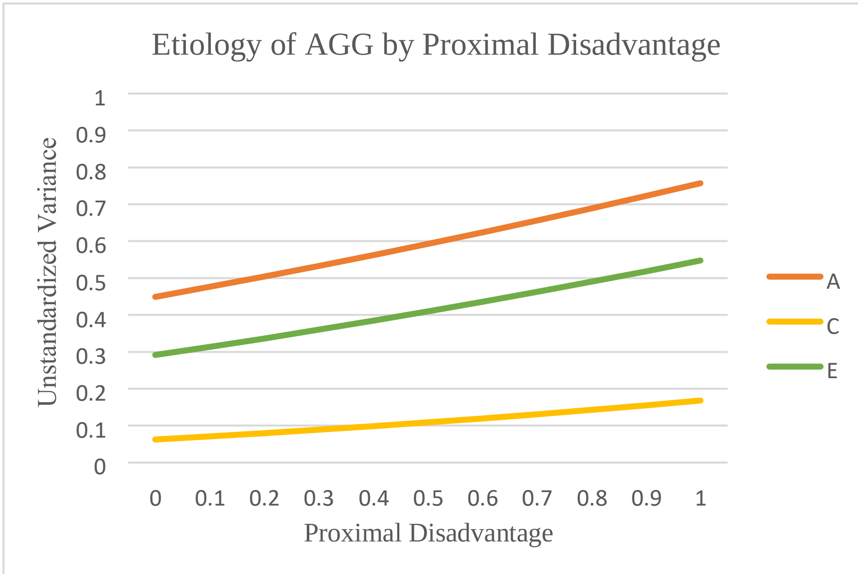
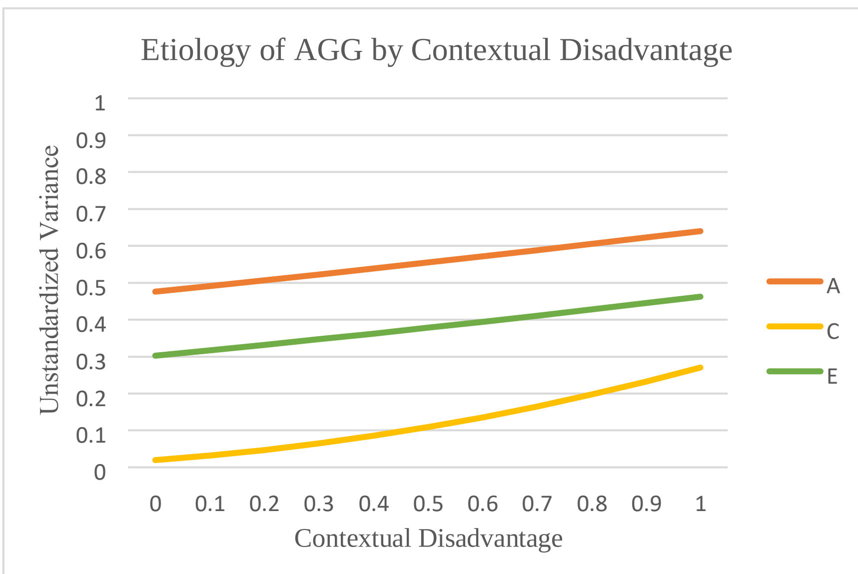


Figure 1: Two-factor confirmatory model of disadvantage indicators.

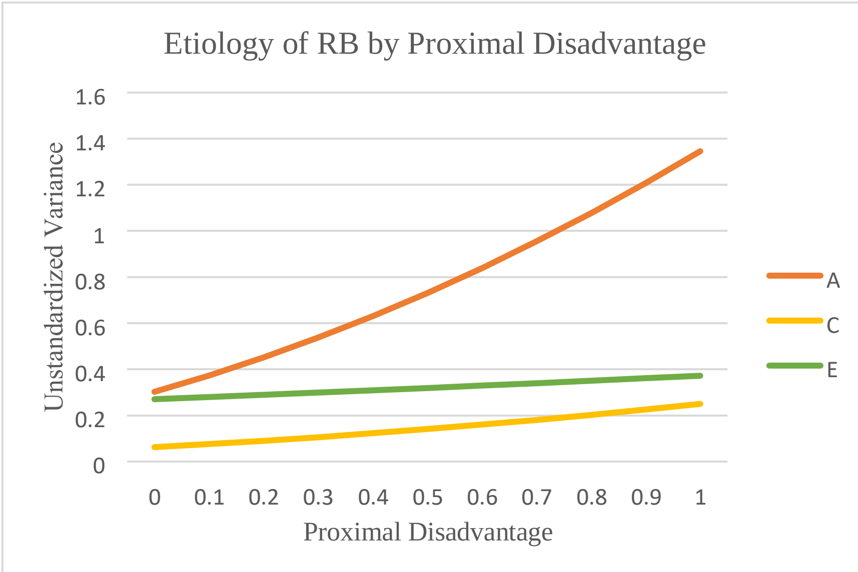


a.



b.

Figure 2: Etiologic moderation of AGG by proximal (a) and contextual (b) disadvantage.



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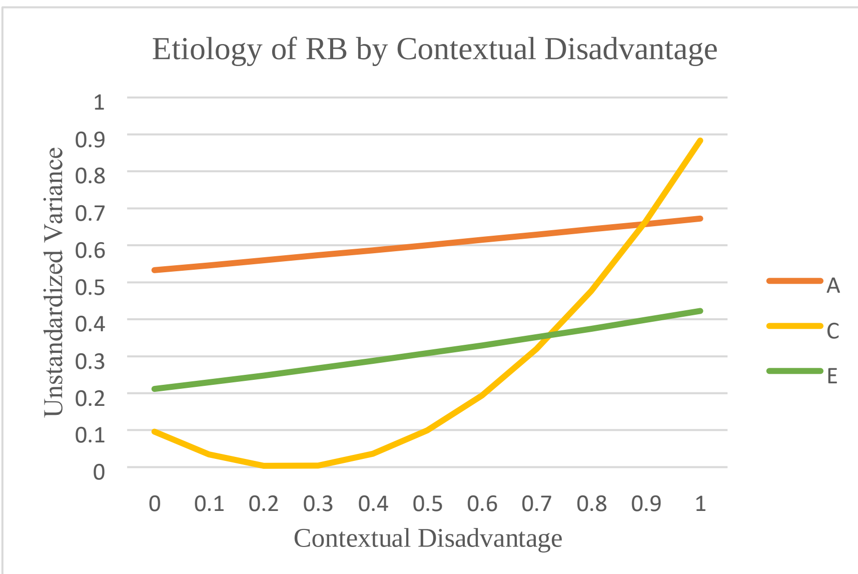

b.

Figure 3: Etiologic moderation of RB by proximal (a) and contextual (b) disadvantage.

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