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Child Antisocial Behavior Is more Environmental in Origin in Disadvantaged Neighborhoods: Confirmatory Evidence Across Residents' Perceptions and Geographic Scales in Two Samples

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Abstract

Prior research has suggested that disadvantaged neighborhood contexts alter the etiology of youth antisocial behavior (ASB). Unfortunately, these studies have relied exclusively on governmental data collected in administratively-defined neighborhoods (e.g., Census tracts or block groups, zip codes), a less than optimal approach for studying neighborhood effects. It would thus be important to extend prior findings of GxE using neighborhood sampling techniques, in which disadvantage is assessed via resident informant-reports of the neighborhood. The current study sought to do just this, examining two independent twin samples from the Michigan State University Twin Registry. Neighborhood disadvantage was assessed via maternal and neighbor informant-reports, the latter of which were analyzed multiple ways (i.e., all neighbors within 1km, nearest neighbor, and all neighbors within the County). Analyses revealed clear and consistent evidence of moderation by neighborhood disadvantage, regardless of informant or the specific operationalization of neighborhood. Shared environmental influences on ASB were observed to be several-fold larger in disadvantaged contexts, while genetic influences were proportionally more influential in advantaged neighborhoods. Such findings indicate that neighborhood disadvantage exerts rather profound effects on the origins of youth ASB. Efforts should now be made to identify the active ingredients of neighborhood disadvantage.

Keywords

Antisocial behavior; GxE; neighborhood disadvantage

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The construct of neighborhood socioeconomic disadvantage, which centers on neighborhood-level characteristics like widespread high unemployment, low household incomes, and high uptake of government assistance programs, has emerged as a robust and long-term contextual risk factor for youth antisocial behavior (ASB), and especially non-aggressive rule-breaking antisocial behaviors like vandalism and theft (Amone-P'Olak, Burger, Huisman, Oldehinkel, & Ormel, 2011; Burt, Klump, Kashy, Gorman-Smith, & Neiderhiser, 2015; Odgers et al., 2012). As one example, Odgers et al. (2012) compared mean levels of ASB across a large sample of children living in deprived versus affluent neighborhoods. They found that the Cohen's *d* standardized mean difference was .38 at age 5 and .51 at age 12, findings that highlight the long-term predictive value of neighborhood disadvantage for youth ASB.

A separate body of literature has simultaneously confirmed that ASB is moderately genetic in origin, with genetic influences accounting for at least 48% of observed individual differences in ASB across the population (Burt, 2009a, 2009b). How do we integrate these two sets of findings? One obvious approach would be via considerations of genotype-environment interplay. GxE is defined as differential responsiveness to environmental risk as a function of genetic variability (Plomin, DeFries, & Loehlin, 1977), and is thought to constitute a fundamental mechanism through which genes influence mental health (Johnson, in press; Moffitt, Caspi, & Rutter, 2006), including youth externalizing and ASB (Hicks, South, DiRago, Iacono, & McGue, 2009).

A handful of studies across the three research teams have thus far examined whether and how neighborhood socioeconomic disadvantage moderates the origins of youth ASB (Burt, Klump, Gorman-Smith, & Neiderhiser, 2016; Burt, Slawinski, & Klump, 2018; Cleveland, 2003; Tuvblad, Grann, & Lichtenstein, 2006). Results have consistently and robustly indicated that youth ASB is considerably more environmental in origin in impoverished neighborhoods relative to wealthy and middle class neighborhoods. Cleveland (2003), for example, examined more than 2,000 sibling pairs from the National Longitudinal Study of Adolescent Health, and found that while genetic influences were important regardless of neighborhood context, shared or family-level environmental influences (i.e., those that increase sibling similarity regardless of the proportion of genes shared) were important only for those residing in disadvantaged neighborhoods. Similarly, Tuvblad and colleagues (2006) examined 1,133 adolescent twin pairs from a population-based study in Sweden, again finding that shared environmental influences on ASB were more important for adolescents residing in disadvantaged environments.

Our lab then constructively replicated and extended these results in the Twin Study of Behavioral and Emotional Development – Child (TBED-C), a study within the broader Michigan State University Twin Registry (MSUTR), further demonstrating that *a*) these larger shared environmental influences reflected actual influences of the environment on the twins and not selection or other confounds (Burt et al., 2016), *b*) these GxE effects do not vary across twin sex (Burt et al., 2018), and *c*) this shared environmental moderation appears to be exacerbated by physical proximity to neighbors with high levels of ASB (Burt, Pearson, Rzotkiewicz, Klump, & Neiderhiser, 2019). Such findings collectively suggest that

the stronger shared environmental influences on ASB found in disadvantaged neighborhoods may operate in part via social contagion and do so equally for both boys and girls.

The above results are interesting, both for their rather unusual consistency across samples, but also because they do not conform to our typical conceptualization of GxE. They are clearly not consistent, for example, with the well-known diathesis-stress model of GxE, in which genetic influences on the outcome are enhanced by environmental risk (Hicks et al., 2009). They also have little in common with “environmental sensitivity” models of GxE (i.e., the ‘biological sensitivity to context’ and ‘differential susceptibility’ models; Belsky & Pluess, 2009; Ellis & Boyce, 2008; Pluess, 2015), both of which posit that genetically-sensitive individuals are particularly responsive to both supportive and risky environments. Linder the differential susceptibility model, we would expect to see more prominent genetic influences in both very advantaged and very disadvantaged environments.

Fortunately, the findings are very much in keeping with another, less frequently discussed model of GxE: the ‘bioecological model’ (Bronfenbrenner & Ceci, 1994; Pennington et al., 2009). This model harkens back to early notions that genetic influences may sometimes be less important (Scarr, 1992), and environmental influences more important (Lewontin, 1995; Pennington et al., 2009), in deleterious environments. The internal logic of the bioecological model was best illustrated by Lewontin (1995) through the 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 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. In behavioral genetic terms, the bioecological GxE model thus predicts absolute increases in shared environmental influences with increasing environmental risk exposure. Genetic influences would be expected to decrease, although this effect may only be observable when examined relative to the shared environmental moderation (i.e., via standardized estimates). When describing low-risk environments, Pennington et al. stated “*unlike in a diathesis-stress model, the environmental factor in a bioecological interaction does not necessarily act on the same biological substrate as the genetic risk factors. Instead, it may just allow those genetic risk factors to account for more of the variance in outcome, because environmental risk factors that affect that outcome have been minimized*” (pg. 80; Pennington et al., 2009).

In sum, all studies examining neighborhood disadvantage as an etiologic moderator of ASB to date have indicated that there is such a strong ‘social push’ (Raine, 2002) for ASB in disadvantaged neighborhood contexts that it can elicit antisocial outcomes regardless of individual genetic risk. Even so, conclusions remain uncertain. In part, this uncertainty stems from the general fact that statistical interactions are considerably less replicable than are main effects (Cohen, Cohen, West, & Aiken, 2013; Whisman & McClelland, 2005), and thus require more rigorous verification efforts (a truism that is particularly trenchant in light of psychology’s replicability crisis; Open_Science_Collaboration, 2015; Tackett et al., 2017). This is especially the case here since all studies conducted to date (including our own) have relied exclusively on administrative boundaries (e.g., Census tracts, zip codes,

municipalities) when defining “neighborhoods”, and have not sought to confirm their findings using other operationalizations of neighborhood.

Indeed, the sole focus on administrative units is potentially problematic. Inherent in this approach is the assumption that aggregate data based on administratively-defined neighborhoods provide a reasonable representation of the “true” neighborhood where one spends time and interacts with others (Coulton, Korbin, Chan, & Su, 2001; Foster & Hipp, 2011). This assumption is a subject of intense scrutiny in both the sociological and geographic neighborhood literatures, most of which has in fact indicated that the operationalization of ‘neighborhood’ does affect our ability to correctly identify its effects (Coulton et al., 2001; Coulton, Korbin, & Su, 2002; Duncan et al., 2013; Foster & Hipp, 2011; Kwan, 2012; Spielman & Yoo, 2009; Spielman, Yoo, & Linkletter, 2013; Weiss, Ompad, Galea, & Vlahov, 2007). Duncan et al. (2014), for example, found that median distances to the closest tobacco retailer were overestimated by 110% when examining Census tract centroids relative to specific home addresses (352.9m versus 168m). They thus concluded that, whenever possible, researchers should make use of small-area, individual-level (or “egocentric”) neighborhood designations based on participant home location (e.g., an 800m radius around the address). Coulton et al. (2001) similarly found that resident-defined neighborhoods (which they considered the single best way of validly identifying a neighborhood) were nearly always smaller than their Census tract, with far smaller population-sizes, augmenting concerns with studies based solely on Census tracts. That said, Spielman et al. (2013) conducted a simulation experiment to examine how residential sorting (or neighborhood selection), spatial structure, and the geographic scaling of a person’s ‘neighborhood’ affected estimates of neighborhood effects of behavior. Although neither selection nor spatial structure emerged as important predictors of neighborhood effect estimation, they did observe systematic links with geographic scaling, such that neighborhood effects on behavior were overestimated when the scaling was too small and were underestimated when the scaling was too large.

Given the general agreement that studies of neighborhood effects should consider neighborhood definition, it is worthwhile noting that researchers have yet to agree on a single gold-standard definition. In light of this confusion, our team surveyed the literature for overarching themes, and in doing so, identified three ‘take-away messages’ to guide our work. First, although Census tracts can serve as reasonable proxies for neighborhood, they may be more useful as ‘first steps’ in the research process. Second, researchers should try to confirm their findings across multiple operationalizations of a given neighborhood, ranging from small, egocentric (or address-based) definitions of neighborhood to definitions that are too large (e.g., Counties). Finally, researchers should incorporate parental informant-reports of neighborhood characteristics, as they can be sure in that case that the neighborhood is the correct one.

Building on the above, yet another problem with the sole focus on administrative units is that it is not uncommon for neighborhoods (regardless of how they are defined) to evidence highly permeable borders, leading to considerable spillover or spatial contagion of disadvantage across ostensibly separate neighborhoods (Sampson, 2008). This autocorrelation bears directly on our core question (i.e., how does exposure to disadvantaged

neighborhood contexts alter the etiology of ASB?) but has yet to be incorporated into GxE analyses, which have thus far treated adjoining Census tracts as independent entities. In geography, proximity is handled empirically using spatial weights that recognize bordering units in the analysis, something that is not yet possible to incorporate into GxE models. Luckily, there are two ways that we can begin to address this issue. The first would be to define ‘neighborhoods’ using very broad definitions (e.g., Counties), as this will necessarily capture multiple neighborhoods (even as they also yield overly conservative estimates of neighborhood effects, as noted above). The second approach is to make use of egocentric or individual-level estimation techniques from the field of geography. This approach is advantageous both because empirical research has shown that children spend most of their leisure time within 500m of their home (Chambers et al., 2017), but also because families who reside within a few kilometers of each other will necessarily share some but not all neighbor informant-reports, thereby better capturing the possibility of spillover across their ‘neighborhoods’.

Current study

Given the above, it would be important to confirm that prior evidence of GxE persists to other operationalizations of neighborhood, with a specific focus on maternal and resident perceptions of neighborhood disadvantage. We made use of two samples, one of which was examined in prior studies from our lab (the TBED-C). The TBED-C oversampled twin families residing in modestly- to severely-disadvantaged neighborhoods. This feature makes the TBED-C an especially useful sample for our study since, to our knowledge, it is the only ‘neighborhood sampled’ twin study in the world. We then augmented this unique design with neighborhood informant sampling, in which we collected informant-reports from twin family mothers and from randomly-selected individuals residing near the twin families. The latter allowed us to employ egocentric geographic techniques to define neighborhoods (although we were unable to incorporate resident perceptions of neighborhood boundaries, since these were not assessed). We were also able to link up our neighbor informant-reports to the large-scale Michigan Twins Project, thereby allowing us to replicate and extend our results in a large, independent twin registry, and to do so using an administrative unit far larger than what one might reasonably define as a neighborhood (i.e., their County). The current project was thus well positioned to more definitively confirm whether disadvantaged neighborhood contexts moderate the genetic and environmental etiology of ASB.

METHODS

PARTICIPANTS

Twin families.—The current study made use of two samples within the population-based Michigan State University Twin Registry (MSUTR; Burt & Klump, 2013; Klump & Burt, 2006): the Twin Study of Behavioral and Emotional Development in Children (TBED-C) and the Michigan Twins Project (MTP). 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 (e.g., a significant developmental delay). Children provided informed assent, while parents provided informed consent for themselves and their children. The twins

collectively ranged in age from 6 to 10 years (mean = 7.99, SD = 1.49; although 24 pairs had turned 11 by the time the family participated) and were 49% female.

The TBED-C includes both a population-based sample (n=528 families) and an independent 'at-risk' sample (n=502 families). Additional inclusion criteria for the 'at-risk' sample specified that participating twin families lived in modestly- to severely-disadvantaged Census tracts. This additional criterion did eventuate in a less advantaged sample. For example, when compared to the population-based sample, the at-risk sample reported lower family incomes (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 ($d = .08$, ns). Other recruitment and sampling details are detailed in prior publications (e.g., Burt et al., 2016; 2018).

The primary aim of the on-going, population-based MTP is to collect health data on Michigan-born child and adolescent twins (current N = 11,883 families) that can be used either for data analysis or to select families for follow-up research. Because nearly all TBED-C families were recruited out of the MTP, families participating in the TBED-C were excluded from the MTP for the current study. The remaining 10,922 MTP twin pairs were thus eligible for the current study. These MTP twins were 50.1% female, and ranged in age from 3 to 17 years (mean age = 8.80 years, SD = 4.6 years) at the time of their assessment, although a few pairs (n=43) had turned 18 by the time their assessment was completed. Twins belonged to ethnic groups at rates comparable to lower Michigan inhabitants (e.g., Black: 7.9%, White: 82.0%, Multiracial: 5.3%, respectively) (Burt & Klump, 2012). In both studies, a parent provided informed consent for themselves and their children.

Neighbors.—The protocol for the at-risk arm of the TBED-C included the recruitment and assessment of randomly-chosen neighbors. Following the participation of a given family in the 'at-risk' study, 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. This approach resulted in a sample of 1,880 neighbors (63.2% women; 80.6% White, 11.6% Black, 7.8% other ethnic group memberships; average age of 52.6 with a range of 18-95 years). The response rate was 70%, of which 70% agreed to participate (for a final participation rate of 49%).

Given the overly broad definition of 'neighborhood' in the MTP, we sought to maximize our sample size (given that neighborhood effects are likely to be underestimated when the neighborhood is too large; Spielman et al., 2013). We thus added a second sample of 1,430 neighborhood informants (46.7% women; 86.2% non-Hispanic Caucasian, average age of 27.9 with a range of 18-70 years) across the state of Michigan. Participants in the second sample were recruited via the web-based Amazon marketplace MTurk (Buhrmester, Kwang, & Gosling, 2011). MTurk has a large (N>100,000) and diverse 'workforce' of individuals who complete surveys, writing, and other such tasks on-line. For the current study, we required that all participants resided in Michigan, and paid \$1.50 for completion of the assessment. Assessments were identical across the two samples of neighborhood informants.

MEASURES

Zygosity.—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% as compared to DNA. Sample sizes by zygosity are presented in Table 1.

Youth Antisocial Behavior.—Prior work in the TBED-C has found that the etiologic moderation of child ASB by neighborhood disadvantage was specific to non-aggressive ASB (see Burt et al., 2016 and Burt et al., 2018), and was observed only inconsistently (if at all) for aggressive ASB. These sorts of distinctive links with neighborhood dovetail nicely with the broader literature (see reviews and meta-analyses by Burt, 2009a, 2012; Tremblay, 2010), which has robustly indicated that, although they are correlated with one another, aggression and rulebreaking evidence consistently different developmental trajectories, different demographic correlates, and different associations with personality and other risk factors. What's more, they evidence differential patterns of genetic and environmental influences, such that physical aggression is significantly more genetic in origin, while rule-breaking is significantly more environmental in origin (Burt, 2009).

Building on the above, Achenbach & Rescorla (2001) found that the Rule-breaking Behavior scale predicts DSM-IV Conduct Disorder symptom counts ($r = .63$) and diagnoses ($r = .32$) better than any other syndrome scale on the Child Behavior Checklist (whereas the Aggressive Behavior scale best predicts Oppositional Defiant Disorder). Given all this, we thus focused on the well-known Rule-breaking Behavior scale (e.g., lies, breaks rules, steals, truant; 17 items on the CBCL and 12 items on the TRF) in the TBED-C. Mothers completed the Achenbach Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001) separately for each twin, while the twins' teacher(s) completed the corresponding Achenbach Teacher Report Form (TRF; Achenbach & Rescorla, 2001). Our teacher response rate was 83%. Mother and teacher reports were combined to form multi-informant composites of child ASB. Only 5 twins had missing composite scores. Consistent with manual recommendations (Achenbach & Rescorla, 2001), analyses were conducted on the raw scale scores. The mean ASB composite score was 1.22 ($SD = 1.64$), with a range of 0 to 14 (skew was 2.68). To adjust for this positive skew, the data were log-transformed prior to analysis to better approximate normality (skew after transformation was 0.74).

In the MTP, primary caregivers (nearly always the mother) completed the Strengths and Difficulty Questionnaire (SDQ; Goodman & Scott, 1999), along with a handful of additional items assessing behaviors not included on the SDQ. Because the SDQ does not distinguish between aggressive and non-aggressive ASB, we focused on the Conduct Problems scale (i.e., stealing, hot temper, physical fights; 5-items, $\alpha = .64$). Given its relatively low reliability, however, we added two items assessing content found on the Conduct Problems scale of the Child Behavior Checklist (i.e., destroys things that belong to others; thinks things out before acting, reverse-scored). The addition of these two items increased the internal consistency reliability ($\alpha = .72$). Moreover, an exploratory factor analysis yielded evidence of a clean break between the one- and two-factor solutions, with only one eigen value above 1.0 (3.907, next one was 0.855) and a reasonable RMSEA (.07). The seven

items evidenced high factor loadings (ranging from .63 to .76). Only 5.5% of twins had missing CP data. The mean CP score was 2.48 (SD=2.20), with a range of 0 to 14 (skew was 1.38). The data were log-transformed prior to analysis to better approximate normality (skew after transformation was -0.016).

Neighborhood disadvantage.—All informant-reports of neighborhood disadvantage were assessed using the Neighborhood Matters questionnaire (Henry, Gorman-Smith, Schoeny, & Tolan, 2014). The Extent of Neighborhood Problems scale consists of 13 items assessing perceptions that graffiti, drugs, abandoned buildings, vandalism, gangs, violent crime, etc., are a problem in their neighborhood ($\alpha = .95$ for both maternal and neighbor reports).

Operationalization of ‘neighborhood’. Because we did not have access to resident perceptions of neighborhood boundaries, we made use of egocentric geographic techniques to evaluate distances between each neighbor and each family. For the TBED-C, we geocoded all neighbor and twin family addresses with a 99.9% success rate using an “.html” code that uses Google Maps address data to assign coordinates. We then mapped the geocoded coordinates using ArcGIS v10.3 (ESRI, Redlands, CA). We verified the spatial accuracy of 20 random geocoded locations by comparing the tabular data to ensure that the assigned county and city names correspond with the Census tract found in the original dataset. Using the geocoded coordinates, we calculated average neighbor perceptions of neighborhood disadvantage for each twin family’s residential location using ArcMap software. Descriptive statistics for these various spatial covariates were then calculated using Stata v13 (College Station, TX).

We made use of several approaches to operationalizing neighborhood (see Table 1), two of which were egocentric (or individual-level), one of which made use of maternal informant-reports, and one of which was overly broad. We first averaged informant-reports across all neighbors residing within 1km of the twins (twins whose ‘neighbors lived more than 1 km away were omitted from these analyses). We then computed the distance to the nearest neighbor, again focusing exclusively on neighbors living relatively close to the twins (within 1 km). The mean distance to the nearest neighbor in these cases was 432m (SD = 255), with a median of 399m and a range of 0.25m to 984m. Finally, to ensure that results persisted across other informant-reports of disadvantage, we examined maternal reports of disadvantage in the twin family’s neighborhood as well. These were available for 723 families (the neighborhood questionnaire was in development during the early years of the study).

Identifying neighbors for the MTP sample was made somewhat more challenging by the fact that we did not have access to twin family addresses for this sample. Because the Department of Vital Records within the Michigan Department of Health and Human Services makes use of confidential driver’s license and birth record data to locate each family’s address for recruitment into the MTP, they will not release family addresses to researchers but will release administrative identifiers. To both account for both spillover within counties and examine a particularly conservative (i.e., overly broad) definition of neighborhood, we calculated average perceptions of neighborhood disadvantage at the level

of the County. Nearly all MTP families in the current sample ($N=10,246$ of $10,922$) resided in a County for which we had at least one neighborhood informant. Of these, $10,116$ were ultimately included in our analyses (we omitted 130 pairs with incomplete zygosity information).

ANALYSES

Classical twin studies (see Neale & Cardon, 1992) 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 additive genetic (A), shared environmental (i.e., environmental factors that make twins similar to each other; C), and non-shared environmental (i.e., factors that make twins different from each other, including measurement error; E) contributions to a given phenotype. We specifically fitted the ‘univariate GxE’ twin model (Purcell, 2002) to evaluate whether the various operationalizations of disadvantage moderated the etiology of youth ASB. Although prone to false positives when twin pairs are imperfectly correlated on the moderator (van der Sluis, Posthuma, & Dolan, 2012), Purcell’s univariate GxE model has been shown to be the most appropriate such model when the twins are perfectly concordant on the moderator (van der Sluis et al., 2012), as is the case here.

Mx (Neale, Boker, Xie, & Maes, 2003) was used to fit the GxE models to the data using Full-Information Maximum-Likelihood 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; $-2\ln L$). Model fit was 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 (e.g., they place different values on parsimony), we reasoned that the best fitting model should yield lower or more negative values for at least 3 of the 4 fit indices.

Prior to analyses, each moderator variable was floored at 0 and divided by its maximum, providing a continuous measure of disadvantage that ranged from 0 to 1. Twin sex and age were regressed out of the twin data, in keeping with prior recommendations (McGue & Bouchard, 1984). Although the interpretation of standardized or proportional ACE estimates may be useful in some cases, it is generally recommended that unstandardized or absolute ACE estimates be presented (Purcell, 2002). We thus standardized our log-transformed ASB score to have a mean of zero and a standard deviation of one to facilitate interpretation of the unstandardized values.

RESULTS

In both samples, child ASB varied significantly across sex, such that boys evidenced higher rates than did girls (Cohen’s d effect sizes in the TBED-C and the MTP were .33 and .18, respectively, $p < .01$). ASB also varied by race and age, such that it was less common in

White participants than in non-White participants (d s were $-.26$ and $-.30$, respectively, $p < .001$) and decreased slightly with age (r s = $-.08$ and $-.18$, respectively, $p < .01$).

As shown in Table 2, the various operationalizations and informant-reports of neighborhood disadvantage in the TBED-C were highly intercorrelated (r s = $.49$ to $.85$, all $p < .001$). Associations between the more proximal indices examined in the TBED-C and the overly broad County-level index were also small-to-moderate in magnitude (r s range from $.24$ to $.40$, all $p < .001$). Consistent with this, the various approaches to operationalizing informant-reports of neighborhood disadvantage were moderately-to-strongly correlated with the Census-tract level Area Deprivation Index (<https://www.neighborhoodatlas.medicine.wisc.edu/>), with r s ranging from $.28$ for County-level reports to $.54$ for all neighbors within 1 km (all $p < .001$). Finally, and again regardless of operationalization or informant, neighborhood disadvantage evidenced small and positively-signed associations with twin ASB (r s were $.06$ for County-level, $.16$ for nearest neighbor, $.19$ for all neighbors within 1 km, and $.20$ for maternal reports; all $p < .001$). Of note, the magnitude of the phenotypic correlation between a moderator and an outcome has no bearing on the presence or magnitude of etiologic moderation (Purcell, 2002).

Intraclass correlations are also presented in Table 2, separately by zygosity and level of disadvantage. In more advantaged neighborhoods, there were large and highly significant differences between MZ and DZ intraclass correlations (all $p < .001$), indicating the likely presence of strong genetic influences and minimal shared environmental influences on youth ASB in those contexts. In more disadvantaged neighborhoods, however, the MZ correlations were less than double the DZ correlations, and were equivalent in magnitude (with the exception of the MTP sample), suggesting the presence of higher C influences and/or lower A influences on youth ASB in these contexts. This impression is further bolstered by the finding that the DZ correlation increased at least somewhat with increasing levels of disadvantage in all four cases (the DZ correlation in disadvantaged neighborhoods was at least marginally larger than that in advantaged neighborhoods (p s range from $.04$ to $.09$)).

GxE results:

Formal tests of moderation were conducted next. Individual GxE model fitting results are presented in Table 3. Given prior findings in the TBED-C (Burt, et al., 2016, 2018, in press), as well as the moderator values in the full linear ACE moderation models (presented in Table 4), we also fitted the no A moderation model (in which the A moderator was fixed to zero and the C and E moderators were allowed to vary) and the no A or E moderation model (in which only the C moderator was allowed to vary).

There was clear evidence that disadvantage moderated the etiology of youth ASB, and did so regardless of the sample, the operationalization of neighborhood, or the specific informant(s) examined. The no A or E moderation model fitted the data best in all four sets of analyses. The C moderator was typically large and positively-signed, and was statistically significant in all but two models. The E moderator was much more modest in magnitude, and only reached statistical significance in the very large MTP sample, but was also consistently positively signed. The A moderator was more variable, and was not significantly different from zero in any of the analyses. The importance of these results was further augmented by

the small and uniformly non-significant *c* path estimates, which highlight the absence of meaningful shared environmental contributions to child ASB in those residing in advantaged neighborhoods. When viewed together, such findings argue that *C* contributes far more to the variance in child ASB when the twins reside in more disadvantaged neighborhood contexts. *A* and *E* influences, by contrast, were important to the etiology of child ASB regardless of level of disadvantage. In short, the evidence, as presented across two independent samples using multiple informants and operationalizations of neighborhood, robustly points to the presence of shared environmental moderation of child ASB by neighborhood disadvantage.

Post-hoc analyses:

There is one key consequence of this strong shared environmental moderation for the genetic components of variance: when *A*, *C*, and *E* are considered relative to one another, *A* could be proportionally less important in disadvantaged contexts than in advantaged contexts (even as its absolute contribution remains unchanged). To empirically evaluate this possibility, we divided the disadvantage scores at their median and, using their best-fitting model, computed standardized estimates of *A* in advantaged and disadvantaged contexts. We then computed the difference score between these standardized estimates of *A*, as well as the 95% confidence interval of that difference score. Confidence intervals that did not overlap with zero would indicate that the proportion of *A* variance differed significantly with disadvantage (Cumming & Finch, 2005; Knezevic, 2008), even as its absolute or unstandardized contribution remained constant. Results of these post-hoc analyses are presented in Table 5. As seen there, *A* accounted for 57-70% of the variance in ASB in more advantaged contexts, but only 24-54% in disadvantaged contexts. Moreover, these differences were statistically-significant, as indicated by confidence intervals that did not overlap with zero. We also observed significant changes in *C* and *E* across all four sets of analyses. Shared environmental influences, for example, were effectively zero (ranging from 0-3%) in the most advantaged neighborhoods, but accounted for 23-65% of the variance in the most disadvantaged neighborhoods.

DISCUSSION

The goal of the current study was to evaluate whether prior findings of bioecological GxE in youth ASB by neighborhood disadvantage persisted to other operationalizations of ‘neighborhood’ across two independent samples, with a specific focus on residents’ perceptions of conditions in their neighborhoods. Analyses indicated that, regardless of the neighborhood informant (i.e., mother or neighbor) or the operationalization of neighborhood (i.e., 1km, County), *C* influences on ASB increased many-fold in disadvantaged neighborhoods. *E* influences may also have increased, but did so only very slightly and less consistently. Absolute *A* contributions to ASB were constant across all permutations of neighborhood, but were proportionally less important in disadvantaged relative to advantaged contexts. In short, disadvantage appears to exert a powerful effect on the etiology of youth ASB, with evidence of etiologic moderation persisting not only to resident’s perceptions of disadvantage, but also across multiple geographic boundaries, including those that are quite broad (County).

These findings serve to constructively replicate and extend prior GxE studies focused on neighborhood disadvantage (Burt et al., 2016; Cleveland, 2003; Tuvblad et al., 2006), with prior findings of C moderation fully replicating across all informants and operationalizations examined. Moreover, the effects were generally large. Standardized estimates indicated that, in wealthy neighborhoods, C influences were effectively zero (ranging from 0-3%). In highly impoverished neighborhoods, by contrast, C influences accounted for 23-65% of the variance in youth ASB. Moreover, as one would expect, moderating effects were particularly pronounced in smaller geographic radii (i.e., 1 km and maternal informant-reports), and appeared to become less so as distances increased. When viewed alongside prior literature, the current findings point to potent effects of disadvantage on the etiology of youth ASB, whereby youth ASB is considerably more shared environmental in origin in disadvantaged neighborhood contexts.

In contrast, prior findings of absolute changes in genetic influences with the level of disadvantage (Tuvblad et al., 2006) were not replicated (i.e., the genetic moderator was uniformly not significant, even in our largest sample), arguing against the notion that genetic influences on ASB are deactivated or silenced in disadvantaged contexts. This lack of replication is not specific to the current study, as other studies have also found little evidence of absolute genetic moderation (e.g., Cleveland, 2003). Interestingly, however, there was clear evidence here that genetic influences accounted for a significantly smaller proportion of variance in ASB when the twins resided in disadvantaged contexts (24-54%) than when they resided in advantaged contexts (57-70%). As these standardized analyses were not conducted in other studies, it is not clear whether the proportional (but not absolute) moderation of A in the current study echoes that of other studies. Regardless, our findings are still very much in keeping with the bioecological model of GxE, which allows for proportional changes in genetic influences as a consequence of absolute environmental moderation.

There are a few key limitations to the current study. First, although we collected resident perceptions of neighborhood characteristics, we did not explicitly collect resident perceptions of neighborhood boundaries. It is thus possible that neighbors were referencing a neighborhood different from (but in close proximity to) the one in which the twin family resides. Our explicit focus on the spatial contagion of disadvantage somewhat obviates this concern, since residing near the twins' neighborhood meets this criterion. What's more, our findings persisted regardless of whether the neighborhood in question was definitely that of the twins (as with maternal informant-reports) or whether the neighborhood in question was less clear (and could be either the same or near to that of the twins). Put another way, the notable consistency of our results across all operationalizations and informant-reports suggests that this limitation does not undermine our results in any substantive way.

Second, because our analyses were already complicated by the several different operationalizations of neighborhood, we regressed sex out of twin ASB prior to analysis. This decision is bolstered by recent work indicating the clear absence of joint etiologic moderation of ASB by sex and neighborhood disadvantage (Burt et al., 2018). Finally, although our neighborhood informant samples were large and typically included several

participants per neighborhood, it is nevertheless unclear whether participating neighbors were representative of adults in their neighborhoods. Future work should clarify this.

Conclusions

When viewed collectively, and in the context of prior studies (Burt et al., 2016; Cleveland, 2003; Tuvblad et al., 2006), the current results indicate that neighborhood disadvantage exerts rather profound effects on the etiology of youth ASB. Environmental influences (and specifically, those that create similarity between siblings) are significantly more potent in the presence of neighborhood disadvantage. Moreover, these effects are strong enough to be detected across a variety of measurement strategies, including maternal and neighbor informant-reports of neighborhood characteristics and, in prior work, Census data (e.g., the % of residents living below poverty). They are also strong enough to be detected across multiple operationalizations of neighborhood, including administratively-defined units like County and egocentrically-defined neighborhoods (e.g., the 1km radii examined here). This consistency is reassuring, since lack of replication is a key issue for studies of interactions (Cohen et al., 2013).

These findings have two key implications. First, although neighborhood researchers rightly note that neighborhoods should be measured with precision and care lest measurement error obscure or inflate estimates of community effects, the current results indicate that, assuming one has access to a very large sample or maternal informant-reports of the neighborhood, the specific operationalization of neighborhood need not hamper our ability to evaluate the etiological effects of neighborhood structural characteristics on child ASB. Such findings likely reflect either the unusually robust nature of neighborhood disadvantage as an etiologic moderator and/or the effects of spatial contagion onto residents of nearby neighborhoods. Regardless, these results point to a clear need to identify the active ingredients of neighborhood disadvantage on etiology going forward.

With regard to those active ingredients, it is worth spending a moment to outline some likely culprits. Extant work across behavioral genetics, developmental psychology, toxicology, and developmental neuroscience points to a few key possibilities. Neuroimaging studies strongly suggest that youth ASB is characterized by functional and structural alterations in limbic and prefrontal neural regions that support the regulation and control of emotion and behavior (Hyde, Shaw, & Hariri, 2013). An emerging line of work further suggests that disadvantage may alter the structure and function of many of these same specific regions (Holz, Laucht, & Meyer-Lindenberg, 2015), a phenomenon sometimes referred to as the ‘biological embedding’ of poverty’. The most promising active ingredients for future study are thus those that demonstrate associations not only with neighborhood disadvantage and youth ASB, but also with neural structure and function of relevant regions (McEwen & Gianaros, 2010). For example, stressors like exposure to community violence, harsh/disrupted parenting, and neurotoxicant exposures (e.g., lead) demonstrate well-documented links to both neighborhood disadvantage and youth ASB, as well as to changes in neural structure and function of prefrontal limbic regions, likely via glucocorticoid signaling (McEwen & Gianaros, 2010). Emerging human MRI studies further suggest that these stressors alter

brain structure and function (e.g., Hackman, Farah, & Meaney, 2010). Future studies should evaluate each of these possibilities.

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Table 1.

Sample sizes for each GxE analysis

Study	Informant	Operationalization	Number of informants				Number of twin families		
			Mean (SD)	Median	Min	Max	Total	MZ	DZ
TBED-C	Mother	Maternal perceptions of family's neighborhood disadvantage	1 (.00)	1	1	1	723	284	439
	Neighbor	All neighborhood informants within 1km	3.12 (2.08)	3	1	16	459	163	296
		Neighborhood informant nearest to the twin family within 1 km	1 (.00)	1	1	1	459	163	296
MTP	Neighbor	All neighborhood informants within twin family's County	188.47 (148.66)	200	1	482	10,116	2738	7378

Table 2.

Phenotypic and intraclass correlations

Phenotype	1.	2.	3.	4.	Intraclass correlations for ASB			
					Advantaged		Disadvantaged	
					MZ	DZ	MZ	DZ
1. All neighbors within 1km of the twins	--				.74 [^]	.42 [^]	.72	.54
2. Neighbor nearest to the twins	.85**	--			.73 [^]	.41 [^]	.72	.54
3. Maternal perceptions of neighborhood disadvantage	.56**	.49**	--		.64 [^]	.33 [^]	.60	.47
4. All neighbors within twins' County	.40**	.33**	.24**	--	.70 [^]	.36 [^]	.69 [^]	.39 [^]

Note.

** indicates that the phenotypic correlation (on the left half of the table) is significantly larger than zero at $p < .01$. Although the TBED-C twins were omitted from all other MTP County-level analyses (including computation of the intraclass correlations on the right side of the table), the correlations among the various operationalizations of neighborhood presented here were conducted only in the TBED-C twins (since we cannot correlate definitions in two other independent samples). All intraclass correlations are significantly larger than zero at $p < .01$.

[^] indicates that the monozygotic (MZ) correlation is significantly larger than the dizygotic (DZ) correlation at $p < .05$.

Table 3.

Fit Indices

Sample and Operationalization of Neighborhood Disadvantage	-2lnL	df	AIC	BIC	SABIC	DIC
<u>All neighbors within 1km of the twin family (TBED-C)</u>						
Full ACE moderation	2425.78	902	621.78	-1548.35	-117.03	-719.47
No A moderation	2425.93	903	616.93	-1551.34	-118.43	-721.54
No A or E moderation	2427.20	904	619.20	-1553.77	-119.27	-723.05
No moderation	2440.64	905	630.64	-1550.11	-114.02	-718.47
<u>Nearest neighbor to the twin family (TBED-C)</u>						
Full ACE moderation	2389.62	886	617.62	-1509.61	-103.71	-695.43
No A moderation	2390.22	887	616.22	-1512.37	-104.88	-697.27
No A or E moderation	2390.43	888	614.43	-1515.31	-106.24	-699.30
No moderation	2404.72	889	626.72	-1511.22	-100.56	-694.29
<u>Maternal report (TBED-C)</u>						
Full ACE moderation	3715.24	1431	853.24	-2849.83	-577.92	-1534.83
No A moderation	3715.26	1432	851.26	-2853.11	-579.61	-1537.19
No A or E moderation	3718.28	1433	852.28	-2854.89	-579.80	-1538.05
No moderation	3733.71	1434	865.71	-2850.47	-573.79	-1532.71
<u>All neighbors within twin family's County (MTP)</u>						
Full ACE moderation	51111.58	18957	13197.58	-61272.20	-31151.01	-43851.88
No A moderation	51111.70	18958	13195.70	-61276.71	-31153.93	-43855.48
No A or E moderation	51116.78	18959	13198.78	-61278.76	-31154.39	-43856.60
No moderation	51127.55	18960	13207.55	-61277.95	-31151.99	-43854.88

Note. The best fitting model for a given set of analyses is highlighted in bold font, and is indicated by the lowest 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 4 fit indices.

Table 4.

Unstandardized path and moderator parameter estimates for the full linear moderation, no A moderation, and no A or E moderation models

	PATHS			LINEAR MODERATORS		
	a	c	e	A ₁	C ₁	E ₁
FULL LINEAR ACE MODERATION						
TBED-C, all neighbors within 1 km	0.81*	0.00	0.47*	-0.11	1.29*	0.18
TBED-C, nearest neighbor within 1 km	0.81*	0.11	0.49*	-0.23	1.25*	0.12
TBED-C, maternal report of neighborhood	0.68*	0.19	0.54*	0.05	0.75	0.17
MTP, County-level	0.83*	-0.04	0.50*	0.02	0.35	0.06
NO A MODERATION						
TBED-C, all neighbors within 1 km	0.79*	-0.01	0.48*	---	1.24*	0.14
TBED-C, nearest neighbor within 1 km	0.77*	0.15	0.50*	---	1.07*	0.06
TBED-C, maternal report of neighborhood	0.68*	0.19	0.54*	---	0.79*	0.18
MTP, County-level	0.84*	-0.05	0.50*	---	0.39*	0.06*
NO A OR E MODERATION						
TBED-C, all neighbors within 1 km	0.79*	-0.03	0.52*	---	1.31*	---
TBED-C, nearest neighbor within 1 km	0.77*	0.14	0.52*	---	1.10*	---
TBED-C, maternal report of neighborhood	0.68*	0.16	0.57*	---	0.90*	---
MTP, County-level	0.84*	-0.08	0.53*	---	0.45*	---

Note. A, C, and E (upper and lower case) respectively represent genetic, shared, and non-shared environmental parameters on youth antisocial behavior. Because the lowest levels of the neighborhood disadvantage variable was coded as 0, the genetic and environmental contributions to youth antisocial behavior at this level can be obtained by squaring the path estimates (i.e., a, c, and e). At higher levels, linear moderators (i.e., A₁, C₁, E₁) were added to the paths using the following equation: $Unstandardized\ Variance\ Total = (a + A_1(neighborhood\ disadvantage))^2 + (c + C_1(neighborhood\ disadvantage))^2 + (e + E_1(neighborhood\ disadvantage))^2$. Bold font and an asterisk indicate that that parameter estimate was significant at $p < .05$.

Table 5.

Standardized genetic and environmental variance estimates at in advantaged versus disadvantaged neighborhoods

	%A			%C			%E		
	Advantaged	Disadvantaged		Advantaged	Disadvantaged		Advantaged	Disadvantaged	
All neighbors within 1km	.70*	.24*	$\begin{matrix} \text{---}^{\wedge} \\ 45^{\wedge} (-.54, -.22) \end{matrix}$	.00	.65*	$\begin{matrix} \cdot^{\wedge} \\ 65^{\wedge} (.33, .77) \end{matrix}$	.30*	.11*	$\begin{matrix} \text{---}^{\wedge} \\ 20^{\wedge} (-.29, -.10) \end{matrix}$
Nearest neighbor within 1 km	.67*	.25*	$\begin{matrix} \text{---}^{\wedge} \\ 43^{\wedge} (-.56, -.20) \end{matrix}$	.02	.64*	$\begin{matrix} \cdot^{\wedge} \\ 62^{\wedge} (.31, .79) \end{matrix}$	.31*	.11*	$\begin{matrix} \text{---}^{\wedge} \\ 19^{\wedge} (-.29, -.10) \end{matrix}$
Maternal-report	.57*	.24*	$\begin{matrix} \text{---}^{\wedge} \\ 33^{\wedge} (-.46, -.17) \end{matrix}$	.03	.59*	$\begin{matrix} \cdot^{\wedge} \\ 56^{\wedge} (.30, .74) \end{matrix}$	.39*	.17*	$\begin{matrix} \text{---}^{\wedge} \\ 23^{\wedge} (-.32, -.12) \end{matrix}$
All neighbors within County	.69*	.54*	$\begin{matrix} \text{---}^{\wedge} \\ 16^{\wedge} (-.29, -.02) \end{matrix}$	.005	.23*	$\begin{matrix} \cdot^{\wedge} \\ 23^{\wedge} (.02, .42) \end{matrix}$	.30*	.23*	$\begin{matrix} \text{---}^{\wedge} \\ 07^{\wedge} (-.13, -.01) \end{matrix}$

Note. %A, %C, and %E represent standardized genetic, shared, and non-shared environmental parameters on youth antisocial behavior respectively. Bold font and an asterisk indicates that the parameter estimate is significantly greater than zero at $p < .05$.

An ^ indicates that the standardized parameter estimates in advantaged versus disadvantaged neighborhoods (i.e., those within a given row) differ from one another at $p < .05$, as indicated by a difference score confidence interval that does not overlap with zero.