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A Quasi-Experimental Analysis of the Influence of Neighborhood Disadvantage on Child and Adolescent Conduct Problems

Jackson A. Goodnight[1,8], **Benjamin B. Lahey**[2], **Carol A. Van Hulle**[3], **Joseph L. Rodgers**[4], **Paul J. Rathouz**[5], **Irwin D. Waldman**[6], and **Brian M. D'Onofrio**[7]

[1]University of Dayton

[2]University of Chicago

[3]University of Wisconsin-Madison

[4]University of Oklahoma

[5]University of Wisconsin School of Medicine and Public Health

[6]Emory University

[7]Indiana University

Abstract

A quasi-experimental comparison of cousins differentially exposed to levels of neighborhood disadvantage (ND) was used with extensive measured covariates to test the hypothesis that neighborhood risk has independent effects on youth conduct problems (CPs). Multilevel analyses were based on mother-rated ND and both mother-reported CPs across 4–13 years (n = 7,077) and youth-reported CPs across 10–13 years (n = 4,524) from the Children of the National Longitudinal Survey of Youth. ND was robustly related to CPs reported by both informants when controlling for both measured risk factors that are correlated with ND and unmeasured confounds. These findings are consistent with the hypothesis that ND has influence on conduct problems.

Children growing up in disadvantaged neighborhoods characterized by poverty, low levels of social organization and cohesion, and high levels of residential instability and crime, are at increased risk for a host of negative outcomes, including academic failure, depression and anxiety, teenage pregnancy, and conduct problems (Harding, 2003; Leventhal & Brooks-Gunn, 2000; Sampson, Raudenbush, & Earls, 1997). These children may experience increased risk for these outcomes for several reasons. One possibility is that environmental factors inherent in such high-risk neighborhoods exert causal influences on conduct problems (CPs). Another possibility is that individual- and family-level factors that are correlated with neighborhood disadvantage (ND), such as inadequate parental supervision and low family income, actually cause increased risk for CPs, with the relation between ND and youth CPs being non-causal. At present, the existing research literature does not clearly support either of these alternative explanations more than the other. This is an important shortcoming, as determining if ND is a causal risk factor for CPs or only a spurious correlate

[8]All correspondence should be sent to: Jackson A. Goodnight, Department of Psychology, University of Dayton, 300 College Park, Dayton, OH 45469-1430. Phone: 937-229-2738. Fax: 937-229-3900. jgoodnight1@notes.udayton.edu.

would have major implications for prevention science and public policy (Leventhal & Brooks-Gunn, 2000).

Factors Associated with the Magnitude of Correlations with Neighborhood Characteristics

A review of regional and national studies found that ND is associated with higher levels of CPs, including defiant, aggressive, and delinquent behaviors, in childhood and adolescence (Leventhal & Brooks-Gunn, 2000). These studies varied widely in their estimates of the magnitude of the association, however, with some studies finding no main effect association at all (e.g., Beyers, Bates, Pettit, & Dodge, 2003; Lynam et al., 2000). Understanding the sources of these inconsistencies in findings may shed light on how research on neighborhood effects should proceed.

Measurement of neighborhood risk

Inconsistencies in findings from previous studies may result from differences in measurement of neighborhood characteristics. Some studies utilized census-based measures of neighborhood characteristics, whereas others used maternal ratings of neighborhood risks. Although there is some evidence that these approaches capture similar information (e.g., Ingoldsby & Shaw, 2002; Sampson, 1997), some studies have found differences depending on measurement approach. Ingoldsby and Shaw (2002) found that mother-reported neighborhood characteristics independently predicted trajectories of CPs, whereas census-based measures of neighborhood characteristics were not associated with CP trajectories after controlling for measured covariates. Sampson et al. (1997) have demonstrated that census-based measures of economic disadvantage and residential instability exert an indirect influence on conduct problems by contributing to low levels of informal social control. Thus, measuring neighborhood social processes may provide a more direct estimate neighborhood risk. However, it is also possible that differences in effects of ND across measurement approaches may be influenced by shared method bias in cases where caregivers report on both ND and conduct problems.

Variations in sampling

Inconsistencies in neighborhood effects also may be attributable to differences in sampling schemes used in different studies. Whereas some studies have used nationally representative samples, others used regional and local samples. Leventhal and Brooks-Gunn (1997) reported that nationally-based studies were more consistent in finding significant associations between ND and youth CPs, perhaps because nationally-based studies include a greater range of neighborhood characteristics than local samples. Sampling across a broad range of ND may be important, as some studies have found that ND is related to CPs only at extreme levels of disadvantage (e.g., Winslow & Shaw, 2007). Alternatively, national studies typically recruited larger samples and may simply have had greater statistical power to detect effects.

Several of the previous nationally-based studies used data from earlier waves of the same Children of the National Longitudinal Survey of Youth (CNLSY) on which the present analyses are based to examine relations between varying constructs reflecting neighborhood characteristics and child behavior problems. Chase-Lansdale and Gordon (1996) used the CNLSY to estimate the effect of neighborhood risk variables on early school-aged children's externalizing behavior problems (n = 673). Neighborhood crowding was associated with greater externalizing behaviors, and other aspects of ND were associated with externalizing behaviors in specific race-ethnic groups and regions of the country. Pachter, Auinger, Palmer, and Weitzman (2006) used a small subsample from the CNLSY

dataset to estimate the association of neighborhood characteristics with mother-reported conduct problems in their 6–9 year old offspring (n = 1,826). They found that the association of mother-rated neighborhood risk and mother-reported conduct problems was partially mediated by parenting variables among Non-Hispanic white and African American children, but no neighborhood effects were detected for Hispanic families.

Colder, Lengua, Fite, Mott, and Bush (2006) used a small subsample from the CNLSY to study the association between mother-rated ND and mother-reported child conduct problems using a measure of ND that is nearly identical to the measure used in the present analyses. They found that greater ND was associated with higher levels of conduct problems at age 12 (n = 232), but found interactions between child temperament risk factors and neighborhood risk in predicting conduct problems. Similarly, Turner, Hartman, and Bishop (2007) used a subset of 513 adolescents to study the association between ND and persistent violent offending. Neighborhood risk was assessed using maternal-rated neighborhood social cohesion when adolescents were 13 years old on average. Turner et al. (2007) did not test main effects of neighborhood risk in their models, but they found an interaction with race-ethnicity indicating that prenatal risk factors increased the association between family dysfunction and probability of persistent offending, but only for African American and Hispanic youth living in the riskiest neighborhoods.

Moderators of neighborhood influence

In addition to race-ethnicity (Pachter et al., 2006) and region of the country (Chase-Lansdale & Gordon, 1996), the association of neighborhood characteristics and youth CPs has been found to vary by youths' sex (Kling et al., 2005), impulsivity (Meier, Slutske, Arndt, & Cadoret, 2008; Trentacosta, Hyde, Shaw, & Cheong, 2009), and child temperament (Colder et al., 2006), as well as by combinations of these factors (Turner et al., 2007). Several studies have also investigated the interaction between neighborhood characteristics and parenting practices. Beyers et al. (2003) tested the interaction between neighborhood risk and parental knowledge of their adolescent offsprings' whereabouts and companions as predictors of growth in conduct problems in early adolescence. Although Beyers et al. (2003) did not find evidence for a main effect of neighborhood risk, they did find that parental knowledge more strongly predicted growth in conduct problems for youths from unstable neighborhoods. Eamon (2001) also found that parental knowledge was more strongly associated with adolescent delinquency in high-risk neighborhoods. Although Lahey, Van Hulle, D'Onofrio, Rodgers, and Walman (2008) did not replicate the moderating effect of neighborhood characteristics on parental knowledge, they found that parental limit setting interacted with neighborhood risk in predicting adolescent delinquency (see also Roche, Ensminger, & Cherlin, 2007).

Selection effects

Inconsistency in findings from neighborhood research also may reflect the degree to which each study controlled selection effects, which are effects due to confounded factors that account for non-random entry into high-risk neighborhoods and may account for negative effects of ND. Markedly different results may be found depending on the adequacy with which studies control for the confounding inherent in selection effects. If controls are insufficient, neighborhood effects may be biased upward. If controls are excessive, as in the case of mistakenly controlling for mediating influences of neighborhood effects, neighborhood effects may be biased downward.

Leventhal and Brooks-Gunn (2000) noted that future studies need to use methods that can control confounding by accounting for non-random selection into high-risk neighborhoods. The call for more rigorous examination of alternative explanations for the association

between ND and offspring adjustment has been mirrored by the recent calls by numerous researchers to more systematically test hypotheses regarding the causal effects of putative environmental risk factors (e.g., Moffitt, 2005; Rutter, 2007; Rutter, Pickles, Murray, & Eaves, 2001, Academy of Medical Sciences Working Group, 2007). Three major approaches have been taken to control for selection effects: using statistical covariates (including measured characteristics of families and previous levels of externalizing problems), quasi-experimental within-family designs, and experimental designs.

The statistical control of measured covariates has been the most frequently used approach to address confounding, including the control of measured variables that are believed to be potential selection factors. Because a full review is available elsewhere (Leventhal & Brooks-Gunn, 2000), we highlight two studies to illustrate the use of statistical covariates to account for selection factors. Lynam, Caspi, Moffitt, Wikstrom, Loeber, and Novak (2000) found that neighborhood risk predicted youth-reported status offenses at age 17, controlling for age 13 delinquency, child race-ethnicity, family household income, and family composition. Similarly, Chase-Lansdale et al. (1996) controlled gender, race, maternal education, family income, maternal age at first birth, maternal employment, and whether or not the family had lived with the children's maternal grandparents at any point in time. The inclusion of statistical controls improves the precision of estimates of neighborhood effects. However, studies that rely solely on statistical controls cannot entirely rule out omitted variable bias, because it is not possible to know if all relevant confounds have been included in such analyses (Rutter, et al., 2001). In addition, statistical control is only successful when the nature of the association of the potential confounding variable and the outcome (e.g., linear) conforms to the statistical model.

An alternative approach is to use research design (Shadish, Cook, & Campbell, 2002) to control for both measured and unmeasured confounds to strengthen causal inferences regarding hypothesized neighborhood effects. One method involves modeling differences in exposure to neighborhood risks and CPs within families. This strategy controls for all characteristics that make the family members similar, such as shared environmental influences and some genetic effects (Rodgers, Cleveland, van den Oord, & Rowe, 2000). The primary advantage of this approach over the use of statistical covariates is that it controls for all variables that make related individuals similar, not just those influences that have been measured and included in the analysis as statistical covariates.

Using a within-family approach to study neighborhood effects on conduct problems, Caspi, Taylor, Moffitt, and Plomin (2000) used a genetically informative sample to estimate the effects of neighborhood characteristics while controlling for confounded genetic and shared environmental effects. A biometric ACE model (Neale & Cardon, 1992), a commonly used method for partitioning variance in a phenotype into genetic and environmental components by modeling differences in levels of similarities among individuals' who vary in their genetic relatedness, was used to determine the contributions of additive genetic effects (A), shared environmental effects (C; environments that make siblings more similar), and unique environmental effects (E; environments that make siblings dissimilar, plus measurement error) on behavior problems in a sample of 2-year-old children. Neighborhood risk was modeled as a predictor of variance attributable to the shared environment. Caspi et al. (2000) found evidence for a small but statistically significant effect of neighborhood on the shared environment parameter, concluding that neighborhood effects were in part environmentally mediated. However, their design could not rule out the possibility that the neighborhood effect reflected a gene-environment correlation (genetic selection factors) rather than a causal neighborhood effect (Purcell & Koenen, 2005; Turkheimer, D'Onofrio, Maes, & Eaves, 2005).

A different within-family design was used to study neighborhood effects on children's academic outcomes. Aaronson (1998) used a fixed-effects sibling comparison design to test the effects of the proportion of high school dropouts in the neighborhood on youths' likelihood of dropping out of high school. In addition to using a sibling-comparison approach to control for selection effects, Aaronson included several measured statistical controls that could be confounded with sibling differences in neighborhood risk. Aaronson found that the siblings living in neighborhoods during childhood with higher dropout rates were more likely to drop out of high school than their siblings exposed to neighborhoods during childhood with lower dropout rates. The results suggested that neighborhood dropout rates cause youth to be more likely to drop out of high school. Aaronson noted, however, that sibling-comparison designs may not be capable of precisely estimating neighborhood effects because there may insufficient between-sibling variation in either the neighborhood factor or the outcome variable. In Aaronson's sample, between 14 and 30% of variability in neighborhood dropout rate was unique to individuals within a family (i.e., not shared by siblings).

Experimental designs provide the best approach for ruling out selection effects. When random assignment is used, the effect of any intervention is made independent of any potential selection effects, making it possible to conclude that any difference between groups was caused by some aspect of the intervention. The Moving to Opportunity (MTO) Study provided one such test of neighborhood effects. Participants in MTO, who were self-selected into the study, were randomly assigned to various conditions. Outcomes for families who received housing vouchers and assistance in moving out of low-income neighborhoods were compared to families in two control conditions. Early analyses showed positive effects of changing neighborhoods on offspring conduct problems (Katz, Kling, & Liebman, 2001; Ludwig, Duncan, & Hirschfield, 2001), but more recent analyses of data from all MTO sites found mixed results (e.g., Kling, Ludwig, & Katz, 2005). Another experimental study of neighborhood effects, the Yonkers Project, found similarly mixed results of neighborhood change depending on the age of the offspring, suggesting that moving to higher-income neighborhoods may be beneficial for children, but unproductive or perhaps even detrimental to adolescents (Fauth, Leventhal, & Brooks-Gunn, 2005)

Experimental designs have the potential to provide strong evidence of causal effects, but the extant experimental studies of neighborhood effects were limited in several ways. First, they have included primarily African American samples and have been conducted solely in urban areas, limiting the generalizability of their findings. Second, one must consider the selection factors that influenced the decision to either agree or decline to participate in the study. Third, the studies have evaluated the effects of moving out of poor neighborhoods, but, for ethical reasons, they did not evaluate whether moving into poor neighborhoods contributes to the development of CPs. These limitations, along with the inconsistency of findings from these studies, suggest that our current understanding of neighborhood effects from randomized studies is far from complete. Thus, further research is warranted, especially research studies that use novel approaches for controlling selection factors.

Current Analyses

The current analyses were undertaken to address several limitations of past research on the potential causal effects of ND on offspring CPs. The present study differs from previous studies in three primary ways. First, the study uses data on large subsamples of youth from the nationally-representative CNLSY, both to insure adequate power for detecting effects and to maximize generalizability to the U.S. population. Because previous studies based on the CNLSY only used data available through the 1986 assessment (e.g., Chase-Lansdale et al., 1996; Colder et al., 2006) or 1994 (Turner et al., 2007), and analyzed only a small portion of the total number of available offspring, their findings may only be generalizable

to women who gave birth at younger ages, whose children are at greater risk for CPs than children of older mothers (D'Onofrio et al., 2009b; Geronimus, Korenman, & Hillemeier, 1994; Turley, 2003). The present analyses include data collected during recent waves of the study (through 2006). The two sets of analyses reported here, which are based on a subset of 7,077 children (ages 4–13 years) and a subset of 4,524 children (ages 10–13 years), includes far more children than any previous study of ND in the CNLSY.

Second, the present analyses are based on both maternal reports of childhood conduct problems (ages 4–13) and youth reports of delinquency (ages 10–13) to examine whether biases due to using reports of both neighborhood characteristics and CPs from the same informant account for the observed association.

Third, the present analyses use numerous measured covariates and the method of cousin comparisons, a quasi-experimental approach that has greatly informed research on putative environmental risk factors in the CNLSY (e.g., Geronimus, et al., 1994; Turley, 2003) and other studies (Slutske et al., 2008). Cousin comparisons control for all unmeasured environmental risks and genetic factors that make cousins similar in their exposure to ND and may confound the association between neighborhood risk and offspring CPs. However, cousin comparisons do not control for genetic and environmental influences that vary between extended family members and that are associated with variations in neighborhood risk. Thus, they are useful in minimizing, but are not capable of completely eliminating, confounding influences. For example, if income is associated with ND and offspring conduct problems and varies within extended families, cousin-comparisons would not control the confounding effect of income on the association between ND and offspring conduct problems. The analyses also controlled for measured risk factors that are correlated with ND in an effort to further reduce the effects of confounding influences that vary between cousins and potentially influence CPs.

By combining a quasi-experimental design and statistical controls, the current analyses respond to the call by Leventhal and Brooks-Gunn (2006) and other researchers (Academy of Medical Sciences Working Group, 2007; Moffitt, 2005; Rutter, 2007) to use designs that can help distinguish independent effects of ND from the influence of confounded risk factors. Although each approach to controlling for selection artifacts and other confounding is imperfect, including the cousin-comparison approach, corroborating evidence from a variety of designs with offsetting threats to their validity would strengthen the conclusion that ND has independent influence on CPs.

METHODS

Sample

The National Longitudinal Survey of Youth 1979 (NLSY79) is an ongoing two-generation longitudinal study funded by the Bureau of Labor Statistics. In the first generation, it has followed a group of individuals (originally assessed between the ages of 14 and 22 years) since 1979 (review in Baker & Mott, 1989). The NLSY79 included all individuals in a sampled household in the target age range. Using a stratified and clustered household probability sample and an over-sample of African American and Hispanic youth, the NLSY79 provides a sample of youth recruited from a representative sample of U.S. households. The sample has been assessed annually from 1979 to 1994 and biennially afterwards, maintaining a response rate > 80% in every wave. The diverse sample includes 4,912 women who have had at least one child four years old or older by 2004 (25.9% are African American, 17.1% are Hispanic, and 57.1% are non-African American, non-Hispanic, by self-report).

Since 1986 researchers have assessed all of the offspring of the women in the NLSY79 (Chase-Lansdale, Mott, Brooks-Gunn, & Phillips, 1991). The biennial assessments of these offspring in the CNLSY have response rates averaging 90%. The total number of offspring is 11,431 children, although a number of children were not included in the present analyses because of (a) insufficient information on their exposure to neighborhood risks or (b) the offspring were never assessed during the target age ranges (i.e., they were too old to be assessed during childhood or not old enough yet to complete assessment during adolescence). Tests for sample bias due to not using the full sample are reported below. Many previous studies of the CNLSY could have been influenced strongly by sample biases because only small subsets of the full sample were used and tests of sample bias were not performed. The current analyses include recent assessments for maternal reports of children under the age of 14 (the 2004 data collection) and for youth (10 years old or older) self-report (the 2006 data collection). Because the current study focuses on outcomes in childhood and the fact that the overwhelming majority (well over 95%) of the expected childbearing was completed by the 2004 survey, the bias due to maternal age at childbearing is relatively small.

Measures

Neighborhood disadvantage—Eight items, answered by mothers, assessed perceptions of neighborhood disadvantage (ND). The items assessed whether the following items were a problem in their neighborhood (1 = big problem, 2 = somewhat of a problem, or 3 = not a problem) and included: people don't have enough respect for rules and laws; crime and violence; abandoned or run-down buildings; not enough police protection; not enough public transportation; too many parents who don't supervise their children; people keep to themselves, don't care about the neighborhood; lots of people who can't find jobs. This mother-report measure of ND was included in the CNLSY assessments conducted between 1992 and 2000. It includes both structural and behavioral indices of ND, which is strongly recommended when exploring the effects of neighborhood risk (Leventhal and Brooks-Gunn, 2000).

The items in the maternal ratings of ND were internally consistent, as the Chronbach α's ranged from 0.83–0.85 across these waves. Principal component analysis of the ND items (averaged from ages 4 to 13) provided evidence for a single neighborhood disadvantage factor, explaining 56% of the variance. The results of this principal components analysis were highly consistent with the principal components analysis conducted by Colder et al. (2006), who also found evidence for a single factor. The items were, therefore, averaged and reverse coded so that higher scores reflect greater ND. The resulting ND scale was identical to the scale used by Colder et al. (2006), except that Colder's measure did not include the item relating to availability of public transportation. A Blom transformation, a well-validated transformation often used in behavior genetic and quasi-experimental studies (van den Oord et al., 2000), was applied to the ND measures because they were skewed. Finally, the measures were standardized (converted to a z-score) to facilitate interpretation of effect sizes. The assessment of ND was then computed for each offspring for their specific ages (in two–year age blocks because of the biennial assessments). Averages of ND across two longer age ranges were then computed. ND across the ages of 4–13 was available for 7,106 offspring and ND across the ages of 10–13 was available for 4,925 offspring. Table 1 presents the correlations of ND for the children in each 2-year age block with the average measures of ND across these age ranges. The average ND (from 4–13) correlated highly with the measures of ND at each age (r = .80–.84). Likewise, ND (from 10–13) was also highly associated with age-specific measures of ND (r = .88–.89). Therefore, the average measures largely reflect highly stable, long-term exposure to varying levels of neighborhood disadvantage.

Because the NLSY79 included all individuals in the original 1979 households and the CNLSY assesses every offspring of the women in the NLSY, the combined dataset includes three levels of nesting: the NLSY79 household level, the individual women in the NLSY79 (the mothers in the current study), and the offspring of the women in the NLSY (the CNLSY sample). This clustering enables the variation in exposure to ND to be broken down at each level. Intra-class correlations, the dividing of variation in ND at each level by the total variation in ND, estimate the extent to which variability in ND is shared with one's cousins, one's siblings, or is unique to individuals within a nuclear family. In the present analyses, approximately one-third (ICC = .34) of the variation in ND (at ages 4–13) was shared among all cousins in an extended family. In addition, siblings within a nuclear family shared approximately half (ICC = .52) of their exposure to ND across the ages of 4–13 years. Siblings, therefore, shared 86% (34%+52%) of the variability in ND. As such, only 14% of the variability in exposure to ND across the ages from 4–13 was found to be unique to individuals within a nuclear family (i.e., was not also shared with one's siblings or cousins), indicating that the sharing of reliable differences in ND was very high.

The sources of variation in ND at ages 10–13 years were similar. All cousins within an extended family shared 26% of total variation in ND. In addition, siblings shared 47% of the total variation in ND, indicating that 73% of the variation in ND in the sample was shared among siblings. Only 27% of the variation in ND is unique to individuals within a family at these ages. Thus, across both age ranges, reliable variation in ND was almost totally shared by all siblings growing up in the same nuclear family, but there was considerable variation in ND among cousins within the same extended family. This provides an opportunity to use cousin comparisons to test ND effects.

Maternal, familial, and individual-level covariates—Women in NLSY79 reported their total family income in each assessment (converted to 1986 inflation-adjusted dollars). This includes all income received by the woman and her spouse (if married), including food stamps and other forms of governmental support. Income from a cohabiting partner was not included, but previous reports suggest few substantive differences when this source of income is included in analyses (Avellar & Smock, 2005). A child-specific measure of family income (averaged across birth and 13 years) was calculated to assess the financial resources available to each individual offspring across childhood. In addition, a measure of family income when the women were approximately 30 years old was calculated to index overall earning capacity. If family income was missing when the mothers were 30, income between the ages of 27 and 33 were used. Family income at the mother's age of 30 was (a) available for an overwhelming majority of the women in the study, (b) highly predictive of future earnings, and (c) was not confounded with income received from their parents, which was included in the measure of family income at younger ages if a woman was still living at home (D'Onofrio et al., 2009a). Both measures of income were log transformed and then standardized as z-scores for ease of interpretation.

Mother's intellectual abilities were measured in 1980, using the Armed Services Vocational Aptitude Battery, an assessment instrument that taps knowledge and skills in 10 areas. A composite score was calculated, which approximates Armed Forces Qualification Test scores, a measure that has shown strong correlations with other measures of intellectual abilities (e.g., Orme, Brehm, & Ree, 1994). As such, the measure is an approximate equivalent of a measure of general intelligence. The scores were converted to percentile scores. Years of completed education by the year 2004 indexed maternal educational attainment.

Maternal history of her own delinquency was based on a 12-item measure of delinquent and aggressive behaviors (e.g., destruction of property, physical aggression, theft), the Self-

reported Delinquency (SRD) measure (Elliott & Huizinga, 1983), which was completed during the initial assessment of the NLSY79. Participants reported their engagement in the behaviors during the previous year. The SRD has been found to be reliable and valid and is frequently used in contemporary delinquency research (e.g., Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996; Rodgers, Buster, & Rowe, 2001). Item responses were summed to create a continuous measure of maternal delinquency. Maternal delinquency scores were then regressed on the woman's age during the initial assessment (14–22 years) to control for age differences at the time of the assessment.

The NLSY79 included an assessment of the mother's alcohol use and abuse in the 1994 wave. Participants were asked to report the number of binge episodes in the previous month and respond to twenty-five items assessing lifetime symptoms of alcohol abuse and dependence (D'Onofrio et al., 2007). The number of symptoms and frequency of binge drinking were used in the present analyses to control for problematic drinking. Mothers also reported on the biological father of each offspring in the sample. As such, families could be categorized as intact or blended (a mother reported having children by more than one man). Finally, the specific maternal age at childbearing for each child was assessed, which has been shown to uniquely predict offspring conduct problems (D'Onofrio, et al., 2009b).

Mother-reported child conduct problems—Women reported on their 4–13 year old children's adjustment via the Behavior Problem Index (BPI; Peterson & Zill, 1986) at each wave of the assessment. This questionnaire asks mothers whether the items were not true (0), somewhat or sometimes true (1), or very true or often true (2) for the child under consideration. The BPI includes 13 externalizing items selected from the Child Behavior Checklist (CBCL; Achenbach, 1978), a well-validated assessment for children. Factor analysis (D'Onofrio et al., 2008) indicated that the BPI externalizing items load on three factors: conduct problems (CPs), attention-hyperactivity-impulsivity problems, and oppositional problems. One set of the current analyses focused on mother-reported CPs, which is based on seven items that substantially overlap with other population-based longitudinal studies (Fergusson & Horwood, 2002; Moffitt, et al., 1996). Previous analyses of NLSY data have shown rank-order stability in levels of CPs across childhood and have demonstrated predictive links between childhood CPs and externalizing problems in adolescence (Lahey et al., 2006). The mean CP scores across 4–13 years was calculated by taking the mean of z-scores for CPs at each assessed year of age, standardized within each age (n = 9,440). A Blom transformation was used to reduce nonnormality in the mean CP variable. CPs at each age were highly correlated with the mean across 4–13 years (r =.75–.81) (also see D'Onofrio, et al., 2007). Mean CPs in this age range have been found to be valid in the sense of predicting conviction for at least one nontrivial criminal offense (not including drug convictions) during 14–19 years of age (Lahey et al., 2006).

Youth-reported delinquency—Children in the CNLSY who were at least 10 years old (but would not turn 15 by the end of calendar year) reported on their own delinquency using seven items from the SRD (Elliott & Huizinga, 1983). These SRD items were: hurt someone badly enough to need bandages or a doctor; lied to parent about something important; took something from a store without paying for it; intentionally damaged or destroyed property that didn't belong to you; had to bring your parent(s) to school because of something you did wrong; skipped a day of school without permission; and staying out overnight without permission. Item response choices were "never in the last year (coded as 0)," "once in the last year (1)," and "twice or more in the last year (2)." These items were selected for the CNLSY from the full SRD because they are high-prevalence activities that are highly correlated with more severe, serious delinquent behaviors. The 7-item SRD correlates highly (r > .90) with a 17-item SRD-based measure that was given to offspring in the CNLSY in

some waves (Lahey, et al., 2008), suggesting that the shorter assessment measure assesses risk for a broad range of serious delinquent activity. The 7-item measure in this age range also exhibits excellent criterion validity as the measure strongly predicts later conviction for at least one nontrivial criminal offense (not including drug convictions) during ages 14–19 years (Lahey, et al., 2008). The child self-report of CPs was based on the average of reported activities on the 7-item measure across 10 to 13 years old (n = 6,097). A Blom transformation was used to normalize the averaged CP variable.

Statistical Analyses

Tests of sample bias—A number of families and children in the study were not included in the current analyses because the maternal reports of ND were not collected at each wave of the CNLSY and not all offspring were assessed during each target age range (e.g., they were too old to be assessed by the maternal report or were not old enough yet to complete the self-report). The first set of analyses, therefore, compared those offspring included in the analyses to excluded offspring. Understanding the selection biases related to the two subsets of data will be important for contextualizing the results. The analyses were based on t-tests for continuously distributed variables and logistic regressions for categorical variables.

Associations between neighborhood disadvantage and covariates—The second set of analyses tested associations between measures of ND and the covariates to assess the extent to which ND covaries with other risk factors. Significant associations would suggest that the covariates have the potential to confound the statistical relation between ND and offspring CPs.

Analyses of the association between neighborhood disadvantage and offspring conduct problems—Because the NLSY79 assessed all sisters (mothers of the CNLSY offspring) in the household, and because the CNLSY assessed all offspring of the sisters from the NLSY79 sample, it is possible to compare offspring who are cousins. The third set of analyses explored the association between ND (ages 4–13) and offspring CPs (reported by mothers at 4–13 years and reported by youth across 10–13 years) using statistical covariates and a quasi-experimental approach—the comparison of cousins differentially exposed to levels of ND. The analyses were conducted using hierarchical linear models (HLMs), which are regression-based analyses that account for the non-independence of the observations (e.g. multiple offspring from the same family; Raudenbush & Bryk, 2002). More details concerning the fitting of HLMs to the current dataset are reported elsewhere (D'Onofrio, et al., 2009b; D'Onofrio, et al., 2007).

A series of analyses were conducted to examine whether ND has an independent effect on offspring CPs, or whether the statistical association is better explained by other correlated factors. First, an HLM examined the linear effect of ND on offspring CPs, controlling for offspring sex and race-ethnicity, to estimate the association. Second, the quadratic term for ND was added to determine if the association was nonlinear. Third, a series of HLMs explored whether the association is moderated by several factors, including offspring sex and race-ethnicity. Fourth, an HLM included the measured statistical covariates to determine if correlated risks account for the statistical association. If ND has an independent effect on offspring CPs, the association would remain robust to the measured covariates.

Fifth, an HLM compared cousins who varied in their childhood exposure to ND, rather than comparing unrelated individuals, as in the previous models. This analysis explored whether siblings growing up in the same family who were exposed to less ND on average have fewer CPs than their cousins who grew up being exposed to greater ND during childhood. As the intra-class correlations showed, a third of the total variation in ND at ages 4–13 years was

shared with one's cousins. The comparison of cousins differentially exposed to levels of childhood ND controlled for many unmeasured "third variables" that could account for the statistical relation between ND and offspring CPs. These analyses used a flexible statistical approach that generates accurate between and within-extended family estimates, consistent with fixed-effects models (Neuhaus & McCulloch, 2006). This approach is based on including two terms for ND in the HLM, the mean ND for all individuals in an extended family (cousins) and the deviation between the average extended-family ND and the mean ND of each nuclear family (all sibling offspring of each mother). The parameter associated with the mean level of ND for all cousins estimates the between extended-family effect (the comparison of unrelated individuals), whereas the parameter associated with the deviation of each mean nuclear family ND and the grand mean for all cousins, quantifies the effect of ND when comparing cousins.

Quasi-experimental test of the hypothesis—An HLM that included both the measured statistical covariates and the comparison of cousins was fit to the data to combine both of these two approaches to controlling confounds of the association with ND. If the influence of ND during childhood is independent of selection effects, then the association between ND and offspring CPs would be robust to the inclusion of the measured covariates and when comparing cousins differentially exposed to ND (Rodgers, et al., 2000). The comparison of cousins automatically and completely rules out all measured and unmeasured genetic and environmental factors shared by cousins as potential confounds. In addition, the inclusion of measured covariates helps to minimize the influence of factors that vary between cousins and thus are not controlled when comparing cousins.

The models that controlled for statistical covariates were conducted on five multiply imputed datasets which estimated missing values for the maternal/familial-level risk factors. Multiple imputation provides more accurate estimates than list-wise deletion (Little & Rubin, 1987; Schafer & Graham, 2002). Using this approach results in unbiased parameter estimates, given the assumption that the missing values are missing at random or completely at random. The following variables were used to simulate the missing values: mean ND (ages 4–13 and 10–13), the offspring's race-ethnicity, maternal intellectual abilities, maternal highest grade in school completed by 2004, maternal income at the age of 30, maternal history of delinquency, maternal average age at childbirth, whether the mother had children with more than one man, maternal history of binge drinking, maternal history of alcohol abuse and dependence symptoms, and mean offspring CPs. Multiple imputation was not used to estimate ND or CPs; rather, the method was only used to account for the missing covariates. Furthermore, a series of HLMs, referred to as sensitivity analyses, were fit to the data to examine whether characteristics of the families and offspring affected the substantive results.

RESULTS

Tests of Sample Bias

For the analyses exploring the effects of ND on mother-reported conduct problems at 4–13 years, 7,077 offspring had available data on both ND and CPs (out of a total of 11,431). Compared to those not included in the analyses, the offspring included had higher family income during childhood and later maternal age at childbearing. The mothers of children included in the present analyses had higher family incomes at the age of 30, more years of education, less delinquency during adolescence, less binge drinking, and less alcohol abuse/dependence. The families included in the analyses were more likely to be African American, more likely to be Hispanic, and less likely to be from a blended family. The mothers of

children used in the analyses did not differ on intellectual abilities from the mothers of children excluded.

The subset of the CNLSY data with maternal reports of ND and youth reports of delinquent behavior at 10–13 years included 4,524 individuals. Compared to those excluded from the analyses, the offspring in these analyses had lower family income during childhood and younger maternal age at childbearing. The mothers included in the analyses had lower intellectual ability scores. The families included in the analyses were more likely to be African American, more likely to be Hispanic, but less likely to be from a blended family. The included mothers did not differ in their family income at the age of 30, years of education, delinquency during adolescence, binge drinking, or reports of alcohol abuse/dependence.

In sum, the analysis of the effects of ND on offspring at 4–13 years old is based on a sample that contains fewer risks associated with child and adolescent CPs than the full sample, whereas the sample used to explore ND (10–13 years old) is at slightly greater risk or comparable to all offspring in full CNLSY sample. All of the covariates on which the included youth differed from the excluded youth are included in the final HLMs exploring the effects of ND to help account for these sample biases.

Associations between Neighborhood Disadvantage and Covariates

Table 2 reports correlations between the measures of ND at the two overlapping age spans and the continuous covariates for the offspring and mothers included in the present analyses. ND was correlated with all of the continuous covariates, except for maternal history of alcohol abuse/dependence. Associations with categorical variables were also tested using t-tests (not tabled). ND at 4–13 years was associated with living in a blended family (*b* (mean difference) = 0.12 SD, *SE* = 0.04, p < .01). The same trend was found for ND at 10–13 years, but the difference was not statistically significant (*b* = 0.08 SD, *SE* = 0.05, p = .09). In comparison to non-white, non-Hispanic families, African American children tended to be exposed to more ND during childhood [(ages 4–13, *b* = 0.57 SD, *SE* = 0.04, p <.001) and (ages 10–13, *b* = 0.55 SD, *SE* = 0.05, p <.001)]. Hispanic offspring also tended to live in neighborhoods with greater disadvantage [(ages 4–13, *b* = 0.24 SD, *SE* = 0.05, p <.001) and (ages 10–13, *b* = 0.16 SD, *SE* = 0.05, p <.001)]. As such, ND covaries with family income (specific to each child at each age and when the mother was 30 years old), maternal age at childbirth for each child, maternal intellectual abilities, level of education, history of delinquency, binge drinking, living in a blended family, and being African American or Hispanic. Therefore, all of these confounded measured covariates are statistically controlled in the present analyses.

Analysis of the Association between Neighborhood Disadvantage and Offspring Conduct Problems (4–13)

Table 3 presents the parameter estimates and their standard errors for the models exploring ND and mother-reported CPs at 4–13 years. Model 1 estimated the association between ND and offspring CPs, controlling for offspring sex and race-ethnicity. The results suggest that every 1 standard deviation (SD) unit difference in greater ND is associated with 0.23 SD unit difference in greater offspring CPs (*SE* = 0.01, *p* < 0.0001). An HLM (not tabled) examined whether this association between ND and CPs was nonlinear (i.e. if the association was stronger in the most extreme neighborhoods). This was accomplished by estimating a quadratic term in addition to a linear term for ND. The quadratic term was neither large nor statistically significant (*b* = −0.003, *SE* = 0.009, *p* = 0.72). Thus, a quadratic term was not included in subsequent analyses.

A series of HLMs (not tabled) also tested possible moderators of the association between ND and offspring CPs by including interaction terms between the potential moderator and ND, controlling for both the main effects of ND and the moderator. The association between ND and offspring CPs was not found to be moderated by offspring sex ($b_{\text{interaction}} = -0.03$, $SE = 0.02$, $p = 0.10$) nor race-ethnicity, as the association was not different for Hispanic ($b_{\text{interaction}} = -0.05$, $SE = 0.03$, $p = 0.09$) or African American families ($b_{\text{interaction}} = -0.03$, $SE = 0.03$, $p = 0.30$) relative to Non-Hispanic white and other ethnic groups.

An HLM (Model 2 in Table 3) estimated the association between ND and offspring CPs while also controlling for a number of child-specific, maternal, and family-level characteristics to determine if the inclusion of these potential confounds would decrease estimates of the association of ND with CPs. The estimate for ND ($b = 0.19$, $SE = 0.02$, $p < 0.0001$) was only slightly attenuated compared to Model 1, indicating the association between ND and offspring CPs was quite robust to the inclusion of these measured covariates. Table 3 includes the parameter estimates associated with these simultaneously entered predictors and covariates. The parameter estimate for each variable represents the unique association between that variable and offspring CPs, controlling for all other variables in the equation.

As summarized in Table 3, Model 3 compared cousins within extended families (i.e., compared the offspring of women who were sisters in the NLSY) who were exposed to different levels of ND in the CNLSY, without maternal and family-level covariates. The parameter for the cousin comparison estimated the extent to which offspring who are exposed to greater ND on average than their cousins have more CPs. The magnitude of the cousin-comparison estimate for ND ($b = 0.26$, $SE = 0.04$, $p < 0.0001$) was quite similar to the estimate in Model 1 and identical to the comparison of unrelated individuals within the same model ($b = 0.26$, $SE = 0.02$, $p < 0.0001$), strongly suggesting that factors shared by cousins do not confound the association between ND and offspring CPs. In Model 4, the maternal and family-level covariates were included with the cousin comparison. These results strongly suggest that the association between ND and offspring CPs ($b = 0.14$, $SE = 0.05$, $p < 0.01$) is largely independent of both the measured covariates and the unmeasured factors that influence all members of an extended family. Two-thirds of the unadjusted association between ND and CPs ($b = 0.23$) was independent of these confounds.

Analysis of the Association between Neighborhood Disadvantage and Youth-Reported Delinquency at 10–13 Years

Table 4 presents the results of the HLMs exploring the association between maternal-rated ND at 10–13 years and self-reported delinquency at 10–13 years. Model 1 estimated the linear association between ND and delinquent behavior while controlling for offspring sex and race-ethnicity and found that every one SD unit increase in ND was associated with a 0.14 SD unit increase in self-reported delinquency ($SE = 0.02$, $p < 0.0001$). A series of HLMs (not tabled) tested whether a non-linear model fit the data better and the possible role of moderators. Adding the quadratic term for ND ($b = 0.01$, $SE = 0.01$, $p = .48$) indicated that a linear model for ND was sufficient. Offspring sex ($b_{\text{interaction}} = -0.02$, $SE = 0.03$, $p = .35$), Hispanic heritage ($b_{\text{interaction}} = -.05$, $SE = 0.04$, $p = .21$), and African American heritage ($b_{\text{interaction}} = 0.02$, $SE = 0.04$, $p = .57$) did not moderate the association of ND with delinquency.

As summarized in Table 4, Model 2 assessed the association between ND and offspring delinquency controlling for measured confounds. The association was generally robust to these measured covariates ($b = 0.11$, $SE = 0.02$, $p < 0.0001$). Model 3 examined whether cousins differentially exposed to ND reported more or less delinquency; the association between ND and delinquency ($b = 0.14$, $SE = 0.04$, $p < .005$) remained consistent with the

estimate based on the comparison of unrelated individuals, indicating that unmeasured factors that influence all members of an extended family do not confound the association between ND and delinquency. Model 4 compared cousins in the same extended families differentially exposed to ND while statistically controlling for the measured covariates. The association between ND and delinquency remained robust ($b = 0.11$, $SE = 0.04$, $p < 0.05$), implying that neither the measured covariates nor unmeasured factors shared by cousins account for the relation between ND and CPs during childhood.

It is important that the analyses reported in Tables 3 and 4 revealed a significant association between mother-rated ND and offspring CPs after an important set of measured and unmeasured confounds were considered for both mother-reported CPs and youth-reported delinquency. These associations within and between informants indicate that the association between ND and offspring CPs is not simply the result of rater bias (i.e., common method variance).

Sensitivity Analyses

A series of HLMs were conducted to test the robustness of the current results by testing critical assumptions in the analytical strategy and the assessment of ND and offspring CPs utilized in the current study (results available upon request). Similar findings to those presented were found when the measures of ND were not transformed, when the analyses were based on families with complete data (not relying on the use of multiple imputation), when controlling for the number of assessments in each age range (to explore whether offspring repeatedly assessed were different than those only assessed once), when exploring the highest level of conduct problems across each age range (instead of predicting the average), when shorter age ranges were used (e.g., the same results were found when predicting mother-reported CPs across the age ranges of 4–8 and 4–10 years old, even though the analyses included smaller samples), and when testing prospective associations between childhood ND from age 4 to 9 (rather than ages 4 to 13) and adolescent-reported CPs from age 10 to 13.

DISCUSSION

The present analyses, based on a large subset of the offspring of mothers from a representative sample of households followed over many years, found consistent evidence for independent effects of ND on CPs in childhood both within and across informants. ND was significantly associated with offspring CPs when comparing unrelated individuals, when comparing cousins, and when both comparing cousins and controlling for family and individual characteristics. This means that the present results indicate that ND is associated with increased risk for childhood CPs, even after controlling for salient measured risk factors that are highly correlated with both ND and CPs (e.g., family income and maternal history of psychopathology) and unmeasured risks that influence all individuals in an extended family. The present analyses strongly suggest that aspects of the environments associated with greater ND exert independent influence on CPs in children.

The present study does not prove that ND causes CPs in youth, however. Only randomized experimental studies demonstrate that exposure to a risk environment causes differences in outcomes. Although the present study is not experimental, it addresses some limitations of the existent experimental studies. Whereas the experimental studies have only tested the effects of moving out of, not into, disadvantaged neighborhoods, the current study considered all variation in ND. In addition, whereas experimental studies have been conducted with poor, ethnic minority samples, the present study included a socioeconomically and ethnically diverse sample, thereby maximizing generalizability of the findings.

The comparison of cousins rules out many confounds and has been proven to be an important quasi-experimental approach for the study of risk factors for externalizing problems (e.g., Geronimus, et al., 1994; Slutske, et al., 2008; Turley, 2003). Nonetheless, as mentioned previously, differences in environments experienced by members of one nuclear family but not one's cousins would not be ruled out as confounds. In addition, because cousins share only 12.5% of their genes, cousin comparisons are also only able to control for some genetic factors that may influence family exposure to ND (perhaps through their effects on parental psychological and/or substance use problems) and offspring CPs. Sibling-comparison designs (e.g, Aaronson, 1997) account for more genetic and environmental selection factors, but the approach requires sufficient variability in ND among siblings. The intraclass correlations for ND in the current sample indicated very little variation in ND unique to individuals within a nuclear family, precluding the use of a sibling-comparison approach.

By using a quasi-experimental design and including stringent statistical controls, the present analyses provided a rigorous test of the hypothesis that ND exerts an effect on offspring CPs. Nonetheless, additional studies designed to rule out the remaining alternative explanations are needed before one can conclude that the influence of ND is causal. In addition, the present findings do not reveal what aspects of the environments inherent in ND are responsible for its effects. It will be important, therefore, for future research to shed light on the processes through which ND exerts its influence. Several possibilities have been outlined by Gephardt (1997). One possible model of influence, the collective socialization model, would suggest that ND increases risk for offspring CPs because adults living in disadvantaged neighborhoods do not provide adequate assistance to parents in monitoring children's behavior and do not provide adequate prosocial role models to children living in the neighborhood. As a result, children living in disadvantaged neighborhoods do not experience the same level of positive socializing influences as children from more advantaged neighborhoods.

In contrast, the epidemic, or contagion, model of influence would suggest that youths in disadvantaged neighborhoods are at increased risk for exposure to antisocial peers who are likely to reinforce CPs and attitudes. Deviancy training has been shown to characterize the friendships of deviant youths and has been found to have a powerful effect on conduct problem development in early childhood (Snyder et al., 2005) and adolescence (Dishion, Spracklen, Andrews, & Patterson, 1996).

Whereas the collective socialization and epidemic models posit socially mediated processes, neighborhood institutional resource models posit that ND exerts its effects by limiting youths' access to positive community resources, like high quality schools and community centers, that promote prosocial development, or a consistent police presence to prevent opportunities for engagement in illegal activities. These models focus on differences in opportunities available across levels of neighborhood advantage. See Leventhal and Brooks-Gunn (2000) for a description of other models that could mediate the effects of ND.

Each of the models of influence described above could account for the influence of ND on conduct problems. Additional research using within-family and experimental designs is needed to identify mechanisms through which ND exerts its influences. Designs relying solely on measured covariates to rule out selection effects are in danger of confounding mechanisms of neighborhood influence with mechanisms of individual and family influences that covary with ND (Leventhal & Brooks-Gunn, 2000).

Several limitation of the present study should be noted. First, many CNLSY participants were not included in the analyses owing to missing data. Although the entire CNLSY

sample will eventually be representative of children born in the U.S., the subsamples of offspring analyzed for the current manuscript were not. Another limitation is that the present measure of ND relies solely on maternal perceptions. This measurement approach may contribute to inflated associations with CPs if mothers also report on offspring CPs. In addition, it is possible that mothers who rate their offspring as being high in CPs are subsequently more likely to perceive their neighborhoods as being disadvantaged. These concerns are addressed in the present study in analyses testing associations with youth-reported CPs. It is also unclear how well maternal perceptions accurately characterize neighborhood-level processes, although there is some evidence for associations between maternal perceptions and census-based measures (e.g., Sampson, 1997). A more effective approach for capturing dynamic, neighborhood-level social processes may be to use an approach that integrates perceptions provided by several individuals living in a neighborhood (Sampson, Morenoff, & Gannon-Rowley, 2002). Nevertheless, maternal perceptions are useful in capturing social processes present in disadvantaged neighborhoods that would otherwise be overlooked using “objective” measures, such as census tract data (Sampson, et al., 2002). The present measure of ND is also limited by its inability to differentiate individual aspects of ND, such as structural disadvantage and poor social cohesion. More precise specification of ND could aid in the differentiation of causal versus noncausal elements of ND and the identification of mechanisms linking ND to maladjustment, such as insufficient parental monitoring or a lack of prosocial role models. Another limitation is that the present findings may not be generalizable to the association of ND with delinquency during later adolescence. This concern is reduced because some studies have found that ND is associated with delinquency in late adolescence in ways similar to the present findings (Kling, et al., 2005; Roche, et al., 2007), although other studies have found ND is associated with decreases in CPs in late adolescence (Lynam, et al., 2000). The measure of ND used in the current paper reflected average exposure to childhood ND and did not explore whether variations in ND over time are associated with variations in CPs (e.g., Lynam, et al., 2000), which is a powerful test of causal influences. Intra-individual analyses would also be well-suited for examining timing-dependent effects of ND, which are not well understood at this time. Growth mixture models that utilize within-family comparisons would be particularly valuable in identifying ND effects within a developmentally informative framework. The current results provide corroborating evidence for the importance of ND in the etiology of childhood CPs, but fall short of answering many important questions regarding the circumstances that account for and moderate the influences of ND. Determining causal influences requires the use of various research methods, each with their own inherent strengths and weaknesses (Rutter, et al., 2001).

The present study did not find evidence for moderation of ND by gender or ethnicity. Although other studies have found evidence for ethnicity as a moderator (e.g., Chase-Lansdale & Gordon, 1996), these studies used more specific measures of neighborhood characteristics (e.g., male joblessness) than the broad measure used in the current study. Thus, it is possible that ethnicity is a moderator of specific aspects of neighborhood disadvantage that were not measured or were used as part of a composite measure of ND in the present study. Other studies have also found evidence for markedly different effects of ND on conduct problems in boys as compared to girls (e.g., Kling, et al., 2005). However, Kroneman, Loeber, and Hipwell (2004) found comparable effect sizes of ND for boys and girls in their review. It is possible that gender differences in ND effects may be a product of developmental timing. Whereas Kling et al. tested effects in middle and late adolescence, the current study tested effects of ND in childhood and early adolescence. Gender-differentiated mediators of ND, such as deviant peer affiliation (Schonberg & Shaw, 2007), may become more prominent in adolescence, resulting in the emergence of gender differences in ND during this time. Because much of the research on ND effects have used male-only samples or have not tested for interactions with gender, more research is

necessary to determine the circumstances and processes by which gender moderates the influence of ND.

Every possible moderating factor was not examined in the current study, however. The present analyses did not consider the possible moderating influences of individual-level traits, such as temperament; family-level factors (e.g., parenting traits); or larger contextual factors (e.g., area of the country). Much remains to be done to understand moderators of the possible causal influences of ND.

Responding to the call by Leventhal and Brooks-Gunn (2000) and other researchers to use designs that can help isolate the effects of ND, the current study provided evidence consistent with the hypothesis that ND contributes to risk for youth CPs. Although this hypothesis needs to be tested further using a variety of quasi-experimental (e.g., Rutter, 2007) and experimental designs that can rule out the remaining alternative explanations, the present results lend further credence to the idea that interventions targeted at facets of communities, not just specific families or individuals, could reduce the prevalence of psychological and behavioral problems that have significant personal and societal costs.

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Table 1

Associations among Assessments of Neighborhood Disadvantage Across Time

Neighborhood Disadvantage	**1**		**2**		**3**		**4**		**5**		**6**		**7**	
Measure	***r***	***n***	***r***	***n***	***r***	***n***	***r***	***n***	***r***	***n***	***r***	***n***	***r***	***n***
1. Average 4–13	1.00	*7106*												
2. Average 10–13	0.91	*4925*	1.00	*4925*										
3. Ages 4/5	0.83	*3395*	0.49	*1445*	1.00	*3395*								
4. Ages 6/7	0.84	*3784*	0.55	*2316*	0.65	*2642*	1.00	*3784*						
5. Ages 8/9	0.80	*3965*	0.59	*3118*	0.55	*2044*	0.63	*2875*	1.00	*2945*				
6. Ages 10/11	0.82	*4045*	0.89	*4045*	0.49	*1371*	0.55	*2195*	0.60	*2945*	1.00	*4045*		
7. Ages 12/13	0.83	*3729*	0.88	*3729*	0.46	*598*	0.51	*1387*	0.53	*2149*	0.60	*2849*	1.00	*3729*

Note: All correlations are significant at $p < .001$.

Table 2

Associations between measures of neighborhood disadvantage and continuous covariates.

	ND (Ages 4–13)[b]		ND (Ages 10–13)[c]	
Covariates	***r***	***n***	***r***	***n***
Child-Specific				
Family Income (Ages 0–13)	−0.44 [d]	6979	−0.38 [d]	4491
Maternal Age at Childbearing	−0.21 [d]	7077	−0.19 [d]	4524
Maternal/Family-Level[a]				
Family Income (Maternal Age = 30)	−.37 [d]	2591	−.35 [d]	1917
Maternal Intellectual Abilities	−.38 [d]	2726	−.34 [d]	1978
Maternal Highest Grade	−.26 [d]	2842	−.21 [d]	2057
Maternal History of Delinquency	.11 [d]	2716	.08 [d]	1966
Maternal Binge Drinking	.07 [d]	2770	.05 [d]	2038
Maternal Alcohol Abuse/Dependence	.03	2771	.02	2038

Note.

[a] Correlations are based on mean exposure to neighborhood disadvantage for all children of each mother.

[b] Out of a possible 7,077 offspring and 2926 mothers.

[c] Out of a possible 4,524 offspring and 2421 mothers.

[d] $p < .05$.

Table 3

Relation Between Neighborhood Disadvantage (ages 4–13) and Mother-Reported Childhood Conduct Problems at Ages 4–13 Years Using Quasi-Experimental and Statistical Controls

	Model 1		Model 2		Model 3		Model 4	
Variables	***b***	***SE***	***b***	***SE***	***b***	***SE***	***b***	***SE***
Neighborhood Disadvantage								
Unrelated Comparison	.23[a]	.01	.19[a]	.02	.26[a]	.02	.12[a]	.04
Cousin Comparison					.26[a]	.04	.14[a]	.05
Child-Specific Covariates								
Child Sex	−.28[a]	.02	−.28[a]	.02	−.28[a]	.02	−.28[a]	.02
Family Income (Ages 0–13)			−.08[a]	.02			−.07	.02
Maternal Age at Childbearing			−.023[a]	.002			−.02[a]	.00
Race-Ethnicity								
Hispanic	.05	.04	.02	.03	.03	.04	.02	.03
African American	.15[a]	.03	.10[a]	.03	.13[a]	.03	.10[a]	.03
Maternal/Family-Level Covariates								
Family Income (Maternal Age = 30)			−.04[a]	.02			−.04[a]	.02
Maternal Intellectual Abilities[b]			.07[a]	.02			.07[a]	.02
Maternal Highest Grade			−.03[a]	.01			−.03[a]	.01
Maternal History of Delinquency			.05[a]	.01			.05[a]	.01
Maternal Binge Drinking			.00	.03			.00	.03
Maternal Alcohol Abuse/Dependence			.08[a]	.03			.08[a]	.03
Blended Family			−.03	.03			−.03	.03

Note: Analyses are based on 7,077 offspring. Parameters are raw regression coefficients.

[a] $p < .05$.

[b] The measure of maternal intellectual abilities was standardized for ease of interpretation.

Table 4

Relation Between Neighborhood Disadvantage (ages 10 −13) and Self-Reported Delinquency at Ages 10 –13 Years Using Quasi-Experimental and Statistical Controls

	Model 1		Model 2		Model 3		Model 4	
Variables	***b***	***SE***	***b***	***SE***	***b***	***SE***	***b***	***SE***
Neighborhood Disadvantage								
Unrelated Comparison	.14[a]	.02	.11[a]	.02	.15[a]	.02	.12[a]	.02
Cousin Comparison					.14[a]	.05	.11[a]	.05
Child-Specific Covariates								
Child Sex	−.41[a]	.03	−.41[a]	.03	−.41[a]	.03	−.41[a]	.03
Family Income (Ages 0–13)			−.03	.03			−.02	.03
Maternal Age at Childbearing			−.013[a]	.004			−.013[a]	.004
Race-Ethnicity								
Hispanic	.16[a]	.04	.11[a]	.04	.16[a]	.04	.11[a]	.04
African American	.32[a]	.04	.25[a]	.04	.32[a]	.04	.25[a]	.04
Maternal/Family-Level Covariates								
Family Income (Maternal Age = 30)			−.06[a]	.02			−.06[a]	.02
Maternal Intellectual Abilities[b]			−.03	.02			−.03	.02
Maternal Highest Grade			−.01	.01			−.01	.01
Maternal History of Delinquency			.06	.01[a]			.06	.01[a]
Maternal Binge Drinking			.10[a]	.03			.10[a]	.03
Maternal Alcohol Abuse/Dependence			.03	.03			.03	.03
Blended Family			−.04	.03			−.04	.03

Note: Analyses are based on 4,524 offspring. Parameters are raw regression coefficients.

[a] $p < .05$.

[b] The measure of maternal intellectual abilities was standardized for ease of interpretation.