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Carolyn Koehnke

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Socioeconomic Status and Conduct Disorder: Evidence for a Moderating Effect on Genetic and Environmental Influences

by

Carolyn Koehnke

Dr. Irwin Waldman, PhD
Adviser

Neuroscience and Behavioral Biology

Dr. Irwin Waldman, PhD
Adviser

Dr. Irene Browne, PhD
Committee Member

Dr. Melvin Konner, PhD
Committee Member

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Carolyn Koehnke

Dr. Irwin Waldman, PhD

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An abstract of
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Abstract

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Previous research has consistently shown an association between lower socioeconomic status and increased rates of conduct disorder and antisocial behavior. However, little work has been done to assess the potential moderating effects of family and neighborhood socioeconomic on conduct disorder, rule-breaking, and aggression. This study aimed to examine those moderating effects, as well as determine the distinct pattern of association between socioeconomic status and conduct disorder and its two symptom dimensions. Our sample contained 728 twin pairs, ranging in age from 4 to 19 years drawn from the Georgia Twin Registry. Socioeconomic status data was gathered through self-report forms and US Census Tract data and conduct disorder was assessed using the Emory Combined Ratings Scales. We regressed conduct disorder and its symptom dimensions on our latent family SES variable, our latent neighborhood SES variable, in both a linear and curvilinear model. We found that our family SES variables explained a small portion of the variance in conduct disorder, rule-breaking, and aggression ($R^2 = .02$, $p = .01$, $R^2 = .02$ (.01), $p = .06$, and $R^2 = .01$ (.01), $p = .01$, respectively), but the latent family SES variable was not associated with conduct disorder ($\beta = .004$ (.03), $p = .88$, $R^2 = .00$). We further found that our latent neighborhood SES variable showed a small relationship with conduct disorder, rule-breaking, and aggression, but only explained a significant amount of the variance in conduct disorder ($\beta = -.07$ (.02), $p = >.01$, $R^2 = .005$, $\beta = .06$, $p = .01$, $R^2 = .01$, and $\beta = .08$, $p = .001$, $R^2 = .01$, respectively). In our behavior genetic analyses, we found evidence for additive genetic ($a^2 = .86$, .79, .77) and nonshared environmental influences ($e^2 = .18$, .13, .26) on conduct disorder, rule-breaking, and aggression respectively with significant rater contrast estimates. Finally, our moderator analyses showed that estimates of genetic and environmental influences varied across high and low SES groups, but there was no clear pattern of this moderating effect and all estimates had overlapping confidence intervals.

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Abstract

Previous research has consistently shown an association between lower socioeconomic status and increased rates of conduct disorder and antisocial behavior. However, little work has been done to assess the potential moderating effects of family and neighborhood socioeconomic on conduct disorder, rule-breaking, and aggression. This study aimed to examine those moderating effects, as well as determine the distinct pattern of association between socioeconomic status and conduct disorder and its two symptom dimensions. Our sample contained 728 twin pairs, ranging in age from 4 to 19 years drawn from the Georgia Twin Registry. Socioeconomic status data was gathered through self-report forms and US Census Tract data and conduct disorder was assessed using the Emory Combined Ratings Scales. We regressed conduct disorder and its symptom dimensions on our latent family SES variable, our latent neighborhood SES variable, in both a linear and curvilinear model. We found that our family SES variables explained a small portion of the variance in conduct disorder, rule-breaking, and aggression ($R^2 = .02$, $p = .01$, $R^2 = .02$ (.01), $p = .06$, and $R^2 = .01$ (.01), $p = .01$, respectively), but the latent family SES variable was not associated with conduct disorder ($\beta = .004$ (.03), $p = .88$, $R^2 = .00$). We further found that our latent neighborhood SES variable showed a small relationship with conduct disorder, rule-breaking, and aggression, but only explained a significant amount of the variance in conduct disorder ($\beta = -.07$ (.02), $p = >.01$, $R^2 = .005$, $\beta = .06$, $p = .01$, $R^2 = .01$, and $\beta = .08$, $p = .001$, $R^2 = .01$, respectively). In our behavior genetic analyses, we found evidence for additive genetic ($a^2 = .86$, .79, .77) and nonshared environmental influences ($e^2 = .18$, .13, .26) on conduct disorder, rule-breaking, and aggression respectively with significant rater contrast estimates. Finally, our moderator analyses showed that estimates of genetic and environmental influences varied across high and low SES groups, but there was no clear pattern of this moderating effect and all estimates had overlapping confidence intervals.

Introduction

Antisocial behavior can have devastating consequences for the life trajectory of those who exhibit symptoms and the communities that they live in (Colman, et al., 2009). Children who exhibit externalizing behaviors are more likely to experience symptoms of depression and anxiety, abuse alcohol, become parents at a young age, and report financial difficulties in adulthood (Colman, et al., 2009). Conduct disorder is a childhood behavioral disorder that is characterized by antisocial behaviors, such as lying, skipping school, bullying, and starting fights. Understanding the causes and pathways of conduct disorder provides the opportunity for community and family-based intervention measures.

Socioeconomic Status

Socioeconomic status is a complex concept that includes ideas about both economic status, including access to resources like nutrition, health care, housing, and education, and social status, such as prestige and social connections (Bradley & Corwyn, 2002). A frequently cited definition of socioeconomic status in the literature comes from James Coleman who described socioeconomic status in the context of capital (Coleman, 1988). Coleman brought up the idea of people needing access to three kinds of capital; financial, such as money to buy necessities, human, nonmaterial resources that children receive from their parents in the form of relationships and learning that is done inside the home, and social, which describes the connections of the individual to the community. He described how these three forms of capital act together through human relationships to create the broader concept of social capital, which can be used as a way to conceptualize socioeconomic status. This is the framework for understanding socioeconomic status that was adopted in the current study. Socioeconomic status describes an individual or community's access to capital, including material resources, nonmaterial resources, and social

connections (Bradley & Corwyn, 2002). However, there is also important evidence for race and ethnicity interacting with socioeconomic status to, in many circumstances, increase the vulnerability of individuals to the influences of socioeconomic status (Ulbrich et al., 1989; Williams & Sternthal, 2010). As a result and in line with guidelines for assessing socioeconomic status described below, we are also including measures of ethnicity in our definition of socioeconomic status.

It is widely accepted that socioeconomic status can impact all aspects of well-being. From a health perspective, neighborhood socioeconomic status has been linked to different outcomes in cardiovascular disease (Boylan et al., 2017), cancer (Kim et al., 2010; Coulon et al., 2016), substance use (Karriker-Jaffee, 2013), hypertension (Mujahid et al., 2008), and overall health (O'Campo et al., 2015; Arcaya et al., 2016). These studies typically used information from the US Census to model neighborhood socioeconomic status. Generally, these variables broke down into four categories: measures of poverty, measures of social status (typically assessed as type of occupation), measures of educational attainment, and household information, such as number of rented housing units or marital status of household leaders. In the context of Coleman's definition of capital, child development researchers have suggested using measures of those four categories, as well as ethnicity, to describe socioeconomic status in a research context (Mueller & Parcel, 1981; Entwisle & Astone, 1994).

Socioeconomic Status and Conduct Disorder

Since the 1990s, there has been increased interest in understanding how environmental factors, like socioeconomic status, contribute to the development of conduct problems and antisocial behavior in children and teenagers. The literature has consistently shown that lower socioeconomic status and neighborhood disadvantage are correlated with higher rates of conduct

problems and antisocial behavior, particularly in males (Loeber & Wilkstrom, 1993; Aneschensel & Sucoff, 1996; Ingoldsby, et al. 2006; Schonberg & Shaw 2007; Mrug & Windle, 2009; Ferguson, 2010; Sundquist et al., 2015; Devenish et al., 2017). Three main pathways have been hypothesized for how socioeconomic status and other neighborhood factors may lead to higher rates of conduct problems and antisocial behavior: parental influences, chronic stressors, and peer deviance.

Parental Influences

Socioeconomic status is significantly associated with parenting styles. Parents in low socioeconomic status neighborhoods are more likely to suffer from maternal depression, tend to overuse aggressive and harsh discipline styles, discipline inconsistently, and fail to provide a warm, supportive home life (Odgers, et al. 2012). It is hypothesized that neighborhood disadvantage, which is highly related to socioeconomic status, creates a set of conditions that are associated with high levels of stress and feelings of powerlessness, and these conditions interfere with the ability of parents to parent in a way that is most effective for their children (Schonberg & Shaw, 2007). These problematic parenting factors are theorized to interact with risk factors already present in the children, leading to a higher probability of that child developing conduct problems (Schonberg & Shaw, 2007; Odgers 2012).

This theory has been supported by studies that have shown that living in a poor neighborhood is associated with worse maternal behaviors, especially less parental warmth (Klebanov, et al. 1994; Taylor 2000; Schonberg & Shaw, 2007). Additionally, there has been evidence found that residing in a poorer neighborhood was associated with higher reports of stress levels (Allison et al., 1999). These characteristics of parents in low socioeconomic status households are important, because these types of parenting have consistently been associated with conduct

problems and youth antisocial behaviors (Sampson, 1994; Ge, 2002; Tolan, 2003; Barber, 2005). A 2003 study showed that both high levels of harsh, inconsistent parenting and low levels of supportive, involved parenting had a statistically significant, positive effect on levels of antisocial behavior (Brody et al., 2003). Additionally, they found that this effect was actually intensified by living in a low socioeconomic status neighborhood, meaning that the association between these problematic parenting styles and antisocial outcomes is stronger in lower-income neighborhoods. A 2005 study found that poverty is best linked to externalizing symptoms, such as antisocial behavior through economic stressors and inconsistent discipline (Grant et al., 2005). A 2007 review by Schonberg & Shaw found that, overall, the literature supports the idea that familial risk factors, particularly parental supervision, have a greater impact on child developmental outcomes in low socioeconomic status neighborhoods.

More recent studies have continued to support this hypothesis. A 2009 study found that negative parenting, characterized by high levels of harsh-inconsistent discipline and low levels of nurturing, was directly associated with children's externalizing behavior (Mrug & Windle, 2009). This study was significant in that its pathways accounted for the variations in children's externalizing behavior equally well for both boys and girls. Additionally, this study provided support for the influence of neighborhood socioeconomic status, rather than familial socioeconomic status, as its associations remained after the researchers controlled for family income and parental education. Additionally, a 2014 study found that the stress of living in lower-income, urban neighborhoods explained the positive relationship between a lack of parental support and externalizing behavior, although this study found evidence for a different pathway for boys and girls (Davis et al., 2014). Further support for this theory was found in a 2012 study that found that maternal warmth and parental monitoring almost entirely mediated the effects of

neighborhood and familial socioeconomic status on childhood antisocial behavior (Odgers et al., 2012).

Additionally, parents in low-income neighborhoods are more likely to experience depression and other negative mental health outcomes (Adler et al., 1993). However, the impacts of maternal depression on child antisocial behavior have not been well defined. In 2007, Schonberg & Shaw demonstrated that boys from low socioeconomic status neighborhoods were more likely to be exposed to maternal depressive symptoms than boys from higher socioeconomic status neighborhoods. However, this study was only able to explain the differences between boys with chronic conduct problems and boys with no conduct problems in lower middle class and lower socioeconomic status neighborhoods, not across all four neighborhood classifications they examined. In contrast, a 2017 study found that high rates of parental depression, were associated with childhood disruptive behavior disorders and that children and parents were more likely to have these psychological symptoms the closer they were to the poverty line (Acri et al., 2017). This suggests that the genetic component of maternal depression may be confounding results in a way that cannot be captured by studies that focus entirely on environmental factors. In 2017, a systematic review found that four studies showed that parental distress and depression at least partially mediated the relationship between socioeconomic status and adolescent externalizing problems (Devenish et al., 2017).

In 2017, Pinquart conducted a meta-analysis of 1,435 studies that evaluated the associations between parenting styles and externalizing behavior. This meta-analysis revealed that, overall in the literature, there is a small association between parenting styles and child externalizing behavior, and this association is particularly strong for psychological control ($r = 0.22$) and harsh control ($r = 0.21$). This is in line with the proposed model of harsh, inconsistent discipline

being a mediating factor for the effects of socioeconomic status on child antisocial behavior. An important note that came out of this meta-analysis was that maternal and paternal parenting behaviors had a similar association with antisocial behavior, meaning it may not matter if the child is being parented by a mother or a father. Additionally, the higher levels of antisocial behavior in boys than girls could not be explained by different styles of parenting, indicating that there may be a genetic or distinct environmental factor that is influencing differing rates and expression of conduct disorder in boys and girls.

Chronic Stressors

A second proposed pathway for the impacts of neighborhood deprivation and socioeconomic status on conduct disorder is the chronic stressors that are associated with growing up in these environments. Children growing up in poverty are exposed to much more stress than children who grow up in higher income environments (McLeod & Kessler, 1990; Steptoe & Feldman, 2001). MRI based studies of neural functioning have shown that there are associations between growing up in poverty and structural and functional differences in the brain, particularly in the hippocampus, amygdala, and prefrontal cortex (Luby et al., 2013; Noble et al., 2012; Kim et al., 2013; Javanbakht et al., 2015; Lawson et al., 2013; Hanson et al., 2012; Johnson et al., 2016).

In addition to research on the effects of poverty on children's neurobiology, studies have also looked at the effects of the stress imposed by poverty on children's antisocial behavior. One chronic stressor that has been proposed as a mediator for childhood antisocial behavior is exposure to violence, as children who live in disadvantaged neighborhoods are more likely to both witness and be victimized by violence (Farrell, et al. 2014). It has been theorized that exposure to violence disrupts the development of appropriate emotional responses and can teach

children violent responses by desensitizing them to the impacts of antisocial behavior. (Gabarino, et al., 1991; Ingoldsby & Shaw; 2002).

Despite a strong theoretical explanation, there has not been significant work substantiating violence as a mediating factor, rather than as a variable associated with participating in antisocial behavior and living in a low socioeconomic status area. In 1997, Farrell and Bruce found that girls but not boys showed an increase in their violent behavior after being exposed to violence, while a 1999 study found that there was a significant association between exposure to community violence and increases in antisocial behavior (Miller, et al., 1999). A 2000 review found that exposure to community violence was associated with antisocial behavior, although that review was unable to determine if community violence was a mediating factor for the relationship between socioeconomic status and antisocial behaviors (Overstreet, 2002).

Additionally, a 2005 study found that, in a sample of urban, African American youths, exposure to violence was only associated with internalizing, rather than externalizing symptoms (Grant et al., 2005). Finally, a 2008 study found that, while there was an association between home violence and poor mental health outcomes that include externalizing behaviors, there was no significant association between community violence and those outcomes (Fredland, et al., 2008)

One possibility, especially given the results of Fredland et al., is that the effects of exposure to violence on conduct disorder is primarily mediated through parenting behaviors. Since children and parents are living in the same neighborhood, they are likely exposed to similar levels of violence. Therefore, it may be that it is the response of the parent to this violence that is being passed on to the child through how they interact in the home. This possibility was actually discussed in Miller et al., who reported parent-child interactions in the home can moderate the relationship between child exposure to violence and conduct disorder (Miller et al., 1999). It is

also possible that, because violent behavior can be an outcome of having conduct disorder, the association between exposure to violence and conduct disorder is mediated through childhood associations with deviant peer groups.

Social Disorganization

A third way neighborhood factors may influence the development of conduct disorder is through social disorganization. Social disorganization can manifest itself in two ways; peer deviant behavior and lack of social cohesion, characterized by community-level apathy towards antisocial behavior. The theory is that, because more disadvantaged neighborhoods have reduced family resources and high population turnover, they are more likely to have low levels of cohesion and social institutions (Ingoldsby & Shaw, 2002). This lack of cohesion means that there is no community-level deterrence against antisocial behavior, as it is typically met with apathy, resulting in children having greater access to delinquent culture amongst their peers (Ingoldsby & Shaw, 2002).

A 1996 study looked specifically at the impact of peer deviance on youth problem behavior and assessed its interactions with socioeconomic status and collective socialization, a term used to define a lack of social cohesion as described above (Simons, et al., 1996). The researchers found that neighborhood disadvantage increases the probability that boys would affiliate with a deviant peer group and that this association increased the likelihood of conduct problems. They additionally found that a lack of collective socialization only explained increased conduct problems to the extent that it explained an increase in associations with deviant peers. This suggests that neighborhood disadvantage acts through deviant peer associations by not effectively discouraging those relationships.

This model has been supported in subsequent literature. A 2001 study found deviant peer pressure was able to predict future antisocial behavior and that adding deviant peer pressure into their model for antisocial behavior decreased the effects of neighborhood poverty, suggesting that it is one of the pathways that neighborhood poverty is acting through (Eamon, 2001). Further research has continued to support this hypothesis that neighborhood poverty may be promoting antisocial behavior by acting through deviant peer groups, which may be influenced by the collective socialization of the neighborhood (Rankin & Quane, 2002).

While there is support for the idea that deviant peers may play a role in mediating the effects of neighborhood disadvantage on youth antisocial behavior, it is interesting to note that all of the studies discussed above found that parenting behaviors influenced both youth association with deviant peer groups and the extent to which interacting with deviant peer groups would lead to future antisocial behavior. The majority of these studies found that either absent parenting (Pettit, et al. 1999; Rankin & Quane 2001) or lower quality of parenting (Simons, et al., 1996) moderated the influence of peer groups on antisocial behavior. Additionally, a 2014 study found that, while social cohesion was not a predictor of behavioral problems, the association between maternal depression and social cohesion was a significant predictor, further supporting the idea that many of these neighborhood-level factors are mediated through how they influence parenting behaviors at home (Brumsey et al., 2014).

The majority of the literature supports the idea that parental behavior is a significant pathway to explain the well-validated associations of socioeconomic status and antisocial behaviors. While exposure to violence and social disorganization also contribute, parents play an important role in mediating the effects of living in a disadvantaged neighborhood and can have a significant role in determining their children's developmental outcomes. There is more work to

be done on the role gender plays on these environmental pathways and how these environmental pathways interact with children's genetics.

Behavior Genetics Studies of Conduct Disorder

Against this backdrop, studies have been conducted analyzing the different genetic and environmental contributions to conduct disorder. A 2002 meta-analysis of twin and adoption studies found evidence of additive genetic, nonadditive genetic, shared environmental, and non-shared environmental influences on antisocial behavior, with the two largest contributors being additive genetic influences ($a^2 = 0.32$) and nonshared environmental influences ($e^2 = 0.43$) (Rhee & Waldman, 2002). Overall this pattern is relatively consistent in the literature, with many studies assessing conduct disorder and childhood antisocial behavior as one domain finding evidence for additive genetic and nonshared environmental influences with low to non-existing estimates for shared environmental influences (Slutske et al., 1997; Gelhorn et al., 2006; Dick, et al., 2005; Ferguson et al., 2010).

More recent work has aimed to see if these genetic and environmental influences vary across aggressive versus non-aggressive behaviors (i.e. rule-breaking) within the context of conduct disorder and antisocial behavior. There is mounting evidence that conduct disorder is best understood as mapping onto two distinct symptom dimensions; rule-breaking, which is characterized by behaviors like lying, skipping school, and substance use, and aggression, which is characterized by behaviors such as bullying, starting fights, and cruelty to people and animals (Frick et al., 1993; Lahey et al., 1998; Tackett et al., 2003; DeMarte, 2008; Burt, 2012). Several studies have found that aggressive behavior was more heritable, while rule-breaking behavior showed more shared environmental influences (Simonoff et al., 1998; Eley et al., 1999; Tackett et al., 2005; Val Hulle et al., 2018). Additionally, a 2009 meta-analysis of studies on antisocial

behavior, which is closely related to conduct disorder, found that genetic influences were significantly larger for aggressive behavior in comparison to rule-breaking behavior and that rule-breaking behavior showed stronger non-shared environmental influences than aggressive behavior (Burt, 2009).

There has also been some investigation into sex differences in the etiology of conduct disorder, both as a whole and assessed across its symptom dimensions. This work is currently inconclusive. Several studies have suggested that there are no sex differences on the influence of genetics and the environment on conduct disorder (Gelhorn et al., 2006; Van Hulle et al., 2007; Meier et al., 2011). However, some other studies (Eley et al., 1998; Bartels et al., 2011), including a meta-analysis (Burt, 2009) did find that there are significant differences in estimates for genetic and environmental influences between the sexes. This suggests that it is important to continue to assess the influence that sex may have as a moderator on any relationships between socioeconomic status and conduct disorder.

Aims

Despite consistent evidence that socioeconomic status is related to conduct disorder, to our knowledge there has been no study to date that has assessed whether family and neighborhood socioeconomic status may moderate the genetic and environmental influences on conduct disorder and its symptom dimensions. In the current study, we examined the distinct patterns of associations between socioeconomic status and conduct disorder and its two dimensions. We additionally evaluated how to best model socioeconomic status in our sample to determine if the previously identified variables provided meaningful information in the context of conduct disorder as opposed to health research. Finally, we evaluated whether our model of

socioeconomic status on both an individual and neighborhood level moderated the etiology of conduct disorder in our sample.

Methods

Participants

Data for this study were drawn from the Georgia Twin Registry, a representative sample of twins (51% female) born in Georgia between 1980 and 1991 ($M_{\text{age}} = 8.5$, $SD = 2.9$, range 4 to 19). The registry contained 1567 families, 838 of whom provided ratings on the psychopathology measures used in the present study. Mothers typically completed the questionnaires (53%), and the remaining questionnaires were completed either by fathers (1%) or both mothers and father (46%). Socioeconomic data on both the family and neighborhood level (e.g. percentage of adults with a college degree) was identified for 738 of those families, who comprised the final sample. The final sample contained 728 twin pairs and their 296 nontwin siblings. Regarding ethnicity, Eighty-four percent were Caucasian, 12% African American, 2% Hispanic, and 1% was of mixed ethnicity. The sample consisted of 392 dizygotic twin pairs (DZ: 54%) and 336 monozygotic twin pairs (MZ: 46%). Twin zygosity was determined from parental reports of the physical similarity of the twins using a 9-item scale previously validated against DNA polymorphisms (Bonnelykke, Hauge, Holm, Kristoffersen, & Gurtler, 1989).

Measures

Socioeconomic Data. Data on socioeconomic status was measured at both the family and neighborhood level. Self-reports of maternal and paternal ethnicity, maternal and paternal highest level of education, and family income level were used to assess family socioeconomic status. Education was assessed across ten levels for the highest completed level; 8th grade or less, 9th grade, 10th grade 11th grade, 12th grade, high school graduate, some college, college graduate,

master's degree, and doctoral or professional degree. Family income was divided into 12 levels, ranging from \$1-10,000 to more than \$150,000, with each level increasing the base by \$10,000. These six variables were combined into a single latent variable for phenotypic analyses. Families were additionally whether they were on Aid for Families with Dependent Children, but too few families in our sample reported this to use in our analyses.

Data on neighborhood socioeconomic status were obtained from the US Census and the US Census Geocoder function was used to translate the addresses of families in the Georgia Twin Registry into Census Tracts. Addresses that could not be matched to a Census Tract by Geocoder were translated into latitude and longitude coordinates, which were then run through the Geocoder program to produce Census Tracts. Eight neighborhood-level SES variables were created: percentage of adults who completed high school or its equivalent (e.g. obtained a G.E.D.), percentage of adults who obtained a college degree or higher, percentage of adults in prestigious occupations (defined as managerial or professional jobs), percentage of unemployed adults over the age of 25, percentage of families living below the poverty line, percentage of families receiving federal assistance, percentage of rented housing units in the neighborhood, and percentage of family households with unmarried parents. These eight variables were combined into a single latent variable for phenotypic analyses. Histograms for all of the socioeconomic status variables are included in the Appendix.

Conduct Disorder. Conduct disorder (CD) symptoms were assessed using the Emory Combined Rating Scale (Waldman et al., 1998). Parents reported the severity of 21 conduct disorder symptoms on a 5-point Likert-type scale, with 0 not describing the child at all and 4 describing the child very well. A conduct disorder symptom dimension was calculated by averaging each child's symptom scores across all of the items in the scale. Items were also

separated into rule-breaking and aggressive items, following evidence suggesting these dimensions are meaningfully separable and have distinct etiological influences (Burt et al., 2009; Van Hulle et al., 2018) and symptom dimensions reflecting these domains were calculated in the same way. Each child's mean symptom scores thus ranged from 0 to 4 per symptom dimension, indicating the severity of his or her level on each of the three CD symptom dimensions. Cronbach's Alphas were 0.80, 0.77, and 0.62 for conduct disorder, rule-breaking, and aggression respectively.

Data Analysis

All analyses were conducted using Mplus (Version 8; Muthén & Muthén, 1998-2017) using full-information maximum likelihood (FIML), which produces less biased parameter estimates compared with listwise and pairwise deletion in the presence of missing data (Enders & Bandalos, 2001). Data was analyzed using the "cluster" option, which nested individual twins within twin pairs to account for non-independence between twins. The MLR estimator (maximum likelihood with robust standard errors), which accounts for non-normality of variables, was used in all analyses.

Phenotypic Relationships between Socioeconomic Status and CD. The CD symptom dimensions were regressed on the latent family SES variable and latent neighborhood SES variable in six separate regressions. These relations were modeled as both linear and curvilinear, to test the possibility that CD symptom dimensions were only related to SES at extreme levels of SES. These analyses were repeated for rule-breaking and aggression dimensions separately. Finally, sex was examined as a moderator of the relations between the CD symptom dimensions and SES.

Goodness-of-fit. The fit of these models was assessed using the Akaike Information Criterion (AIC), Bayesian Information Criterion (BIC), Chi-Squared Value, Root Mean Square Error of Approximation (RMSEA), Comparative Fit Index (CFI), Tucker-Lewis Index (TLI), and Standardized Root Mean Square Residual (SRMR). AIC and BIC are relative fit indices, so the best fitting model was identified as the model with the lowest AIC and BIC. CFI, TLI, RMSEA, and SRMR are absolute fit indices; good fit was indicated by high CFI and TLI scores (.8 indicates acceptable fit and .95 indicates good fit), low RMSEA scores (.08 is acceptable and .05 is good), and low SRMR scores (0.05 or lower indicate a good fit). For behavior genetic models, although we considered all model fit indices in assessing relative fit, we relied primarily on BIC to adjudicate among models (Loehlin, 2004; Markon & Krueger, 2006).

Behavior Genetic Analyses. We controlled for age and sex, to account for mean level age and sex differences and to avoid over-estimation of environmental influences based on twins being the same age and sex. First, we conducted univariate behavior genetic analyses of conduct disorder, rule-breaking, and aggression symptom dimensions to estimate the underlying etiological influences on each dimension. In these behavior genetic models, additive genetic influences (A), nonadditive genetic influences (D), shared environmental influences (C), and nonshared environmental influences (E) are estimated from the MZ and DZ twin correlations. It is assumed that additive genetic influences are correlated 1.0 for MZ twins and 0.5 for DZ twins because they share an average of 100% or 50% of their genes, respectively. Nonadditive genetic influences are correlated 1.0 for MZ twins because they share 100% of their genes, but only 0.25 for DZ twins, as they have only a 25% chance of receiving the same gene from both parents. Shared environmental influences are assumed to be correlated 1.0 between both MZ and DZ twins, as these are environmental influences that both twins experience. Nonshared

environmental influences are assumed to be uncorrelated for both MZ and DZ twins, as they represent environmental influences that are experienced by only one twin.

We initially tested six possible models of conduct disorder, ACE, ACE + s, ADE, ADE + s, AE, and AE + s, where s represents a sibling interaction parameter, which describes the tendency for raters to score twins more differently from each other than they actually are. We then tested the same six models for both the rule-breaking dimension and the aggressive dimension of conduct disorder symptoms.

Next, we tested the hypothesis that SES would moderate the magnitude of etiological influences on conduct disorder, rule-breaking, and aggression dimensions. First, we identified the family and neighborhood SES variable that accounted for the most variance in and was significantly related to each conduct disorder dimension. Family income was selected to represent family SES and percent of people in prestigious occupations was selected to represent neighborhood SES. These variables were then each dichotomized at the mean and recoded to identify families that fell above and below the mean. They received a score of 0 if below the mean and 1 if they were above the mean. The low neighborhood SES group contained 385 twin pairs (66%), 213 were DZ (55%) and 172 were MZ (45%). The high neighborhood SES group contained 303 twin pairs (44%), 158 DZ pairs (52%) and 145 MZ pairs (48%) in the high neighborhood SES group. The low family SES group contained 373 twin pairs (60%), 202 were DZ (54%) and 171 were MZ (46%). The high family SES group contained 250 twin pairs (40%), 138 DZ pairs (55%) and 112 MZ pairs (45%). Using the best fitting model from the univariate analyses, we contrasted models in which the etiological influences were constrained to be equal across levels of SES and models in which they were freely estimated. The resulting models were compared using the aforementioned fit statistics.

Results

Descriptive Statistics of Family Socioeconomic Status

The majority of our sample (85% of mothers and 84% of fathers) identified as white, with African Americans making up the next largest ethnicity group (12%). The majority of our sample had completed high school or some level of college education (80% of mothers and 76% of fathers). The most commonly reported income level (16%) was \$40,000 to \$50,000. The next most common income levels were \$30,000 to \$40,000 (14%) and \$50,000 to \$60,000 (13%), meaning that 43% of families had an income between \$30,000 and \$60,000. Family socioeconomic status data were gathered in 1992-1993, and reported results are not adjusted for inflation. For full results, see Table 1. Histograms revealed an approximately normal distribution for the family income variable and normal distributions with a left skew for the education variables, whereas ethnicity variables showed a non-normal distribution with the bar indicating white ethnicity being substantially higher than every other bar (Figures 1-5).

Descriptive Statistics of Neighborhood Socioeconomic Status

The families who provided behavioral data in this study resided in 28 different states, with the majority residing in Georgia. An average of 55% of adults graduated high school or its equivalent and 34% graduated from college. Histograms showed that the distribution of adults who graduated high school or its equivalent was approximately normal with a left skew, while the distribution of adults who graduated college was less normal and showed a right skew. An average of 38% of adults over the age of 25 reported holding prestigious occupations and this was normally distributed. Unemployment (5%), families living below the poverty line (11%), families receiving federal aid (7%), households with unmarried parents (26%), and rented

housing units (31%) all showed a normal distribution with a large right skew. Neighborhood ethnicity was assessed as percentages of African American and Hispanic residents. Data on residents of Hispanic ethnicity was sparse, with 64.64% of Census Tracts missing data on Hispanic ethnicity status. See Table 2 for full percentages and Figures 6-15 for histograms.

Latent Variable Measures of Family and Neighborhood SES

Table 4 describes the factor loadings of each individual variable on the latent factors of family SES and neighborhood SES. All variables except for maternal education loaded significantly onto the family SES latent variable, with maternal and paternal ethnicity loading most highly. All of the neighborhood SES variables loaded significantly onto the neighborhood SES latent variable, with the education variables and percent prestigious occupation loading the most significantly.

Family income and percent prestigious occupation were selected as the best representatives of family SES and neighborhood SES, respectively. They were initially identified as the only variables that had a statically significant relationship with conduct disorder dimensions, and further analyses revealed that the individual variables explained the same proportion of variance in conduct disorder as did the latent SES variables. As such, these two variables were used in later moderation behavioral genetic analyses as representatives of family and neighborhood SES.

Main Effects of Family SES

The associations of the family SES variables with conduct disorder, rule-breaking, and aggression are shown in Table 5. Multiple regressions of conduct disorder dimensions with family SES indicated that only family income was significantly related to conduct disorder ($\beta = -.09$ (.04), $p = .01$). The family SES variables accounted for a small percentage of the variance in

conduct disorder ($R^2 = .02$, $p = .01$). The latent family SES variable was not significantly associated with conduct disorder ($\beta = .004$ (.03), $p = .88$, $R^2 = .00$). When rule-breaking and aggressive symptoms were analyzed separately, we again found that only family income was significantly associated with aggression ($\beta = -.08$ (.04), $p = .01$) while both family income and father's education level were significantly associated with rule-breaking ($\beta = -.08$ (.04), $p = .02$ and $\beta = -.09$ (.04), $p = .04$, respectively). Family SES variables explained a small percentage of the variance in rule-breaking and aggression ($R^2 = .02$ (.01), $p = .06$ and $R^2 = .01$ (.01), $p = .01$, respectively). The family SES latent variable was not significantly associated with rule-breaking or aggression ($\beta = .01$ (.02), $p = .74$ and $\beta = -.01$ (.03), $p = .81$) and did not explain any of the variance in conduct disorder ($R^2 = .00$).

We also tested the possibility that family SES was related to conduct disorder symptom dimensions in a curvilinear fashion. In the multiple regressions, the variance explained in conduct disorder did not increase with the addition of the squared term of family income ($R^2 = .02$ (.01), $p = .01$). When the squared term of the family SES latent variable was included in the model, the percentage of variance explained increased substantially, but was not significant, likely because of the high standard error ($R^2 = .15$ (.13), $p = .23$). When the squared term of family income was added to the multiple regression of rule-breaking and aggression on family SES variables, the percentage of variance explained remained unchanged ($R^2 = .02$ (.01), $p = .01$ and $R^2 = .01$ (.01), $p = .07$, respectively). The addition of a squared term of latent family SES to the models examining the association between rule-breaking and aggression increased the percent of variance explained, although it was not significant ($R^2 = .01$ (.01), $p = .38$ for both variables).

Main Effects of Neighborhood SES

When we regressed conduct disorder on a latent variable of neighborhood SES in a linear model, we found a small but significant relationship ($\beta = -.07$ (.02), $p = .01$). Neighborhood SES variables accounted for 0.5% of the variance in conduct disorder, but this small effect was non-significant. When conduct disorder was regressed on each individual component of neighborhood SES, the only association that was identified was with the percentage of adults over the age of 25 in prestigious occupations ($\beta = -.92$, $p = 0.03$). When rule-breaking and aggression symptoms were separated, neighborhood SES did not explain a significant amount of the variance in either dimension as the R^2 estimates hovered around zero and were non-significant ($R^2 = .01$, $p = 0.09$ and $R^2 = .01$, $p = .44$ respectively). However, both rule-breaking and aggression showed a similar, significant association with the latent variable of SES ($\beta = .06$, $p = .01$ and $\beta = .08$, $p = .001$).

All three dimensions of conduct disorder were also tested in a curvilinear model. In this model, conduct disorder showed no significant association with neighborhood SES ($\beta = -.02$ (.02), $p = .44$). Neighborhood SES was estimated to account for 0.4% of the variance in conduct disorder but the effect was non-significant. When the rule-breaking and aggression dimensions were assessed separately, the previously identified association between those symptom dimensions and neighborhood SES disappeared ($\beta = -.02$ (.02), $p = .15$ and $\beta = .01$ (.02), $p = .77$). See Table 6 for full results of the association between neighborhood SES and conduct disorder dimensions.

Sex Moderation

We next tested the hypothesis that sex moderates the relationship between latent family and neighborhood SES and conduct disorder. Our results indicate that boys display more

symptoms of conduct disorder ($\beta = .34 (.26)$, $p < .001$), but the interaction term of sex and family SES were not significant ($\beta = .11 (.22)$, $p = .62$). Sex also did not moderate the relationship between the latent neighborhood SES variable and conduct disorder ($\beta = .02 (.02)$, $p = .40$). For both family and neighborhood SES, sex explained a small amount of the variance in conduct disorder ($R^2 = .03 (.01)$, $p = < .001$ and $R^2 = .01 (.005)$, $p = .01$ respectively).

Behavior Genetic Analyses

Univariate Analyses. We fit univariate behavior genetic models for a composite measure of conduct disorder symptoms, as well as for measures of the two symptom dimensions of aggressive and rule-breaking. Fit statistics and parameter estimates are reported in Table 7. An AE + S model with significant rater contrast effects emerged as the best fitting model for conduct disorder, rule-breaking, and aggression symptom dimensions ($s = 0.02^{***}$, 0.01 (n.s.) and 0.02^{**} respectively). For conduct disorder and rule-breaking, an ACE model with rater contrast effects fit best, but these models were excluded as C was estimated at zero. Parameter estimates indicated appreciable additive genetic (0.86 , 0.77 , and 0.91 respectively) and nonshared environmental (0.18 , 0.26 , and 0.13 , respectively) influences. There was no evidence for nonadditive genetic or shared environmental influences on any of the conduct disorder dimensions. Finally, taking into account the additive genetic influences' parameter estimates and their confidence intervals, there was no evidence that the estimates were significantly different across the symptom dimensions.

SES Moderator Analyses. We then tested the hypothesis that family and neighborhood SES would moderate the etiological influences on the conduct disorder symptom dimensions. For all conduct disorder symptom dimensions, models in which the etiological influences were

free to vary across levels of family and neighborhood SES fit best, indicating that these parameter estimates vary as a function of SES.

For conduct disorder, estimates of additive genetic influences were higher in the low SES groups ($a^2 = .89$ for family SES and $.90$ for neighborhood SES) compared with the high SES group ($a^2 = .78$ for family SES and $.77$ for neighborhood SES). Non-overlapping confidence intervals suggest that these parameter estimates are significantly different between the two groups. For rule-breaking, estimates of the genetic influences were slightly higher in the high family SES group ($a^2 = .75$) compared with the low family SES group ($a^2 = .72$), although the confidence intervals were overlapping suggesting that this difference is not statistically significant. In contrast, estimates of the genetic influences on aggression in the high family SES group ($a^2 = .43$) were substantially lower compared to the low family SES group ($a^2 = .91$). These results also had overlapping confidence intervals, suggesting that the difference is not statistically significant. However, the standard error for the heritability estimate in the high family SES group was substantially higher than the standard error for the low family SES group (.25 and .03 respectively), which would contribute to a wider confidence interval for the high family SES group.

The genetic influences on rule-breaking and aggression in the neighborhood SES analysis showed the opposite pattern. Here, genetic estimates for the rule-breaking dimension were higher in the low neighborhood SES group ($a^2 = .80$) compared to the high neighborhood SES group ($a^2 = .52$). However, estimates of the genetic influences on aggression were higher in the high neighborhood SES group ($a^2 = .86$) compared to the low neighborhood SES group ($a^2 = .75$). Similar to the family SES analyses, these results were not statistically significant as the estimates had overlapping confidence intervals. However, the high SES group in both the rule-breaking

and aggression groups had a higher standard error than the low SES group (.18 and .06) which may have contributed to the lack of significance.

Discussion

To our knowledge, this is the first behavioral genetic study to examine family and neighborhood SES as moderators of the heritability of conduct disorder, rule-breaking, and aggression. Previous studies have demonstrated that conduct disorder is associated with SES such that living in a lower SES neighborhood is associated with higher rates of conduct problems and antisocial behavior (Loeber & Wilkstrom, 1993; Aneschensel & Sucoff, 1996; Ingoldsby, et al. 2006; Schonberg & Shaw 2007; Mrug & Windle, 2009; Sundquist et al., 2015; Devenish et al., 2017) and that conduct disorder is highly heritable (Rhee & Waldman, 2002; Slutske et al., 1997; Gelhorn et al., 2005; Dick, et al., 2005; Ferguson et al., 2010). There has been some work suggesting that socioeconomic status acts as a moderator for the genetic and environmental influences on antisocial behavior, but none of these studies assessed conduct disorder specifically or looked at differences across rule-breaking and aggressive behaviors (Tuvblad et al., 2006; Middeldorp et al., 2014; Burt et al., 2016). In this study, we sought to examine the relationship between family and neighborhood SES and conduct disorder and its symptom dimensions.

In sum, we found that both family and neighborhood SES explain a small portion of the variance in conduct disorder. We were able to identify that family income and percentage of adults in prestigious occupations accounted for the relationship between family and neighborhood SES and conduct disorder respectively. We found evidence that conduct disorder is primarily influenced by additive genetic and non-shared environmental influences across both symptom dimensions and that these influences are moderated by family and neighborhood SES.

Finally, we found evidence that suggests that family and neighborhood SES factors may act differently on the two symptom dimensions. Rule breaking showed more genetic influences in the high family SES group than the low family SES group, while there were more genetic influences in the low neighborhood SES group than the high neighborhood SES group and aggression showed the opposite pattern.

Interpretation and Implications of Findings

Our findings revealed that family and neighborhood socioeconomic status each explained a small portion of the variance in conduct disorder, with family SES explaining more than neighborhood SES. Prior work has established that, of the three pathways through which SES may influence child externalizing behavior (e.g. through parenting, chronic stressors, or social disorganization), the pathway most supported by the literature is parenting style (Sampson, 1994; Simons et al., 1996; Pettit et al., 1999; Rankin & Quane, 2001; Ge, 2002; Tolan, 2003; Barber, 2005; Piquart, 2017). Lower family income can lead to increased parental stress (Newland et al., 2013), which has been linked to harsher parenting styles (Schonberg & Shaw, 2007; Adler, 1993), which in turn is associated with increased rates of externalizing disorders (Piquart, 2017; Grant, 2005; Davis et al., 2014). Thus, family income, the variable that best represented family SES, may have explained more variance in conduct disorder because of the downstream effects it has on parenting behavior.

Our hypothesis regarding sex differences was not borne out in the results. Existing research has consistently demonstrated that conduct disorder symptoms are more common in boys than in girls and some evidence suggests the etiology of conduct disorder differs in boys and girls (Burt et al., 2009; Meier et al., 2011), which led us to hypothesize that the relationship between conduct disorder and SES may vary by sex. Nevertheless, we did not find evidence that

sex moderated the relationship between conduct disorder and SES. This suggests that, although there may be sex differences in the etiology of conduct disorder, family and neighborhood SES are not the factors that will explain these differences.

Of note were our findings related to the latent variable measures of family and neighborhood SES. While all of our variables, with the exception of maternal education, loaded significantly onto the latent models of family and neighborhood SES, one variable in each model explained the same amount of variance as the overall latent model. In our family SES model, family income on its own explained the same proportion of the variance in conduct disorder as did the latent model of family SES. This provides some evidence that it may not be necessary to evaluate family SES across a variety of indicators. Researchers could instead focus just on measures of family income, simplifying the amount of data they need to collect. The same can be concluded about the percentage of adults in prestigious occupations. This variable also independently explained the same amount of variance in conduct disorder as did our latent neighborhood SES variable and as such is a candidate to be an independent indicator of neighborhood SES.

We found evidence of moderate additive genetic and nonshared environmental influences on the aggression and rule-breaking dimensions, which is consistent with previous work that has found low to non-existing estimates for shared environmental influences (Slutske et al., 1997; Gelhorn et al., 2005; Dick, et al., 2005; Ferguson et al., 2010). However, we found no evidence of shared environmental influences, which is contrary to some previous studies that have found a small effect of the shared environment (Rhee & Waldman, 2002; Burt, 2009). Moreover, both dimensions of conduct disorder were underpinned by nearly equal contributions of additive genetic influences. This is also somewhat inconsistent with the literature, which has

demonstrated etiological differences between aggression and rule-breaking (see Burt, 2009 for review).

SES seems to play an important role in moderating the etiological influences on conduct disorder and its dimensions (e.g. aggression and rule-breaking). Overall, genetic influences appeared to play a more substantial role in conduct disorder in children who have lower family and neighborhood SES. There is no clear answer in the literature as to why this pattern would emerge. One hypothesis is that it is related to peer affiliation. A 2014 study found that affiliating with prosocial peers reduces the amount of genetic influence on rule-breaking antisocial behaviors (Burt & Klump, 2014). This is in line with our results, where individuals living in a lower SES neighborhood showed higher genetic influences on their rule-breaking behaviors than individuals living in higher SES neighborhoods. Low SES neighborhoods tend to have higher levels of deviant peer behavior than higher SES neighborhoods (Ingoldsby & Shaw, 2002; Rankin & Quane, 2002). Additionally, these lower SES communities and families, are characterized by less involved parents and community members (Ingoldsby & Shaw, 2002) and children are less likely to experience environmental pressures against engaging in these antisocial behaviors (Simons et al., 1996). This may mean that the children in those neighborhoods are experiencing less of the protective effects of prosocial peers described by Burt & Klump, and as a result they have more freedom to act in the way they want, including selecting a more deviant environment in an active gene-environment correlation.

Another possibility is that the differences in heritability estimates are due to parenting styles directly. It has been found that the etiology of conduct problems and antisocial behavior is moderated by parenting style, where more directive and harsher parenting styles were associated with higher estimates of genetic influences on conduct problems (Feinberg et al., 2007; Burt et

al., 2013). Living in a lower SES neighborhood is associated with higher rates of those types of parenting behaviors (Schonberg & Shaw, 2007; Adler, 1993), so we would predict higher estimates of genetic influences on conduct disorder, rule-breaking and aggression based on this hypothesis. However, our results showed that for family SES influences on rule breaking and neighborhood SES influences on aggression, genetic influences were actually higher in the high SES levels. This indicates that either the association between parenting style and family and neighborhood SES is not as strong as previously described or, more likely, that parenting style acts differently on aggression and rule-breaking and these influences are moderated by SES through more than just SES's influence on parenting style.

Finally, it is possible that similar genetic factors influence both harsh parenting behaviors and conduct disorder symptoms. SES can amplify the effects of parenting behaviors on child antisocial behavior (Brody et al., 2003), perhaps because of these similar underlying genetic influences. However, this is also unable to explain our results where higher family SES individuals showed more genetic influences on rule-breaking and higher neighborhood SES individuals showed more genetic influences on aggression.

Furthermore, we found evidence that rule-breaking and aggression are influenced differently by neighborhood and family SES. Rule breaking showed more genetic influences in the high family SES group, but not the high neighborhood SES group, while aggression showed more genetic influences in the high neighborhood SES group but not the high family SES group. This adds to the evidence that there are meaningful differences between the rule-breaking and aggression dimensions of conduct disorder and they should be assessed separately in behavioral genetic analyses. This also provides new evidence that, while both lower family and neighborhood SES are associated with an increase in conduct disorder symptoms, these two

measures of SES interact with rule-breaking and aggressive symptoms differently. This indicates not only that there may be differences in the genetic pathways of these two symptom dimensions, but that the non-shared environmental influences may also differ between the two.

In our moderator analyses, all of the differences in the estimates of genetic and non-shared environmental influences had overlapping confidence intervals, implying that they were non-significant. However, this may have been due in large part to the substantial standard errors found in the high SES groups we analyzed. As a result, we should be careful about over-interpreting the findings of those analyses. More work needs to be done in large samples to replicate these findings to be able to make stronger conclusions about their statistical significance, but this study provides evidence for socioeconomic status's role as a moderator.

Limitations and Future Directions

Limitations to this study include its relatively small sample size in the zygosity-SES groups and the low rates of endorsement of conduct disorder in our sample. Both of these factors contributed to relatively high standard error estimates, particularly in our main effects analyses. Future studies should aim to assess the relationship between conduct disorder and family and neighborhood SES in larger sample sizes and should consider looking at clinical populations or populations that have a higher endorsement of conduct disorder symptoms as this could help correct issues that arose from our high standard error estimates.

Another limitation to this study is its reliance on maternal ratings and the heterogeneity of ages included. Burt's 2009 meta-analysis found that maternal ratings of children's antisocial behavior showed higher estimates for shared environmental influences on aggression and rule-breaking than teacher ratings and lower estimates for nonshared environmental influences on rule-breaking than teacher ratings (Burt, 2009). This provides evidence that the type of rater

being used in a study can influence and bias the parameter estimates that are produced. Additionally, the same metaanalysis showed the relative genetic and environmental contributions to aggression and rule-breaking varied across age groups, with the genetic influences on aggression increasing with age and the genetic influences on rule-breaking decreasing with age. Our sample included a wide range of ages from four to 19 years old, so it is unclear to what extent our results would persist across different developmental stages.

Additionally, while Census data is comprehensive and frequently relied upon (Boylan et al., 2017; Kim et al., 2010; Coulon et al., 2016; Karriker-Jaffee, 2013; Mujahid et al., 2008; O'Campo et al., 2015; Arcaya et al., 2016), there may be better ways to assess neighborhood SES. Studies cited in this work assessed neighborhood SES in an observational manner, with researchers visiting the neighborhoods to assess what has been termed "social disorganization", such as trash in the street, graffiti, broken windows, and other indicators of a community that is lacking in social cohesion. Researchers should continue to carefully consider how they assess SES, particularly at a neighborhood level. However, as described above, the strongest evidence appears to indicate that parenting styles are the most important factor to explain the relationship between SES and conduct disorder. Further studies should consider adding a parenting style measure and assessing its moderating effects on the relationships described here.

Conclusion

The results of this study indicate that family and neighborhood SES are important influences on the development of conduct disorder in children. Overall, children who grow up in higher SES families and in higher SES neighborhoods show less genetic influence on the development of conduct disorder than children who grow up in lower SES families and neighborhoods, but these differences vary across the rule-breaking and aggression symptom

dimensions. As researchers and clinicians, it is important that we continue to consider the impacts that SES has on the development of antisocial behaviors.

Ultimately, there is still much we do not know about the relationship between family and neighborhood SES and conduct disorder. While ours and other's results indicate that there is an important relationship between the two, it is still unknown which specific aspects of SES are acting to moderate the heritability and environmental influences on conduct disorder.

Understanding which factors are the most important, whether it is parenting style, peer deviance, family income, or another factor not identified in this study will be informative in increasing our knowledge of the etiology of conduct disorder as well as opening doors to new treatment and prevention options.

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Appendix 1 – Tables

Table 1. *Descriptive Statistics of Family Socioeconomic Status*

	Maternal	Paternal
<i>Ethnicity</i>		
White	85%	84%
African American	12%	12%
Hispanic	2%	2%
Asian American	1%	1%
American Indian	0.6%	0.4%
Mixed race or other	0.6%	0.5%
<i>Highest Education Level</i>		
8 th Grade or less	0.3%	1%
9 th Grade	0.2%	0.4%
10 th Grade	0.4%	1%
11 th Grade	1%	2%
12 th Grade	3%	3%
High School graduate	18%	19%
Some college	29%	28%
College graduate	33%	29%
Master's Degree	2%	11%
Doctoral or Professional Degree	3%	11%
<i>Family Income</i>		<i>Overall Family Income</i>
\$1-10,000		2%
\$10-20,000		5%
\$20-30,000		8%
\$30-40,000		14%
\$40-50,000		16%
\$50-60,000		13%
\$60-70,000		10%
\$70-80,000		9%
\$80-90,000		6%
\$90-100,000		4%
\$100-150,000		7%
More than \$150,000		6%

Table 2. *Descriptive Statistics of Neighborhood Socioeconomic Status*

	Mean (SE)	Minimum	Maximum
Percentage of adults who complete high school or its equivalent	55% (0.15)	9%	87%
Percentage of adults who obtained a college degree or higher	34% (0.30)	0%	90%
Percentage of families living below the federal poverty line	11% (0.09)	0%	50%
Percentage of families receiving federal assistance	7% (0.06)	0%	38%
Percentage of unemployed adults over 25	5% (0.05)	0%	23%
Percentage of rented housing units	31% (0.27)	0%	100%
Percentage of family households with unmarried parents	26% (0.23)	3%	88%
Percentage of adults over 25 in prestigious occupations	38% (0.15)	4%	84%
Percentage of residents who identify as African American	22% (0.23)	0%	97%
Percentage of residents who identify as Hispanic	14% (0.16)	0%	86%

Table 3. *Descriptive statistics and twin correlations of conduct disorder.*

	Mean Conduct Disorder Score	Correlation with Twin 1	Correlation with Twin 2
<i>Twin 1</i>			
MZ	2.80	1.0	0.69
DZ	3.11	1.0	0.26
<i>Twin 2</i>			
MZ	2.88	0.69	1.0
DZ	3.13	0.26	1.0
	Mean Aggression Score	Correlation with Twin 1	Correlation with Twin 2
<i>Twin 1</i>			
MZ	0.98	1.0	0.67
DZ	0.93	1.0	0.22
<i>Twin 2</i>			
MZ	1.01	0.67	1.0
DZ	0.97	0.22	1.0
	Mean Rule-Breaking Score	Correlation with Twin 1	Correlation with Twin 2
<i>Twin 1</i>			
MZ	1.83	1.0	0.60
DZ	2.19	1.0	0.27
<i>Twin 2</i>			
MZ	1.90	0.60	1.0
DZ	2.17	0.27	1.0

Table 4. Factor loadings of family SES and neighborhood SES on respective latent variables.

	Estimate (SE)	P-value	R-square (SE)	P-value
<i>Family SES</i>				
Family Income	-0.18 (.02)	<.01	0.03 (.01)	<.01
Mother's Education	0.05 (.03)	0.06	0.00 (.00)	0.33
Father's Education	-0.22 (.03)	<.01	0.05 (.01)	<.01
Mother's Ethnicity	0.98 (.01)	<.01	0.96 (.01)	<.01
Father's Ethnicity	1.0 (.01)	<.01	0.99 (.01)	<.01
<i>Neighborhood SES</i>				
Percent High School	-0.91 (.01)	<.01	0.82 (.01)	<.01
Percent College	0.99 (.00)	<.01	0.98 (.06)	<.01
Percent Prestigious	0.93 (.00)	<.01	0.86 (.01)	<.01
Percent Unemployed	-0.47 (.01)	<.01	0.22 (.01)	<.01
Percent Below Poverty	-0.63 (.01)	<.01	0.40 (.02)	<.01
Percent Receiving Aid	-0.60 (.01)	<.01	0.40 (.01)	<.01
Percent Single Parents	-0.53 (.02)	<.01	0.30 (.01)	<.01
Percent Rented Homes	-0.29 (.02)	<.01	0.09 (.01)	<.01
Percent African American	-0.36 (.02)	<.01	0.13 (.01)	<.01
Percent Hispanic	-0.32 (.02)	<.01	0.10 (.01)	<.01

Table 5. Associations of family SES with conduct disorder dimensions.

	Linear Model			Curvilinear Model		
	Beta (SE)	t	P-value	Beta (SE)	t	P-value
<i>Conduct Disorder</i>						
Family Income	-0.09 (.04)	-2.62	0.009	-0.30(.14)	-2.20	0.03
Mother's Education	0.02 (.04)	0.51	0.609	0.03 (.04)	0.63	0.53
Father's Education	-0.06 (.04)	-1.50	0.146	-0.06 (.04)	-1.50	0.14
Mother's Ethnicity	-0.07 (.07)	-1.10	0.272	-0.05 (.04)	-0.96	0.59
Father's Ethnicity	0.06 (.07)	0.87	0.383	0.03 (.08)	0.43	0.67
Family SES	0.02 (.11)	0.16	0.88	-0.35 (.15)	-2.39	0.02
Family SES Squared	—	—	—	0.21 (.13)	1.68	0.10
<i>Rule-breaking</i>						
Family SES	0.01 (.02)	-0.24	0.74	0.04 (.05)	0.81	0.42
Family SES Squared	—	—	—	-0.04 (.02)	-1.97	0.05
<i>Aggression</i>						
Family SES	-0.01 (.03)	0.34	0.81	0.01 (.07)	0.12	0.91
Family SES Squared	—	—	—	-0.06 (.03)	-2.0	0.05

Table 6. *Associations of neighborhood SES with conduct disorder dimensions.*

	Linear Model			Curvilinear Model		
	Beta (SE)	t	P-value	Beta (SE)	t	P-value
<i>Conduct Disorder</i>						
Percent HS	0.02 (.13)	0.18	0.86			
Percent BA	0.09 (.16)	0.56	0.58			
Percent Prestigious	-0.14 (.07)	-2.14	0.03			
Percent Unemployed	0.04 (.03)	1.30	0.19			
Percent Poverty	0.03 (.05)	0.65	0.52			
Percent Federal Aid	-0.08 (.05)	-1.65	0.10			
Percent Unmarried	0.04 (.05)	0.81	0.42			
Percent Rental	-0.03 (.03)	-0.80	0.43			
Percent AA	-0.03 (.06)	-0.56	0.58			
Percent Hispanic	0.04 (.07)	0.52	0.60			
Neighborhood SES	-.07 (.02)	-3.06	0.002	0.06 (.03)	1.65	0.10
Neighborhood SES Squared	—	—	—	-0.02 (.02)	-0.77	0.44
<i>Rule-breaking</i>						
Neighborhood SES	0.06 (.02)	2.70	<.001	0.05 (.03)	1.45	0.15
Neighborhood SES Squared	—	—	—	-0.02 (.02)	-0.73	0.46
<i>Aggression</i>						
Neighborhood SES	0.08 (.02)	3.37	0.001	0.08 (.03)	2.47	0.01
Neighborhood SES Squared	—	—	—	0.01 (.02)	0.29	0.77

Table 7. *Univariate BG Models of Conduct Disorder, Rule-breaking, and Aggression*

Model	Model Fit Statistics							Parameter Estimates			
	χ^2 (df)	CFI	TLI	AIC	BIC	RMSEA	SRMR	a^2	c^2/d^2	e^2	s
<i>Conduct Disorder</i>											
ACE	22.1 (13)	0.10	0.10	7989	8049	0.10	0.07	0.71***	0.00*	0.27***	---
ACE + s	5.8 (12)	1.00	1.03	7964	8028	0.02	0.03	0.41***	0.79***	0.09**	0.14**
ADE	17.2 (13)	0.98	0.98	7978	8038	0.06	0.07	0.20	0.27***	0.18***	---
ADE + s	10.1 (14)	1.00	1.01	7971	8035	0.05	0.05	0.6***	0.00	0.18***	0.02***
AE	23.8* (14)	0.96	0.95	7887	8042	0.07	0.07	0.71***	---	0.27***	---
AE + s	11.0 (13)	1.00	1.01	7969	8028	0.05	0.05	0.86***	---	0.18***	0.02***
<i>Aggression</i>											
ACE	12.7 (13)	1.00	1.00	5657	5711	0.05	0.04	0.65***	0.00	0.33***	---
ACE + s	8.1 (12)	1.00	1.03	5640	5705	0.04	0.02	0.69***	0.17	0.21**	0.02
ADE	9.3 (13)	1.00	1.03	5642	5701	0.04	0.04	0.14	0.53**	0.31***	---
ADE + s	7.4 (12)	1.00	1.04	5640	5705	0.03	0.03	0.79***	0.00	0.24***	0.01
AE	13.7 (14)	1.00	1.00	5649	5704	0.05	0.04	0.65***	---	0.33***	---
AE + s	8.0 (13)	1.00	1.04	5639	5698	0.03	0.03	0.79***	---	0.13***	0.02*
<i>Rule-breaking</i>											
ACE	12.7 (13)	1.00	1.00	7082	7141	0.05	0.06	0.65***	0.00	0.36***	---
ACE + s	2.38 (12)	1.00	1.08	7066	7131	0.00	0.03	0.26**	1.05***	0.09*	0.21*
ADE	11.7 (13)	1.00	1.01	7078	7137	0.05	0.06	0.29*	0.35*	0.34***	---
ADE + s	7.5 (12)	1.00	1.01	7074	7138	0.03	0.05	0.77***	0.00	0.26***	0.01
AE	13.7 (14)	1.00	1.00	7080	7135	0.05	0.06	0.62***	---	0.36***	---
AE + s	8.1 (13)	1.00	1.04	7072	7132	0.03	0.05	0.77***	---	0.26***	0.01

Table 8. *BG Models of Conduct Disorder, Rule Breaking, and Aggression Moderated by Family SES*

Model	Model Fit Statistics							Parameter Estimates							
	χ^2 (df)	CFI	TLI	AIC	BIC	RMSEA	SRMR	Low SES			High SES				
								a^2	c^2/d^2	e^2	s	a^2	c^2/d^2	e^2	s
<i>Conduct Disorder</i>															
Constrained AE + s	62.9*** (31)	0.87	0.85	6904	6997	0.08	0.14	0.85***	---	0.16***	0.02***	0.88***	---	0.17***	0.02***
Unconstrained AE + s	33.0 (28)	0.98	0.97	6863	6969	0.03	0.09	0.89***	---	0.13***	0.02*	0.78***	---	0.26***	0.01
Unconstrained ADE	35.9 (28)	0.97	0.96	6869	6975	0.04	0.10	0.21	0.55***	0.19***	---	0.00	0.80***	0.59***	---
Unconstrained ACE	45.6* (28)	0.93	0.91	6884	6990	0.06	0.11	0.75***	0.00	0.21***	---	0.61***	0.00	0.62***	---
<i>Rule-Breaking</i>															
Constrained AE + s	65.0*** (31)	0.79	0.76	5927	6020	0.08	0.12	0.71***	---	0.27***	0.01	0.74***	---	0.30***	0.01
Unconstrained AE + s	32.1 (28)	0.97	0.97	5884	5990	0.03	0.07	0.72***	---	0.29***	0.01	0.75***	---	0.29***	0.02
Unconstrained ADE	29.1 (28)	0.99	0.99	5884	5991	0.02	0.08	0.40**	0.20	0.36***	---	0.00	0.59***	0.40***	---
Unconstrained ACE	33.9 (28)	0.96	0.95	5893	6000	0.04	0.08	0.90***	0.00	0.22***	---	0.36***	0.00	0.41***	---
<i>Aggression</i>															
Constrained AE + s	54.5*** (32)	0.91	0.90	4957	5046	0.07	0.10	0.77***	---	0.25***	0.01	0.78***	---	0.24***	0.01
Unconstrained AE + s	29.3 (28)	1.00	0.99	4926	5032	0.02	0.07	0.91***	---	0.13***	0.02*	0.43***	---	0.54***	0.00
Unconstrained ADE	37.9 (30)	0.97	0.96	4936	5034	0.04	0.08	0.40**	0.20	0.36***	---	0.00	0.53***	0.40**	---
Unconstrained ACE	35.4 (28)	0.97	0.96	4940	5046	0.04	0.08	0.78**	0.00	0.19***	---	0.45***	0.00	0.52***	---

Table 9. *BG Models of Conduct Disorder, Rule Breaking, and Aggression Moderated by Neighborhood SES*

Model	Model Fit Statistics							Parameter Estimates							
	χ^2 (df)	CFI	TLI	AIC	BIC	RMSEA	SRMR	Low SES			High SES				
								a ²	c ² /d ²	e ²	s	a ²	c ² /d ²	e ²	s
<i>Conduct Disorder</i>															
Constrained AE + s	49.5* (31)	0.93	0.92	7542	7637	0.06	0.12	0.86***	---	0.17***	0.02*	0.89***	---	0.17***	0.02*
Unconstrained AE + s	21.3 (28)	1.00	1.03	7501	7609	0.00	0.07	0.90***	---	0.14***	0.02*	0.77***	---	0.27***	0.00
Unconstrained ADE	27.2 (28)	1.00	1.00	7509	7617	0.00	0.08	0.13	0.65***	0.20***	---	0.45**	0.21	0.33***	---
Unconstrained ACE	31.8 (28)	0.99	0.98	7518	7627	0.03	0.08	0.76***	0.00	0.21***	---	0.65***	0.00	0.35***	---
<i>Rule Breaking</i>															
Constrained AE	31.2 (32)	1.00	1.01	6498	6589	0.00	0.08	0.72***	---	0.29***	0.01	0.75***	---	0.29***	0.02
Unconstrained AE + s	15.2 (28)	1.00	1.09	6480	6589	0.00	0.05	0.80***	---	0.23***	0.01	0.52***	---	0.47	0.00
Unconstrained ADE	16.9 (28)	1.00	1.08	6484	6593	0.00	0.06	0.29	0.39*	0.30***	---	0.55***	0.00	0.45***	---
Unconstrained ACE	19.0 (28)	1.00	1.06	6486	6595	0.00	0.06	0.66***	0.00	0.66***	---	0.48**	0.06	0.46***	---
<i>Aggression</i>															
Constrained AE + s	128.0*** (31)	0.59	0.52	5356	5451	0.14	0.23	0.79***	---	0.23***	0.01	0.80***	---	0.23***	0.01
Unconstrained AE + s	34.0 (28)	0.98	0.97	5212	5321	0.04	0.07	0.75***	---	0.27***	0.00	0.86***	---	0.16***	0.03**
Unconstrained ADE	35.4 (28)	0.97	0.96	5217	5326	0.04	0.07	0.33	0.34	0.31***	---	0.00	0.69***	0.27***	---
Unconstrained ACE	42.9 (28)	0.94	0.92	5233	5342	0.06	0.08	0.65***	0.00	0.33***	---	0.65***	0.00	0.31***	---

Appendix 2 – Figures

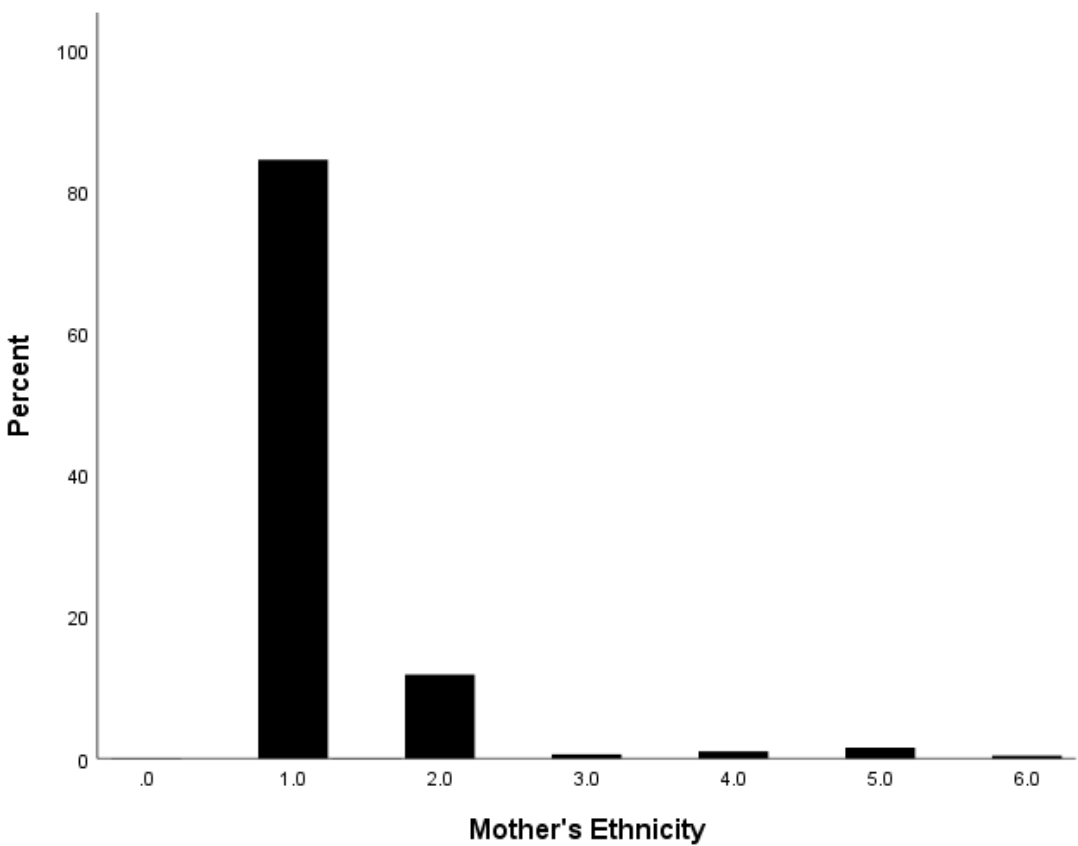


Figure 1. Histogram of self-reports of maternal ethnicity. (Note. 1.0 = White, 2.0 = African American, 3.0 = Asian American, 4.0 = Mixed race, 5.0 = Hispanic, 6.0 = Other).

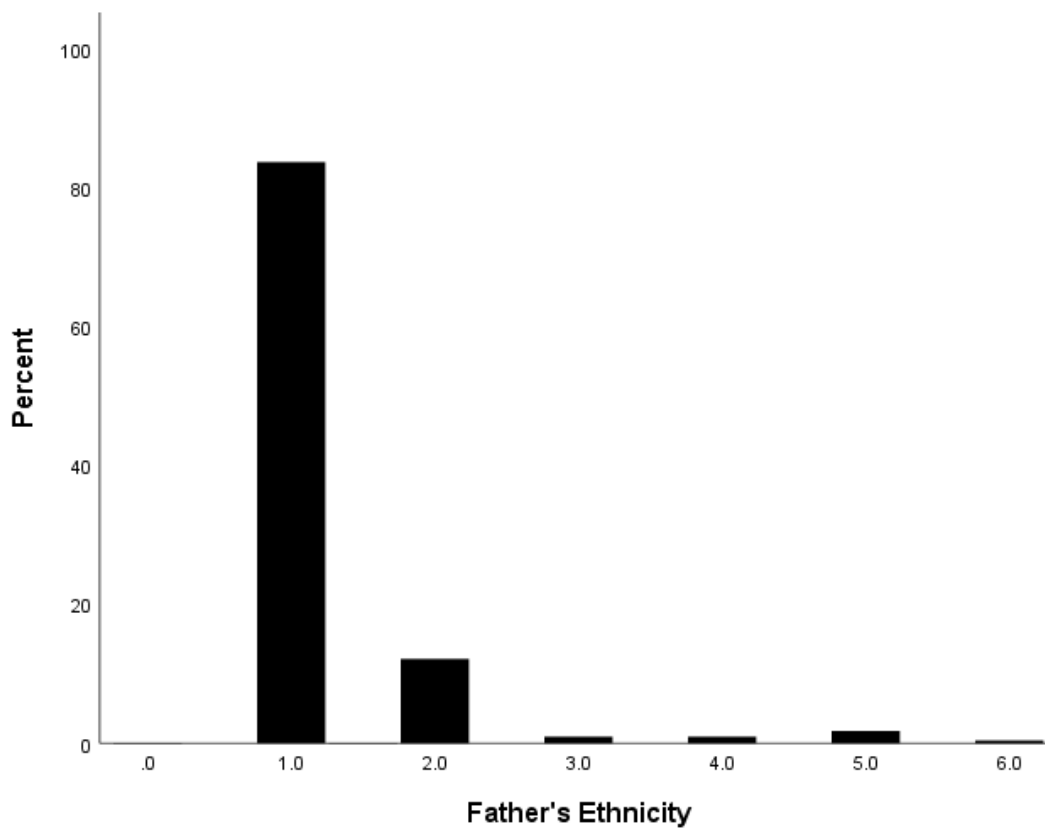


Figure 2. Histogram of self-reports of paternal ethnicity. (Note. 1.0 = White, 2.0 = African American, 3.0 = Asian American, 4.0 = Mixed race, 5.0 = Hispanic, 6.0 = Other).

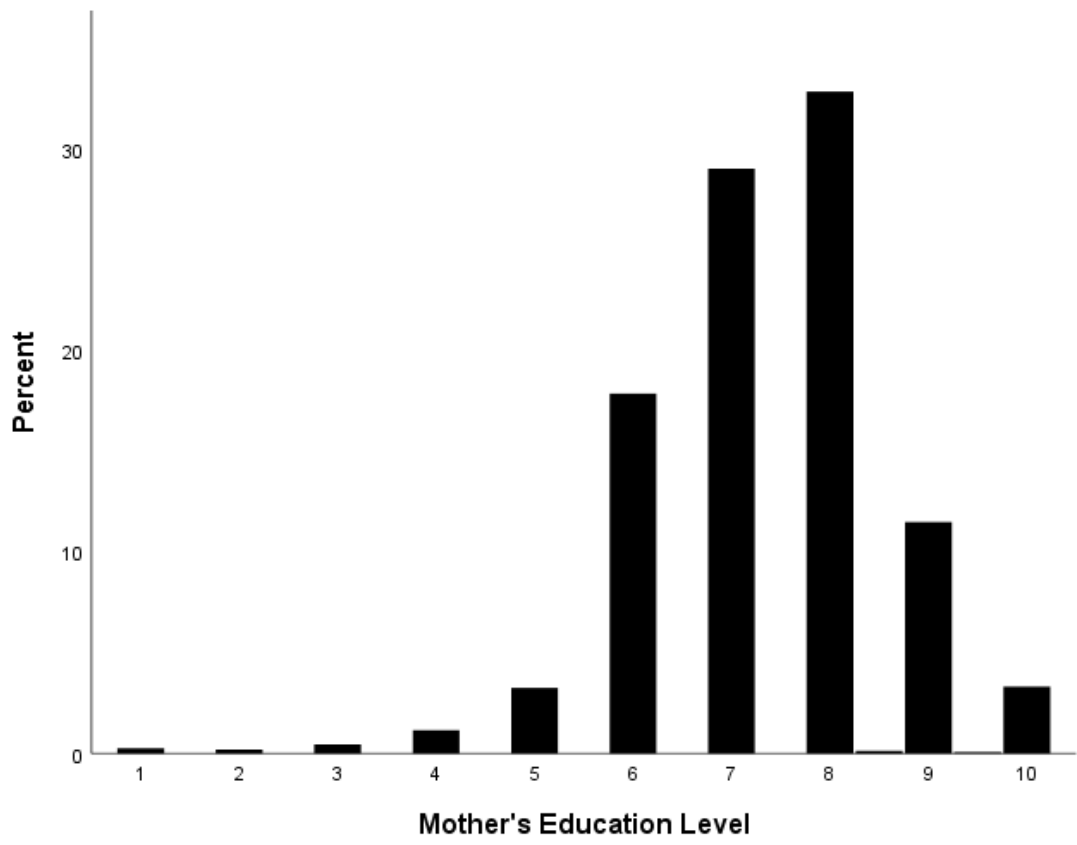


Figure 3. Histogram of highest level of maternal education. (Note. 1 = less than 8th grade, 2 = 9th grade, 3 = 10th grade, 4 = 11th grade, 5 = 12th grade, 6 = high school graduate, 7 = some college, 8 = college graduate, 9 =master's degree, 10 = professional or doctoral degree)

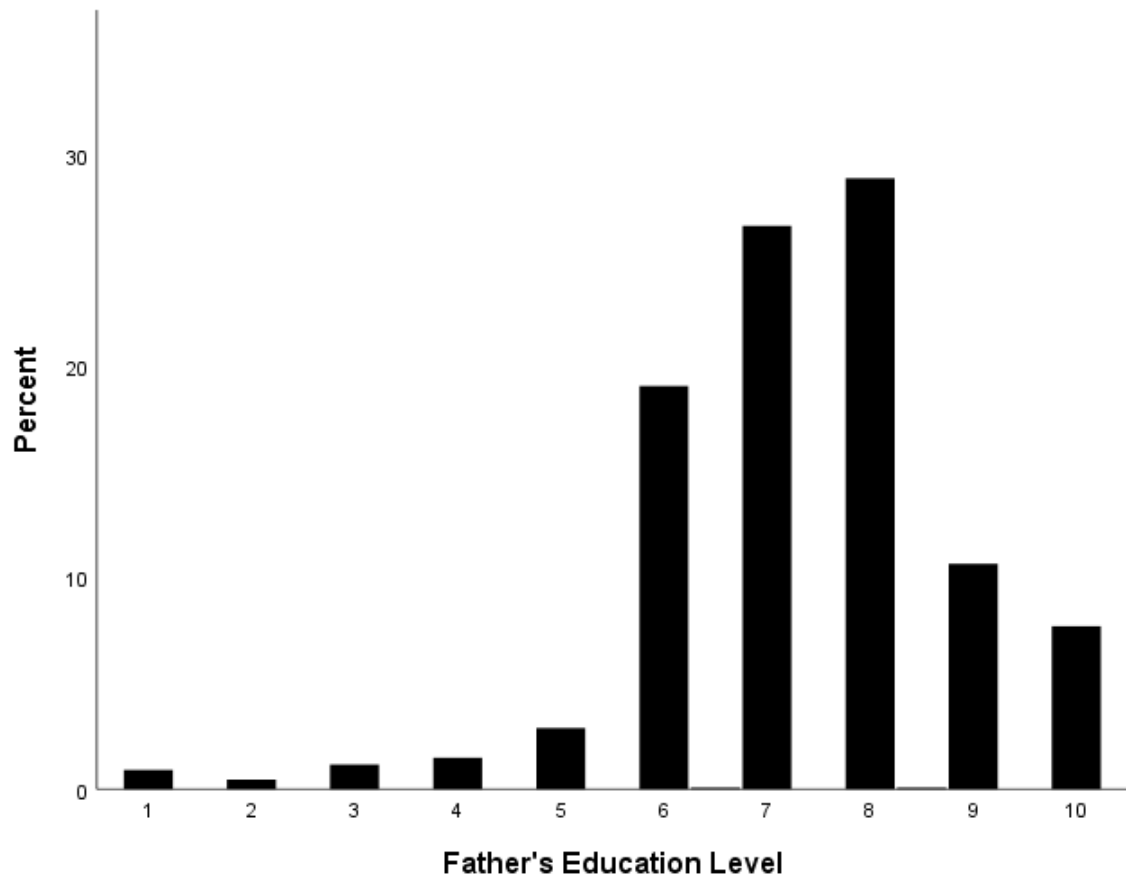


Figure 4. Histogram of highest level of maternal education. (Note. 1 = less than 8th grade, 2 = 9th grade, 3 = 10th grade, 4 = 11th grade, 5 = 12th grade, 6 = high school graduate, 7 = some college, 8 = college graduate, 9 =master's degree, 10 = professional or doctoral degree)

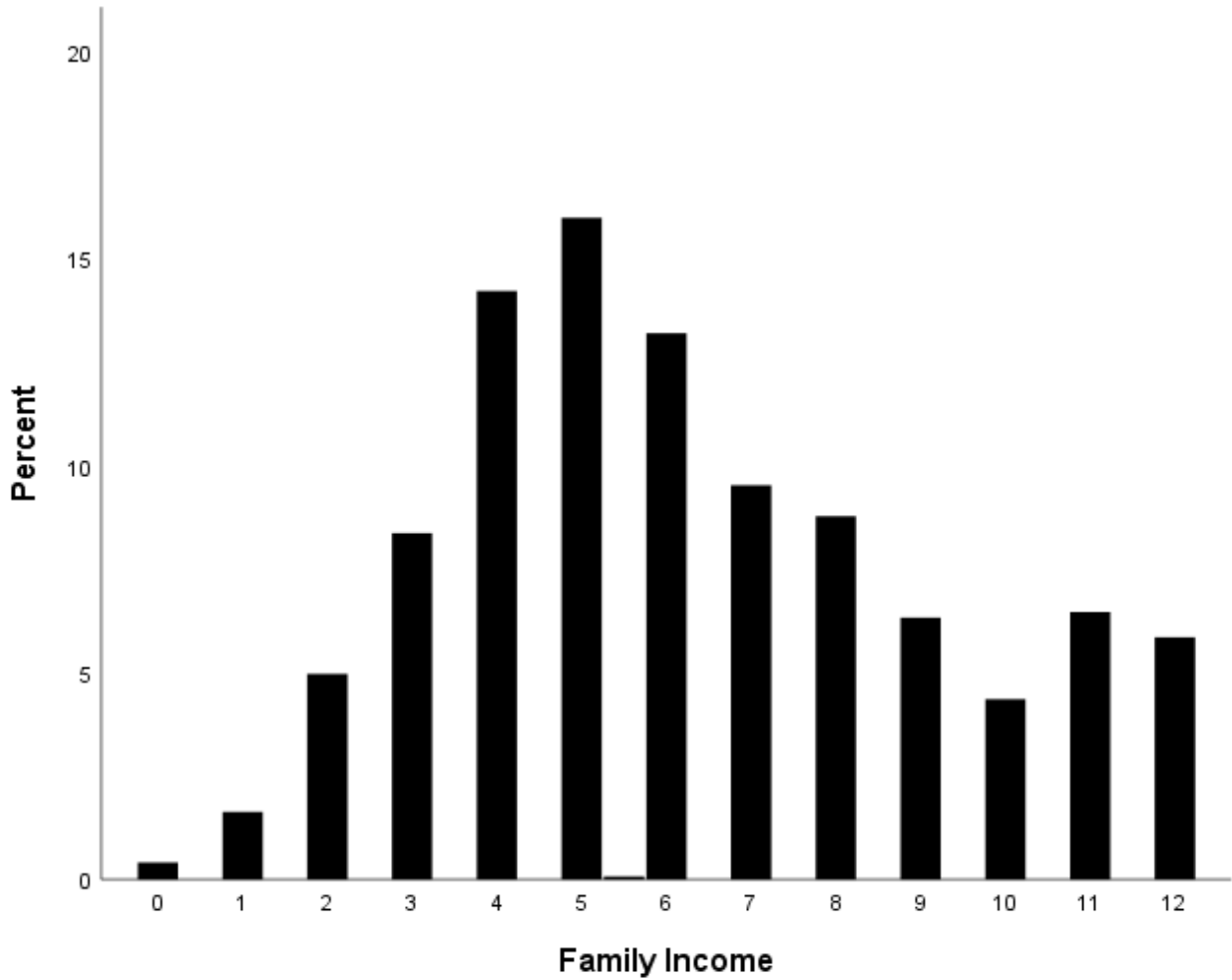


Figure 5. Histogram of family income ranges. (Note. 0 = income not reported, 1 = \$1-10,000, 2 = \$10-20,000, 3 = \$20-30,000, 4 = \$30-40,000, 5 = \$40-50,000, 6 = \$50-60,000, 7 = \$60-70,000, 8 = \$70-80,000, 9 = \$80-90,000, 10 = \$90-100,000, 11 = \$100-150,000, 12 = more than \$150,000).

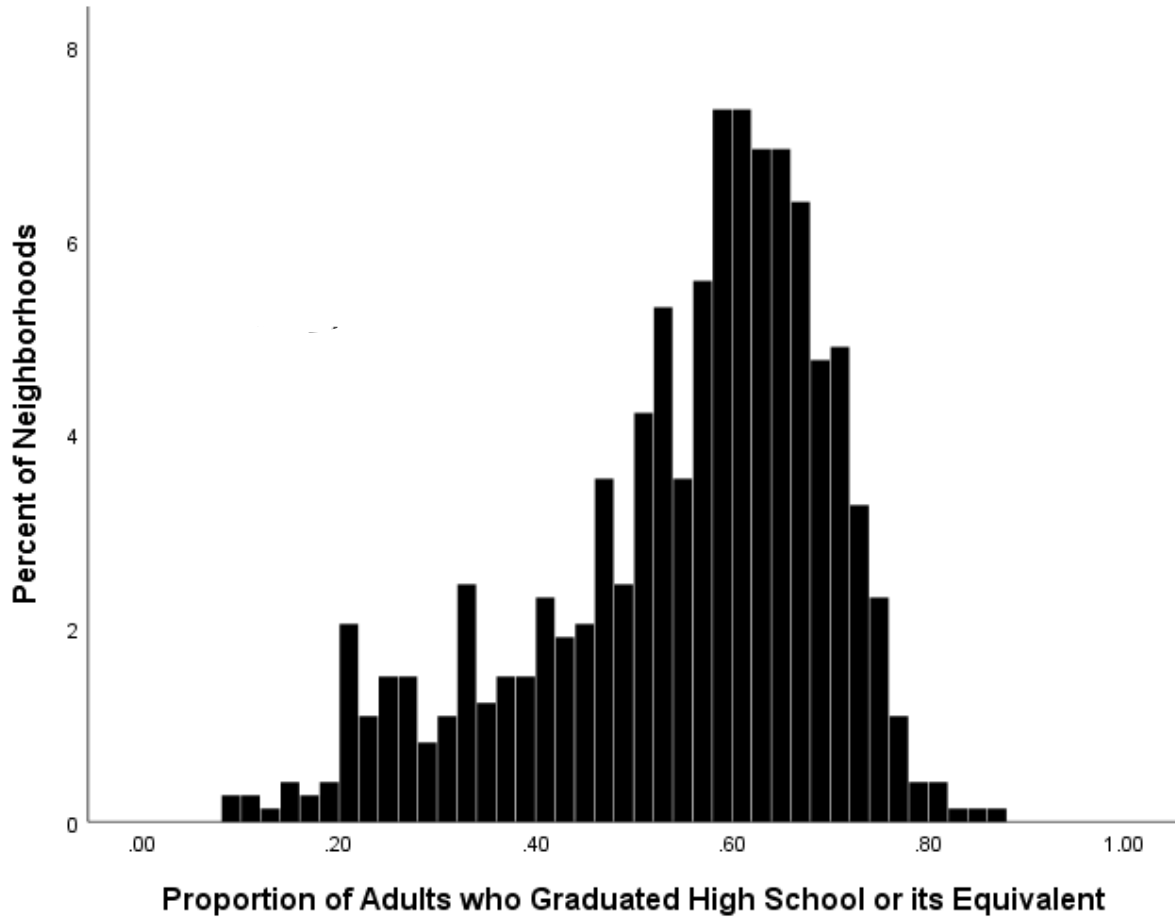


Figure 6. Histogram of the percentage of neighborhoods with each proportion of adults who graduated high school or its equivalent

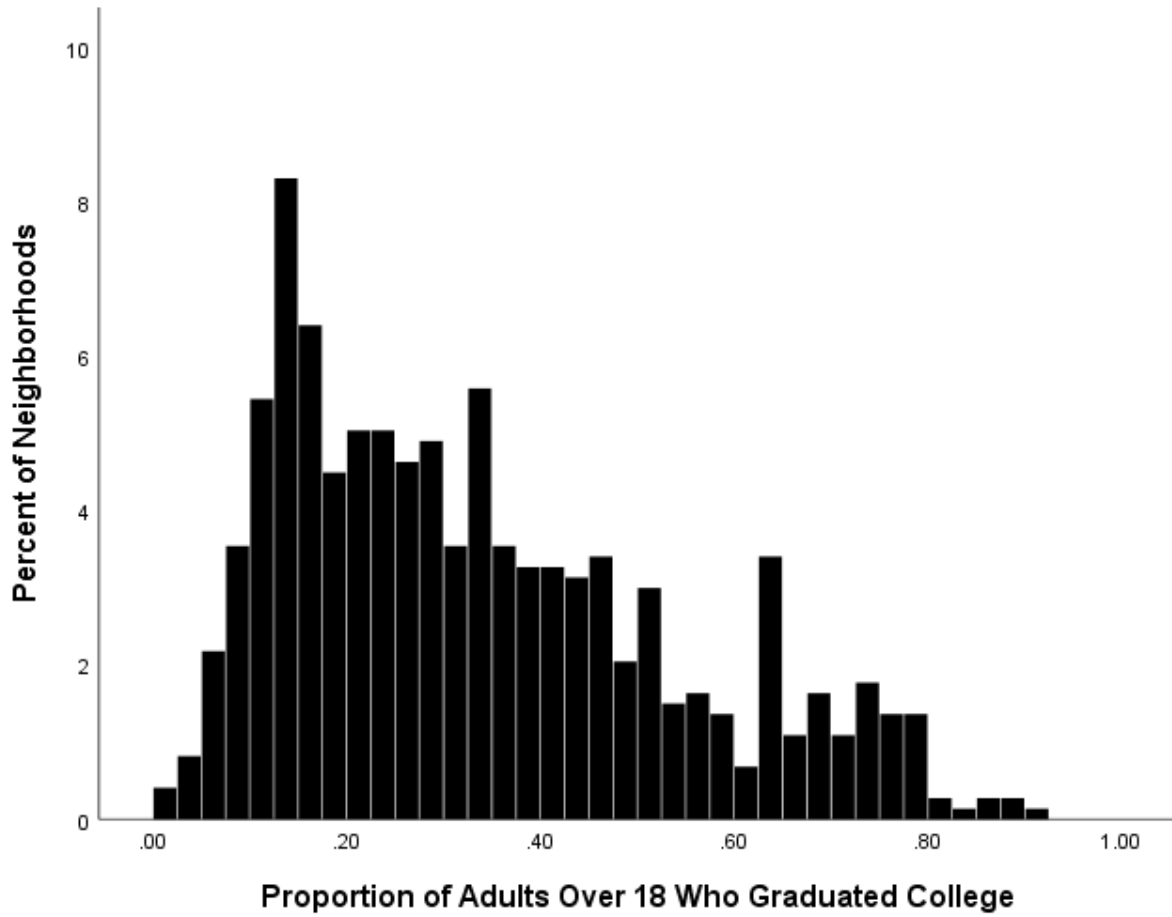


Figure 7. Histogram of the percentage of neighborhoods with each proportion of adults who graduated from college

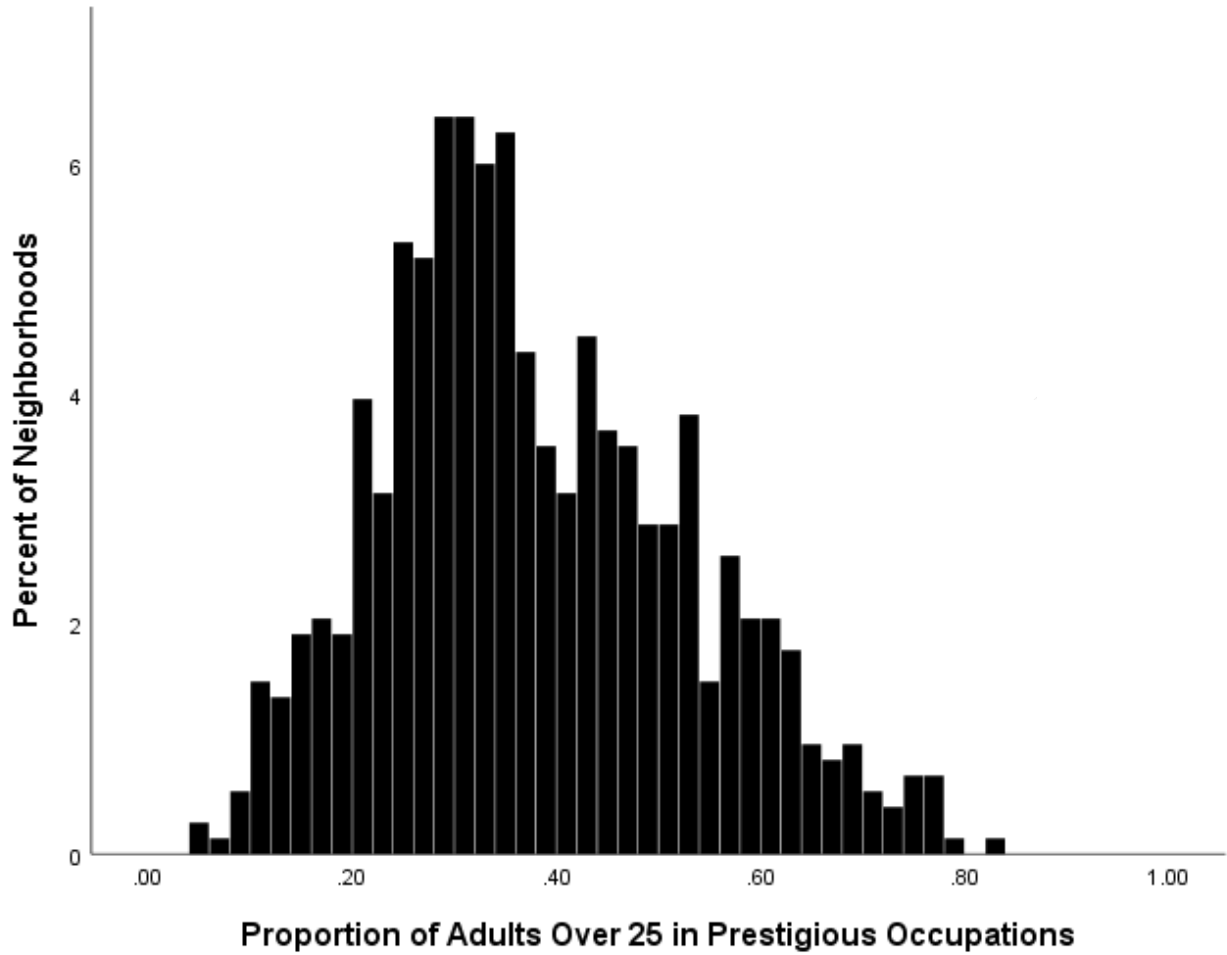


Figure 8. Histogram of the percentage of neighborhoods with each proportion of adults over 25 who are in a prestigious occupation

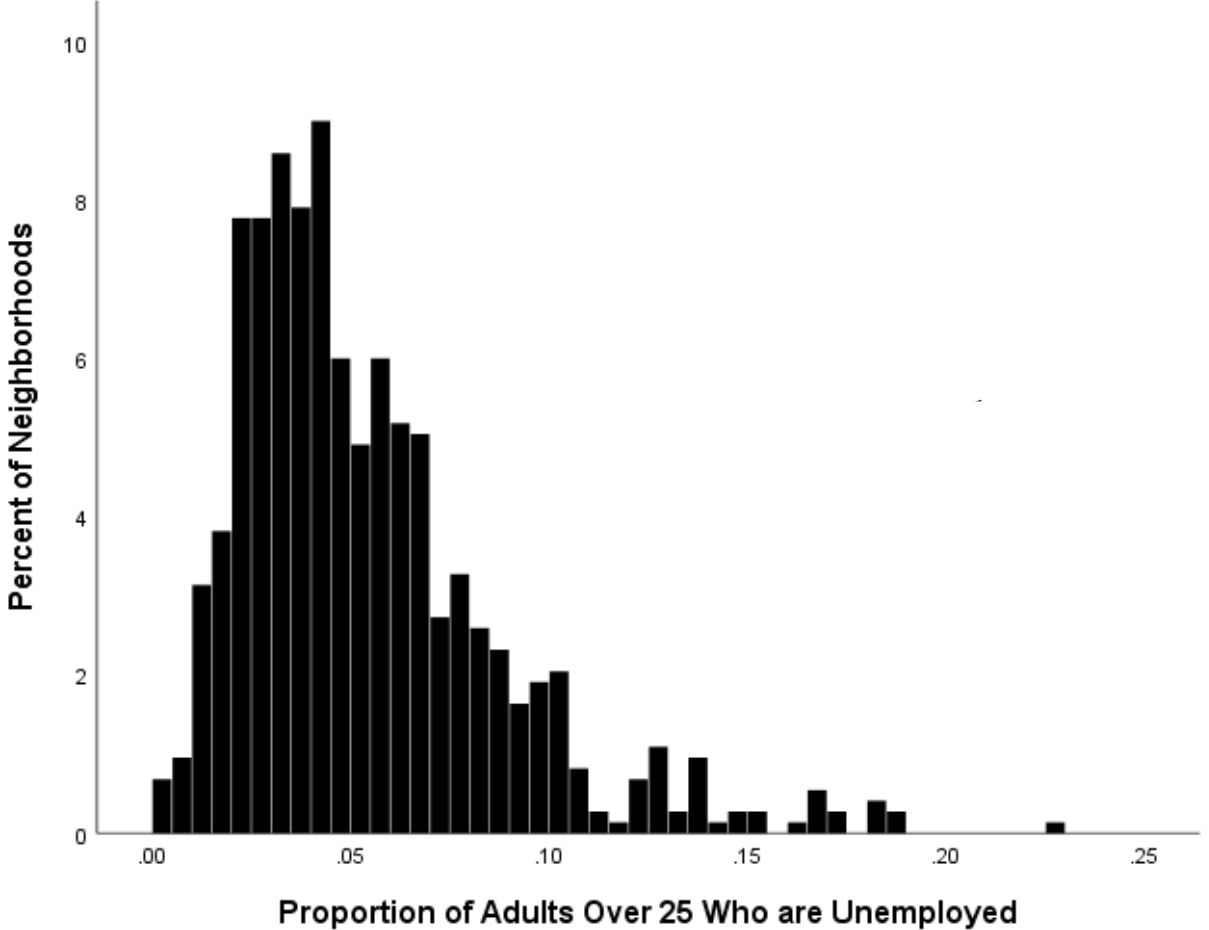


Figure 9. Histogram of the percentage of neighborhoods with each proportion of adults over 25 who are unemployed

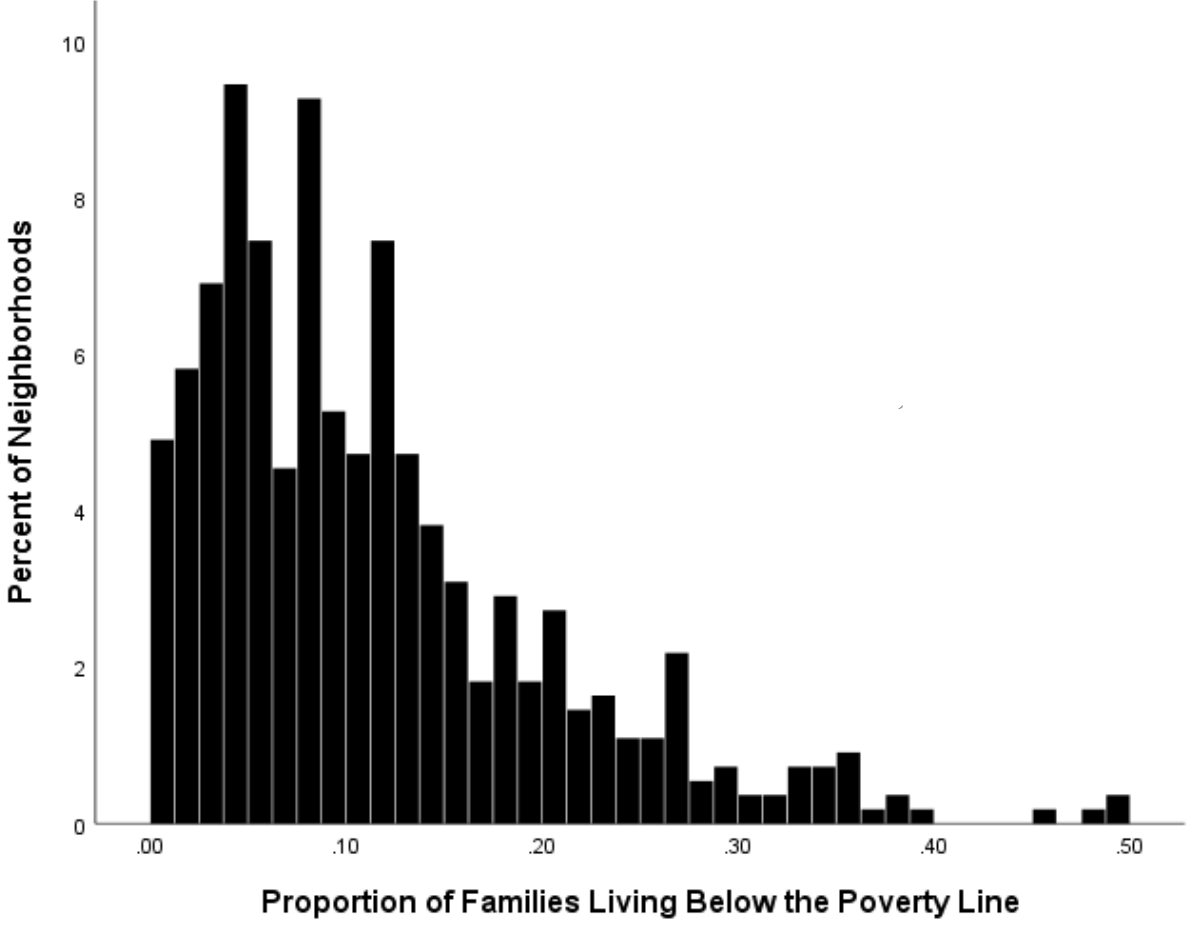


Figure 10. Histogram of the percentage of neighborhoods with each proportion of families living below the poverty line

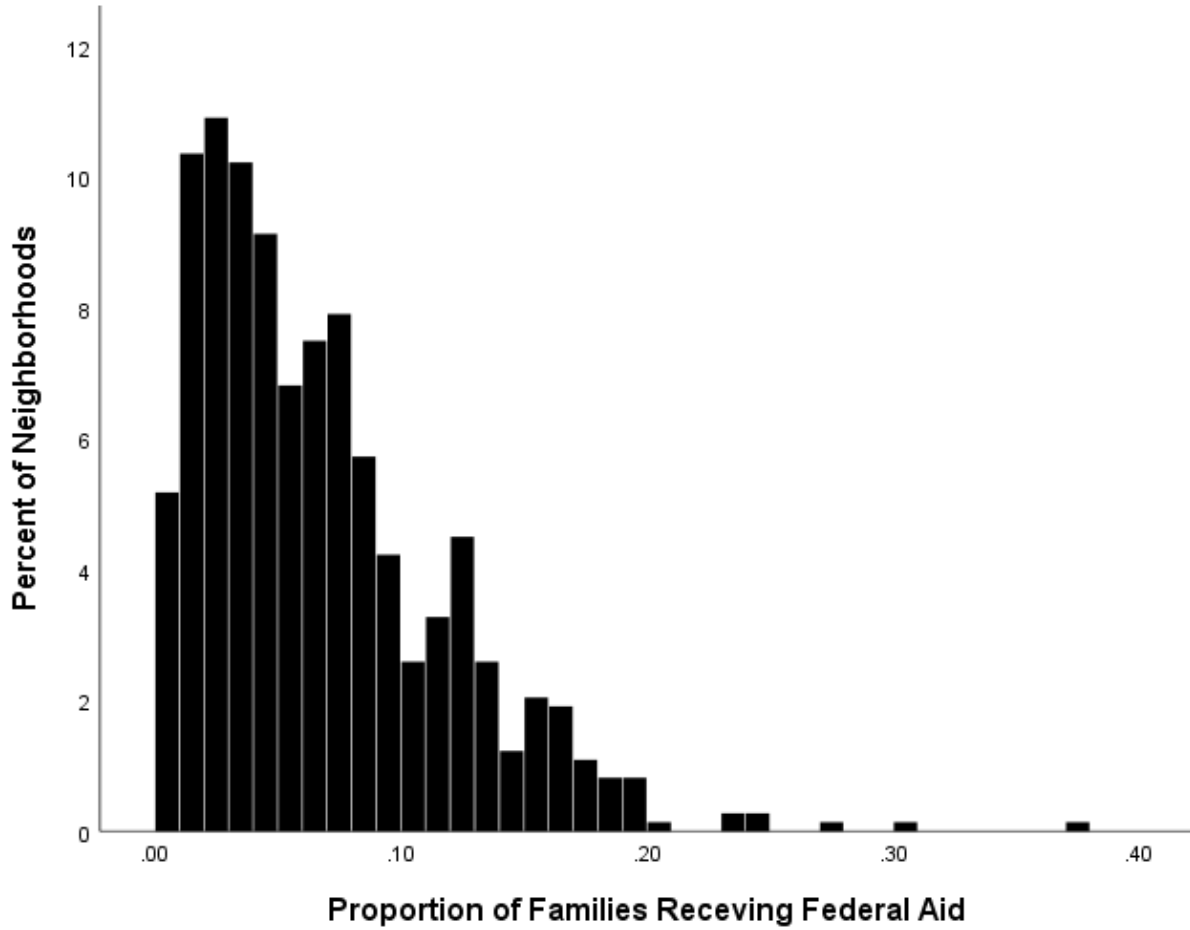


Figure 11. Histogram of the percentage of neighborhoods with each proportion of families receiving federal aid

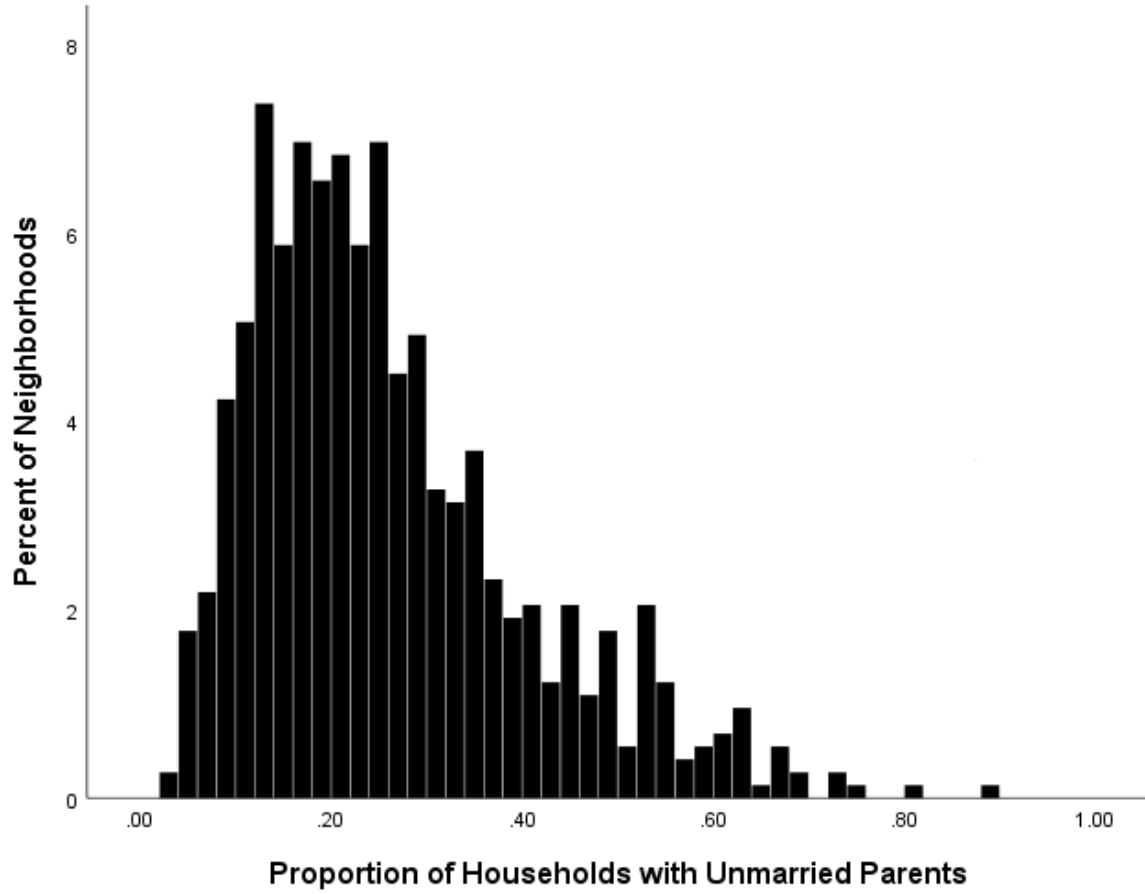


Figure 12. Histogram of the percentage of neighborhoods with each proportion of households with unmarried parents

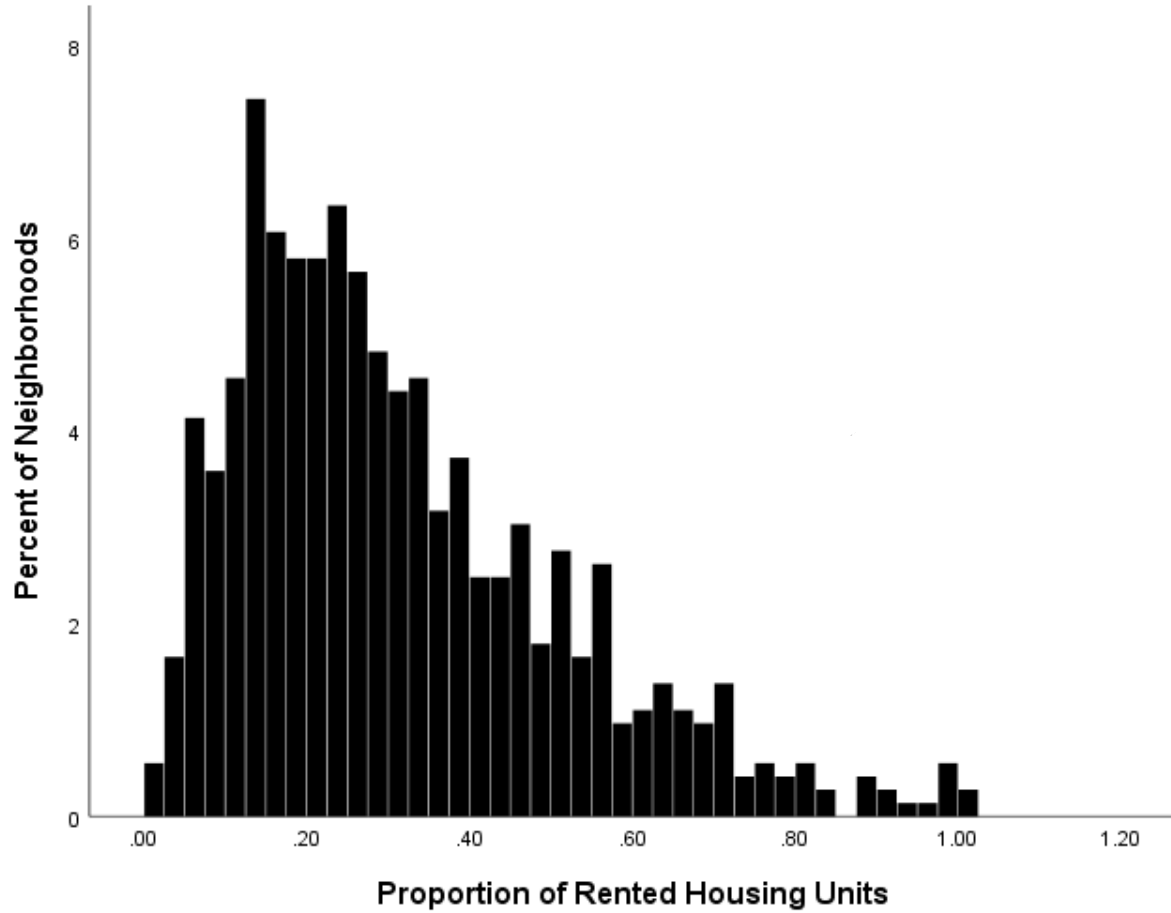


Figure 13. Histogram of the percentage of neighborhoods with each proportion of rented housing units

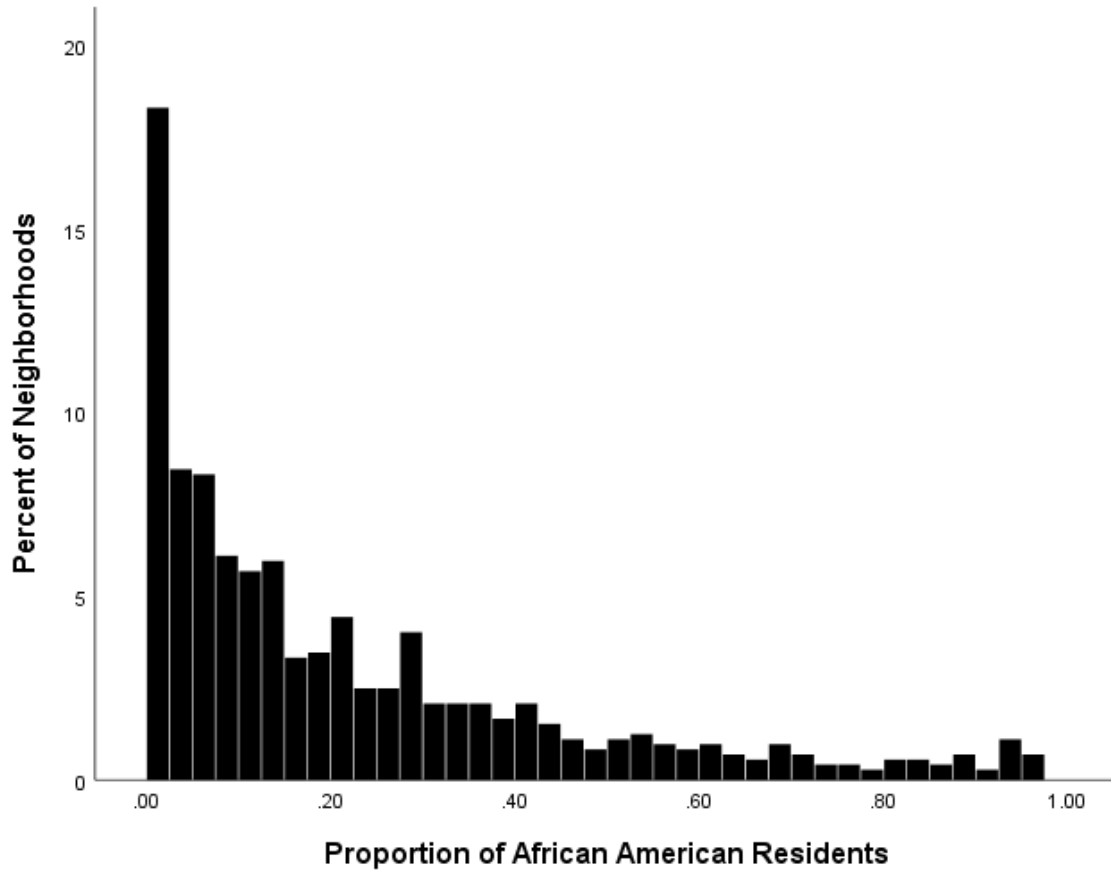


Figure 14. Histogram of the percentage of neighborhoods with each proportion of African American residents

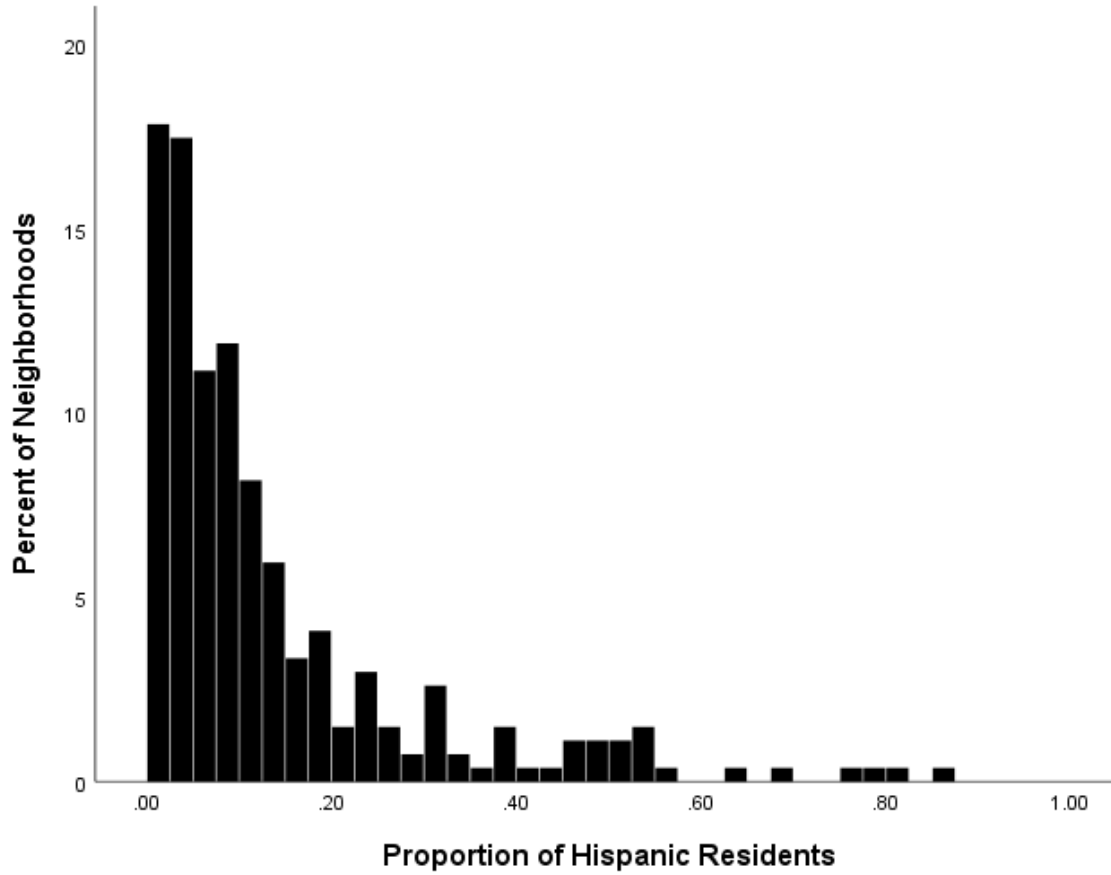


Figure 15. Histogram of the percentage of neighborhoods with each proportion of Hispanic residents