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SOCIAL ADVERSITY, GENETIC VARIATION, STREET CODE, AND AGGRESSION: A GENETICLLY INFORMED MODEL OF VIOLENT BEHAVIOR

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Abstract

Elijah Anderson (1997, 1999) argues that exposure to extreme community disadvantage, residing in “street” families, and persistent discrimination encourage many African Americans to develop an oppositional culture that he labels the “code of the street.” Importantly, while the adverse conditions described by Anderson increase the probability of adopting the code of the street, most of those exposed to these adverse conditions do not do so. The present study examines the extent to which genetic variation accounts for these differences. Although the diathesis-stress model guides most genetically informed behavior science, the present study investigates hypotheses derived from the differential susceptibility perspective (Belsky & Pluess, 2009). This model posits that some people are genetically predisposed to be more susceptible to environmental influence than others. An important implication of the model is that those persons most vulnerable to adverse social environments are the same ones who reap the most benefit from environmental support. Using longitudinal data from a sample of several hundred African American males, we examined the manner in which variants in three genes - 5-HTT, DRD4, and MAOA - modulate the effect of community and family adversity on adoption of the street code and aggression. We found strong support for the differential susceptibility perspective. When the social environment was adverse, individuals with these genetic variants manifested *more* commitment to the street code and aggression than those with other genotypes, whereas when adversity was low they demonstrated *less* commitment to the street code and aggression than those with other genotypes.

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Both official statistics and self-report studies have documented the high rates of criminal violence that exist in many disadvantaged African American communities (Peterson et al., 2006; Sampson, Morenoff, & Radenbush, 2005; LaFree, Baumer, & O'Brien, 2010). In recent years, much attention has been given to Elijah Anderson's (1999) street culture explanation for this phenomenon. Based upon his ethnographic research in Philadephia, Anderson (1997, 1999) argues that exposure to extreme community disadvantage, residing in "street" families, and persistent discrimination encourage many African Americans to adopt an oppositional culture that he labels the "code of the street." The code entails a cynicism regarding societal rules and a distrust of others combined with the belief that one must be prepared to use any means necessary to protect oneself, avoid exploitation, and maintain respect. Anderson contends that commitment to this code dramatically increases the probability of involvement in acts of aggression and violence. Several recent studies have provided support for Anderson's thesis (Brezina et al., 2004; Ousey, 2005; Stewart & Simons, 2006; 2010).

Importantly, however, while the adverse conditions described by Anderson increase the probability of adopting the code of the street, most of those exposed to these conditions do not do so. This raises the question of what distinguishes those who adopt the code from those who do not. The present study examines the extent to which genetic factors might account for some of this difference in response to a hostile/demoralizing environment. Recently, studies have reported that certain variants (alleles) of the serotonin transporter gene (5-HTTLPR), the dopamine receptor gene (DRD4), and the monoamine oxidase gene (MAOA) interact with environmental adversity to increase the probability of aggression and crime (Guo et al. 2008; Rutter, Moffitt, and Caspi 2006; Carver, Johnson, and Joormann 2008). The present study takes this line of research in a different direction.

To begin, past genetically informed studies of crime and other antisocial behaviors have assumed a diathesis-stress paradigm where particular genetic alleles increase an individual's vulnerability to environmental adversity. This perspective investigates the extent to which particular variants of a gene amplify the probability that exposure to some adverse social condition, such as abusive parenting, will lead to a problem behavior (e.g., Caspi et al., 2002). In contrast, the present study is informed by the differential plasticity perspective recently proffered by Jay Belsky and his colleagues (Belsky, Bakermans-Kranenburg, and von IJzendoorn 2007; Belsky and Pluess 2009a, 2009b).

Belsky and his collaborators (Belsky and Pluess 2009a, 2009b) have reviewed a number of studies reporting that adverse environmental conditions are more likely to lead to problem behaviors when individuals possess a particular genotype. Although these studies appear to support a stress-diathesis model,, Belsky and company conclude that a careful inspection of the results points to a different interpretation. Rather than simply showing that some individuals are more vulnerable to adversity than others, they assert that the data support the idea that some people are genetically predisposed to be more susceptible to environment influence than others. They contend that those persons most vulnerable to adverse social environments are the same ones who reap the most benefit from environmental support. In other words, some people are programmed by their genes to be more sensitive to environmental context, for better or worse (Belsky, Bakermans-Kranenburg, and van IJzendoorn 2007). The present study tests several implications of this proposition.

First, we test the differential susceptibility hypothesis by examining the extent to which individuals with particular variants of the genes 5-HTT, DRD4, and MAOA show higher rates of aggression than the comparison group when exposed to the conditions described by Anderson but lower rates of aggression than the comparison group when the environment is benign. More importantly, however, we investigate the extent to which these genetic

variants also interact with the environment to predict acquisition of the street code. Virtually all GxE studies have focused upon general behavioral outcomes such as aggression or conduct problems. Presumably, however, genes are related to such behavioral outcomes because they influence brain processes involving patterns of attending, emotion, and learning (Shanahan, 2010; Simons et al., 2010). If the differential plasticity model is correct, some individuals are genetically predisposed to be more attentive and responsive to their environment than others. Thus individuals with this genotype would be expected to learn the street code more quickly when exposed to the adverse conditions described by Anderson than other genotypes. Conversely, these same environmentally susceptible individuals would be expected to show the lowest levels of commitment to the street code when they grow up in a benign milieu. Finally, our analyses examine the extent to which the effect of the interaction of environmental adversity and genotype on aggression is fully mediated by the interaction of environmental adversity and genotype on street code. In other words, as elaborated below, we test a mediated moderation model.

Before turning to a more detailed consideration of our model and hypotheses, we feel compelled to issue the following caveat. The DRD4, 5-HTT, and MAOA polymorphisms that are the focus of our study are widely prevalent among all racial and ethnic groups and there is virtually no evidence suggesting that they are directly associated with aggression and antisocial behavior (Hohmann et al. 2009; Rutter et al. 2006). Indeed, the differential plasticity perspective predicts the absence of such direct effects (Belsky et al., 2007; Belsky & Pluess, 2009). Thus our concern is not with the main effects of genetic variation; rather our focus is on whether environmental conditions interact with 5-HTT, DRD4, and MAOA alleles in a “for better or worse” manner to predict commitment to the street code and involvement in violent behavior during early adulthood These ideas are developed more fully in the following sections.

THE STREET CODE AS AN ADAPTATION TO ADVERSE SOCIAL CONDITONS

The code of the street emphasizes maintaining the respect of others through a violent identity (Anderson, 1997, 1999). It entails displaying a tough demeanor and exacting retribution when someone disrespects (or “disses”) you (see also Baron, Kennedy, & Forde, 2001; Cao, Adams, & Jensen, 1997; Luckenbill & Doyle, 1989; Oliver, 1994, 2006; Wilkinson, 2003). Thus, the code regulates the use of violence. Anderson (1999) argues that this orientation is an adaption to various hostile and demoralizing community and family conditions.

First of all, the code is an adaptation to living in disadvantaged areas where violence is common. Such an environment fosters mistrust of others and development of the belief that one must project a tough, aggressive image in order to avoid exploitation (Anderson, 1999; Oliver, 1994, 2006; Sullivan, 1989; Vélez, 2001). In an effort to shield against victimization, residents learn that they must use or appear ready to use violence to defend their lives, property, and honor (Anderson, 1999; Courtwright, 1996; Fagan & Wilkinson, 1997; Oliver, 2006).

In addition to community violence, Anderson (1999) contends that exposure to discrimination contributes to the adoption of the street code. He argues that being a victim of discrimination causes individuals to adopt the view that people are untrustworthy and exploitive and that violence is a necessary and legitimate means for defending oneself against unjust treatment. Consistent with this thesis, studies have reported that being a victim of racism and discrimination increases the likelihood that adolescents will come to view street code behaviors as necessary and effective interpersonal strategies for solving

problems and garnering respect (Simons, Chen, Stewart, & Brody, 2003; Simons et al., 2006).

Finally, Anderson asserts that family environment influences the extent to which an adolescent adopts the code of the street. Using the vernacular of the residents of the neighborhoods included in his ethnographic research, he argues that two opposing family orientations—decent and street—often coexist in the same disadvantaged African American neighborhoods. Most families are "decent" in that the parents are committed to mainstream values and encourage their children to work hard and to avoid trouble. Decent families tend to be authoritative and vigilant in their parenting styles, punishing problem behaviors and discouraging violence (Burton & Jarrett, 2000; Furstenberg, Cook, Eccles, Elder, & Sameroff, 1999; Jarrett, 1997). Although they are stern, they also show high levels of warmth and support and spend quality time with their children.

In contrast, street families do not embrace mainstream values and often show a lack of consideration for others. Anderson (1999) describes street families as having home lives that are disorganized, and filled with anger, hostility, physical altercations, and antisocial behavior that is often observed by their children. They frequently engage in ineffective parenting strategies, such as yelling, poor supervision, verbal insults, and harsh and inconsistent discipline. There is often little explanation given for harsh, explosive punishments, and children learn to solve problems through violence. Anderson (1999) suggests that street families are so immersed in the code of the street that they aggressively socialize their children into it as a normative process.

Summarizing these arguments, adolescents raised in street oriented families and communities characterized by violence and discrimination are at risk for developing a commitment to the code of the street that, in turn, increases the likelihood of engaging in aggressive behavior (Stewart & Simons, 2006, 2010). Stated differently, the street code operates as a cognitive schema that mediates much of the association between adverse family and community characteristics and involvement in violence (Stewart & Simons, 2006, 2010).

We focus upon persistent exposure across the adolescent years to several of the hostile and demoralizing community and family conditions specified by Anderson (1999): community crime, low social control, racial discrimination, and a violent, antisocial caregiver. For the sake of parsimony, we utilize a cumulative measure of exposure to these various social conditions. We expect that this cumulative measure of hostile, demoralizing treatment will predict adult aggression, and that commitment to the street code will mediate much of this association. Street code is expected to mediate only a portion of the association between hostile, demoralizing treatment during adolescence and adult aggression as such environmental conditions undoubtedly foster the development of several social psychological factors that increase the probability of criminal behavior (e.g., anger, hostile attribution bias, low self-control) besides adoption of the code of the street (Simons & Burt, 2011). Although we expect our analysis to show that commitment to the street code moderates a significant proportion of the association between persistent exposure to a hostile, demoralizing environment and aggression, that it not the major focus of our paper. Rather, our purpose is to investigate the extent to which genetic polymorphisms modulate the effect of a hostile, demoralizing environment on adoption of the street code and aggressive behavior in a manner consistent with the differential susceptibility hypothesis.

DIFFERENTIAL SUSCEPTABILITY TO CONTEXT

Most research investigating the molecular genetic basis of aggressive and antisocial behavior has focused upon variations in genes involved in regulation of the serotonergic and

dopaminergic neurotransmitter systems. Much of this research has focused upon the serotonin transporter gene (5-HTT), the dopamine D4 receptor (DRD4), and the neurotransmitter–metabolizing enzyme monoamine oxidase A (MAOA).

Studies of the serotonin transporter gene have concentrated upon a polymorphism located in the promoter region (5-HTTLPR) that consists of 14 or 16 repeats of a 22 bp unit (Murphy, Lerner, and Rudnick 2004). Most research distinguishes between those carrying at least one short allele (s/s, s/l) and those homogenous for the longer allele (l/l). The short allele has been linked to reduced expression of the serotonin transporter molecule (Lesch et al. 1996; Champoux et al. 2002). Approximately half of European Americans and African Americans carry at least one copy of the short allele.

Although research on the direct effect of 5-HTTLPR on antisocial behavior reports mixed results, others studies have reported evidence suggesting that it is in interaction with an adverse environment that variations in this gene influence the chances of problem behavior. In both humans and non-human primates, unfavorable early rearing experiences have been related to negative emotionality and aggression for individuals carrying the s-allele (Barr et al. 2004a and 2004b; Pauli-Pott, Friedl, Hinney and Hebebrand 2009; Schwandt et al. 2010). Also, compared to those with the l-allele, males carrying the s-allele are at increased risk for aggression in response to environmental stressors (Verona, Loiner, Johnson, and Bender 2006) and more prone to violent criminal behavior if they were raised in an adverse environment (Reif et al. 2007; Retz et al. 2008). And, most recently, Brody et al. (forthcoming) found the longitudinal association between racial discrimination and adolescent conduct problems to be strongest among males with the s-allele of 5-HTTLPR.

The DRD4 gene has a polymorphic 48 bp variable number of tandem repeats (VNTR) in exon III. This polymorphism varies from 2 to 11 copies. There is a long (l-DRD4; 6–8 repeats) and a short polymorphism group (s-DRD4; 2–5 repeats). Although the s-allele is most common, roughly half of European Americans and African Americans carry at least one copy of the l-allele. Evidence suggests that the longer exon III repeats code less efficiently at the level of transcription, translation, and second messenger generation than the shorter repeats (Ebstein 2006; Oak and Van Tol 2000). Although the effects are often quite small, there is evidence suggesting an association between the l-allele of DRD4 and antisocial behavior (De Young et al. 2006; Schmidt et al. 2002). Of greater importance for our purposes are studies suggesting that the effects of the DRD4 gene on aggression is moderated by environmental factors. These studies indicate that persons carrying the DRD4 l-allele are at high risk for conduct problems if they were exposed to non-optimal parenting during childhood (Bakermans-Kranenburg and van IJzendoorn 2006; Propper et al. 2007).

Finally, several studies of antisocial behavior have focused upon polymorphisms in the MAOA gene. This gene selectively degrades serotonin, norephinephrine, and dopamine and therefore plays a key role in regulating behavior (Sabol, Hu, & Hamer, 1998; Shih, Chen, & Ridd, 1999). It is located on the X chromosome and therefore males have a single copy of the gene. The prevalent functional polymorphism involves a low activity genotype (MAOA-L) consisting of those with the 2R, 3R, or 4R alleles and a high activity genotype (MAOA-H) consisting of those with alleles of more than 4 repeats. Roughly 40–50% of males have the low activity genotype. Several studies have reported that MAOA moderates the effect of childhood exposure to harsh and abusive parenting and subsequent involvement in antisocial behavior. This research finds that the link between childhood maltreatment and either adolescent conduct problems or adult aggression is much stronger for the MAOA-L genotype than for the MAOA-H genotype (Caspi et al., 2002; Ducci et al., 2008; Foley et al., 2004; Kim-Cohen et al., 2006; Nilsson et al., 2006; Widom & Brzustowicz, 2006).

Summarizing, several studies indicate that individuals with either the 5-HTTLPR s-allele, the DRD4 l-allele, or the MAOA l-allele tend to show high levels of aggression if they have experienced environmental adversity. These findings are usually interpreted within a stress-diathesis perspective where risk alleles are viewed as diatheses that amplify the probability that some adverse circumstance, such as harsh parenting, will foster aggression. As noted earlier, however, Belsky and his colleagues (Belsky, Bakermans-Kranenburg, and von IJzendoorn 2007; Belsky and Pluess 2009a, Belsky and Pluess, 2009b) have argued for a different interpretation. They posit that individuals with these genetic alleles are not simply more sensitive to adverse conditions; they are also more sensitive to supportive conditions. They label this idea the differential susceptibility hypothesis and suggest that polymorphisms in genes such as 5HTT, DRD4, and MAOA influence the extent to which individuals are responsive to environmental context with some individuals being programmed by their genes to be more sensitive or plastic than others (Belsky, Bakermans-Kranenburg, and van IJzendoorn 2007).

Support for the differential susceptibility or plasticity argument is evident when the slopes for a gene by environment interaction show a crossover effect with the susceptibility group showing worse outcomes than the comparison group when the environment is negative but demonstrating better outcomes than the comparison group when the environment is positive (Belsky et al. 2006; Belsky and Pluess 2009). Recently, Belsky and Pleuess (2009) reviewed scores of GxE studies that detected interactions of a crossover nature. In most of these studies, however, this pattern was not recognized or discussed because the authors were operating out of the stress-diathesis paradigm. Several of these studies focused upon 5HTTLPR, DRD4, and MAOA.

How would genes cause some individuals to be more sensitive than others to their environment? Belsky & Pleuss (2009) observe that the genes included in the studies that they reviewed involved the dopaminergic system which has been implicated in reward sensitivity and the 10 serotonergic system which has been linked to sensitivity to punishment and displeasure (see Carver, Johnson, and Joormann 2008; Frank et al. 2007). They therefore posit that some individuals may be more responsive to their environment than others because they have different thresholds for experiencing pleasure or displeasure. That is, because of their genetic endowment, the behavior of some individuals may be more readily shaped by salient environmental rewards and punishments than are others. Further, they go on to speculate that the more plasticity alleles one carries, the more susceptible he or she will be to environmental context. At least three study has provided support for this idea (Belsky & Beaver, 2010; Belsky, Pluess, Comings, and MacMuray 2010; Simons et al., 2010).

The present study extends past gene by environment studies of aggression in several ways. First, with only a few exceptions (e.g., Simons et al., 2010) past gene by environment studies of antisocial have largely limited their focus to one type of environmental adversity – abusive parenting during the childhood years. We extend this research by examining exposure to a range of hostile and demoralizing community and family conditions. Adversity is most apt to foster aggression when it is persistent and occurs in a variety of forms. Thus we form a composite measure of these factors as they occur across the adolescent years. We investigate the extent to which the effect of our composite measure of hostile, demoralizing treatment on adult aggression is moderated by genotype in the manner predicted by the environmental susceptibility hypothesis.

Second, we extend prior research by testing Belsky and Pleuss' (2009) contentions regarding cumulative susceptibility. Whereas most studies focus upon a single gene, we examine the combined effect of having the l-allele DRD4, s-allele 5HTTLPR, and L-allele MAOA. Since

these genes influence sensitivity to *both* pleasurable (dopamine system) and aversive (serotonin system) circumstances (Robbins and Everitt, 1999), persons with this genotype might be expected to show greater responsiveness than those with only one of these alleles to the full range of events that occur in their everyday. If the susceptibility hypothesis is correct, persons with this genotype should display higher levels of aggression than the comparison group when exposed to an adverse environment such as wide-ranging and recurrent adverse treatment, but lower levels of aggression than the comparison group when the environment is benign or supportive. The present study tests this idea.

Third, and most importantly, we go beyond prior studies by testing models that include a cognitive schema – code of the street - that has been linked to aggression. If some individuals are genetically predisposed to be more sensitive to their environment than others, they would be expected to show more of an emotional response to environmental conditions and to learn the lessons inherent in recurrent environmental events more quickly than less environmentally sensitive individuals (Simons et al., 2010). Thus we expect individuals with a combination of l-allele DRD4, s-allele 5HTTLPR, and l-allele MAOA to score higher than others on code of the street when hostile and demoralizing conditions are highly prevalent, but to score lower than others on code of the street in the absence of such adverse conditions. The stress-diathesis paradigm would make the first prediction but not the second.

Finally, we examine the extent to which the effect of the interaction of a hostile, demoralizing environment and genotype on crime is mediated, in large measure, by street code. As just noted, the differential susceptibility perspective would suggest that genotype modulates the probability that a hostile, demoralizing environment will result in the adoption of the street code. Past research has established that commitment to the street code mediates much of the effect of a hostile, demoralizing environment on perpetration of violent acts (e.g., Stewart & Simons, 2006, 2010). Elaborating this model to include genetic effects, we expect that a significant proportion of the interaction of genotype and environmental adversity on aggression will be mediated by commitment to the street code. In other words, we expect street code to operate as a mediated moderator. We are not aware of only one study (viz., Simons et al., 2010) that has investigated the extent to which cognitive factors mediate the effect of GxE on antisocial behavior. Such effects would be expected if the differential susceptibility argument is correct.

RESEARCH DESIGN

Sample

We tested our hypotheses using data from waves 2 through 5 of the Family and Community Health Study (FACHS), a multi-site investigation of neighborhood and family processes that contribute to African American children's development in families living in a wide variety of community settings. The FACHS sample consists of several hundred African American families living in Georgia and Iowa at the initiation of the study. Additional details regarding the sampling procedures utilized can be found in Gibbons et al. (2004) and Simons et al. (2002).

The first wave of the FACHS data were collected in 1997–1998 from 867 African American, fifth-grade children (417 boys and 450 girls; 445 from Iowa and 422 from Georgia). The second, third, fourth, and fifth waves of data were collected in 1999–2000, 2001–2002, 2004–2005, and 2007–2008 to capture information when the target children were ages 12–13, 14–15, 17–18, and 20–21, respectively. Of the 867 targets interviewed at wave 1, 779 were reinterviewed at Wave 2; 767 at Wave 3; 714 at Wave 4, and 687 at wave 5 (79% of the original sample). As part of wave 5 data collection, targets were asked to provide DNA (saliva sample) for purposes of genetic analyses. Of the 689 participants, 544

(80%) agreed to do so. Successful genotyping for MAOA, 5-HTTLPR, and *DRD4* was achieved for 224 males and 320 females. The present study focuses only on the male respondents as commitment to the street code and involvement in violence is much more common among males than females and the MAOA gene is located on the sex chromosome and its allelic variation appears to have little impact upon the aggressive behavior of females.

Current study participants

Our analyses are based upon the 224 males who could be genotyped for MAOA, 5-HTTLPR, and DRD4. Analyses indicated that those individuals who did not participate at various waves did not differ significantly from those who participated with regard to age, sex, delinquency, primary caregivers' education, household income, or neighborhood characteristics. To further assess attrition bias, we used Heckman's (1979) two-step procedure to estimate sample selection bias. Including Heckman's Lambda in our models did not change the findings. There were no differences between those remaining in the panel and those dropping out with regard to a variety of community and family measures.

Procedures

The questionnaires were administered in the respondent's home and took on average about 2 hours to complete. In waves 1 – 4, the instruments were presented on laptop computers. Questions appeared in sequence on the screen, which both the researcher and participant could see. The researcher read each question aloud and the participant entered an anonymous response using a separate keypad. At wave 5, in an effort to further enhance anonymity, the questionnaires were administered using audio-enhanced, computer-assisted, self-administered interviews (ACASI). Using this procedure, the respondent sat in front of a computer and responded to questions as they are both presented visually on the screen and auditorily via earphones.

Participants' were also asked to contribute DNA at wave 5 using Oragene™ DNA kits (Genotek; Calgary, Alberta, Canada). Those who chose to participate rinsed their mouths with tap water, and then deposited 4 ml of saliva in the Oragene sample vial. The vial was sealed, inverted, and shipped via courier to a laboratory at the University of Iowa, where samples were prepared according to the manufacturer's specifications.

VARIABLE MEASUREMENT

Delinquency/Aggression

Delinquency at wave 1 was assessed using respondent self-reports on the conduct disorder section of the Diagnostic Interview Schedule for Children, Version 4 (DISC-IV). Respondents reported whether in the past year they had engaged in 18 delinquent behaviors (1 = yes; 0 = no) such as shoplifting, lying, setting fires, cruelty to animals, vandalism, and burglary. Coefficient alpha for the instrument was .91.

Aggression at wave 5 was assessed using 8 items adapated from Elliott's (Elliott, Huizinga, & Ageton, 1985; Elliott, Huizinga, & Menard. 1989) widely used instrument. Respondents reported whether in the past year they had engaged in aggressive behaviors such as fighting with weapons, carrying a hidden weapon, shooting or stabbing someone, purposefully damaging property, hurting someone, or pulling a knife on someone. The maximum possible score of eight corresponds to a subject responding that he engaged in all of the different acts. Coefficient alpha for the instrument was .76.

Hostile/Demoralizing Environment

This composite measure included five components: harsh/inept parenting, caregiver antisocial behavior and substance use, racial discrimination, community crime, and absence of social control. Harsh/inept parenting was assessed at wave 2 – 4 using a 20 item scale concerned with harsh, explosive parenting, inconsistent discipline, lack of monitoring, and absence of warmth (see Simons, Simons, Chen, Brody, & Lin, 2007). Response format for the items ranged from 1 (never) to 4 (always). Coefficient alpha was .84, .89, and .79 for waves 2, 3, and 4 respectively. Scores were standardized and then summed across waves.

Caregiver antisocial behavior was assessed using the primary caregiver's responses to the antisocial personality and substance abuse sections of the Composite International Diagnostic Instrument (CIDI) developed at the University of Michigan for the NIMH National Comorbidity Study (UM-CIDI;Kessler 1991). A variety of studies point to the validity of the diagnostic classifications rendered by the UM-CIDI (Anthony et al., 1985; Robins & Reiger 1991). The antisocial behavior section contains a series of 27 questions regarding how often during the preceding year the respondent engaged in various crimes acts such as shoplifting, physical assault, lying, setting fires, vandalism, burglary, and robbery. The substance use section included measures for past year use of marijuana and other types of illegal drugs including cocaine, heroin, inhalants, and stimulants. Coefficient alpha for the measure was approximately .80 at each wave. Scores were standardized and then summed across waves to form a composite score of the primary caregiver's antisocial behavior and substance use.

Racial discrimination was assessed at waves 2–4 using the 13-item Schedule of Racist Events. This instrument has strong psychometric properties and has been used extensively in studies of African Americans of all ages (Klonoff and Landrine 1999). The items focus on the extent (1 = never, 4 = several times) to which respondents experienced various discriminatory events during the preceding year (e.g., How often has someone yelled a racial slur or racial insult at you just because you are African American? "How often have the police hassled you just because you are African American? How often has someone threatened you physically just because you are African American?). Coefficient alpha for the scale was roughly .90 at each wave. Scores were standardized and then summed across waves to form a composite measure of persistent exposure to discrimination.

Community crime was assessed at waves 2–4 using a revised version of the community deviance scale developed for the Project on Human Development in Chicago Neighborhoods (PHDCN; Sampson et al. 1997). This 5-item measure asks respondents to report how often behaviors such as fighting with weapons, gang fights, violent arguments between neighbors, sexual assaults, or robberies occur within their community. Coefficients alpha was .62, .78, and .79 at waves 2, 3, and 4, respectively. Scores were standardized and then summed across waves to form a composite measure of persistent exposure to community crime.

Neighborhood social control was assessed using a scale adapted from the Project on Human Development in Chicago Neighborhoods (PHDCN; see Sampson, Raudenbush, and Earls, 1997). The first scale asks respondents to rate (1 = very likely, 4 = very unlikely) the extent to which neighborhood residents can pick out outsiders who obviously don't live in the area and the probability that would do something about it if children spray-painted graffiti on a building, showed disrespect to an adult, or skipped school and were hanging out on a street corner. Coefficient alpha for this 4-item scale was approximately .60 at each wave.

Finally, factor scores from the principal component analysis were used to form the measure of hostile/demoralizing environment. The factor scores were .62 for racial discrimination, . 75 for harsh/inept parenting, .67 for neighborhood crime, and .38 for antisocial parent.

Commitment to the Street Code

At wave 5, the respondents completed the 7-item street code scale developed by Stewart (Stewart & Simons 2010). This instrument asks respondents to indicate how much they agree (1=strongly disagree; 4=strongly agree) with statements such as: People do not respect a person who is afraid to fight for his/her rights; People tend to respect a person who is tough and aggressive; and, Being viewed as tough and aggressive is important for gaining respect; Coefficient alpha for this scale was .85.

Genotyping

Genotype at DRD4 was determined for each youth as described by Lichter et al. (1993). This approach involved using the primers F-CGCGACTACGTGGTCTACTCG and R-AGGACCCTCATGGCCTTG, standard *Taq* polymerase and buffer, standard dNTPs with the addition of 100 μM 7-deaza GTP and 10% DMSO. The resulting PCR products were electrophoresed on a 6% non-denaturing polyacrylamide gel and the products visualized using silver staining. Following genotyping of DRD4, individuals were classified into one of two groups (Dreber et al., 2009): 1= alleles with at least one 7 or longer repeat (l-allele); 0 = alleles with two less than 7 repeats (s-allele). Using this criteria, 75 (39.5%) individuals were classified as l-allele.

Genotype at 5-HTTLPR located on chromosome 17q11.1-q12 has a functional polymorphism in the variable repeat sequence in the promoter region (Bradley, Dodelzon, Sandhu, and Philibert 2005). The homozygous long allelic variant (16 or 18 repeats) is related to higher concentrations of 5-HTT messenger RNA and a greater rate of reuptake than the homozygous short allelic variant (14 repeats). Based on previous studies, individuals were classified into two groups based upon 5-HTTLPR genotype: 1 = at least one s-allele (s-allele); 0 = pair of long alleles (l-allele). Using this criteria, 94 (49.5%) individuals were classified as s-allele.

The neurotransmitter-metabolizing enzyme monoamine oxidase A (MAOA) is an enzyme and catalyzes dopamine. The MAOA is located in Xp11.3–11.4 and are more likely relate to males' violent behavior and delinquency. The reason is that the MAOA gene is on the X chromosome, and males have a single copy of the MAOA. According to previous studies, individuals were classified into two groups: 1 (MAOA-L) = pair of 2R, 3R, or 3R alleles; 2 (MAOA-H) = other alleles. Using this criteria, 100 (52.6%) individuals were classified as MAOA-L.

Finally, the three genotypes (DRD4, 5-HTTLPR, and MAOA) were summed to form a measure of cumulative plasticity alleles: 0 plasticity allele (15.3%), 1 plasticity allele (36.8%), 2 plasticity alleles (38.9%), and 3 plasticity alleles (8.9%).

ANALYTIC STRATEGY

Hierarchal regression models were employed to test the differential susceptibility hypothesis. We used the statistical software Mplus 6.1 (Muthen and Muthen 2010). Post hoc analyses of significant interaction terms were conducted using the Johnson-Neyman (J-N) technique (Johnson and Neyman 1936; Hayes and Matthes 2009). This procedure identifies regions of significance for interactions between continuous and categorical variables.

Next, we employed the mediated-moderation model (Morgan-Lopez et al. 2006; Little et al. 2007) to examine the extent to which adopting the street code mediates the main effect of hostile/demoralizing environment, and, more importantly, the dual effects of environment and genotype, on delinquency. Given that delinquency is a count variable, we considered using the negative binomial procedure to do our analyses. A major concern of our paper, however, is mediated moderation and the indirect effects of variables. The mediated-moderation model and statistical test for indirect effects cannot be performed with the negative binomial procedure. Therefore, we used robust standard errors and bootstrapping to adjust model fit for the non-normal distributions of the symptom count variable and to adjust the standard errors of the parameter estimates (Efron and Tibshirani 1991).

To assess the goodness-of-fit of our models, Standardized Root Mean Squared Residual (SRMR; Browne and Cudeck 1992), the comparative fit index (CFI; Bentler 1990), and the chi-square divided by its degrees of freedom (fit ratio) were used. The CFI is truncated to the range of 0 to 1 and values close to 1 indicate a very good fit (Bentler 1990). A RMSEA smaller than .05 indicates a close fit, whereas a RMSEA between .05 and .08 suggests a reasonable fit (Browne and Cudeck 1992).

RESULTS

Table 1 presents the means, standard deviations, and zero-order correlations among all variables. As expected, respondents who reported persistent exposure to a hostile/ demoralizing environment report commitment to the street code (r = .22) and involvement in aggressive behavior (r = .21). Commitment to the street code is also related to aggression (r = .23). Cumulative plasticity, on the other hand, is not associated with either street code or aggression. This finding is consistent with previous molecular genetic studies indicating that so-called risk alleles generally have little main effect on conduct problems or antisocial behavior (Caspi et al. 2003; Moffitt et al. 2006). Table 1 also shows that there are no significant correlations between hostile/demoralizing environment and cumulative plasticity alleles, indicating that there is no gene-environment correlation (rGE) in our study.

The Effect of G × E on Adopting the Street Code and Aggression/Crime

Table 2 shows the results of using hierarchical regression models to investigate the effects of hostile/demoralizing environment and genetic plasticity on commitment to the street code and aggression, controlling for earlier assessments of these outcomes. Thus the focus is upon change in these variables. Family structure and family SES were included as controls in the all of the models. Models 1a and 2b show the main effects of the two independent variables. Model 1a indicates that hostile/demoralizing environment has a significant effect on increases in street code (β = .19) whereas genetic plasticity does not. Model 2a shows a similar pattern using aggression as the outcome. Hostile/demoralizing environment predicts increased aggression (β = .19) but genetic plasticity has no significant effect. Models 1b and 2b add the interaction of hostile/demoralizing environment with cumulative plasticity. As expected, this interaction term is a significant predictor of change in both street code (β = . 32) and aggression (β = .31). Indeed, Table 2 shows that hostile/demoralizing environment no longer has a significant effect on either street code or aggression once this interaction term is added to the model. Thus the effect of hostile/demoralizing environment on these two variables is largely limited to those who possess multiple plasticity alleles. Further, adding the interaction term increases the amount of variance explained in adoption of the street code by 23% and the amount of variance explained in aggression by 60%.

Having established gene-environment interaction effects, the next step was to graph these interactions to see if they display the cross-over pattern predicted by the differential susceptibility argument. Figure 1 depicts the association between hostile/demoralizing

environment and adoption of the street code for respondents with zero, one, two or three plasticity alleles. Using the simple slope procedure (Aiken and West 1991), the slopes for respondents with one, two or three genetic plasticity alleles are significantly different from zero, whereas the slope for those with no plasticity alleles does not differ from zero. More importantly, the slopes are steeper the greater the number of plasticity alleles and demonstrate the expected crossing pattern. The graph shows that individuals with several plasticity alleles are more likely than those with few to adopt the street code when exposed to a hostile/demoralizing environment. Persons with these genotypes are less likely than those with few plasticity alleles, however, to adopt the street code in the absence of a hostile/demoralizing environment. The Johnson-Neyman (J-N) technique (Preacher, Curran, and Bauer 2006) was used to investigate the significance of this pattern of findings. As shown in Figure 1, cumulative genetic plasticity significantly ($p < .05$) increases an individual's adoption of the street code when level of hostile/demoralizing environment is above the 44nd percentile, whereas it decreases adoption of the street code when hostile/demoralizing environment is less than the 11th percentile.

Figure 2 shows a similar pattern of findings when aggression is the outcome. All of the slopes differ from zero except those for individuals with no plasticity alleles. Consonant with the differential susceptibility hypothesis, the slopes are steeper the greater the number of plasticity alleles and demonstrate the expected crossing pattern. The graph shows that respondents with several plasticity alleles are more likely than those with few to engage in aggression when exposed to a hostile/demoralizing environment, whereas those with multiple plasticity alleles are less likely than those with few plasticity alleles to engage in aggression in the absence of a hostile/demoralizing environment. Using the J-N technique, hostile, cumulative plasticity significantly increases an individual's involvement in aggression when hostile, demoralizing environment is greater than the 59th percentile while it significantly ($p < .05$) decreases delinquency when it is less than the 17nd percentile. These findings provide strong support for the differential susceptibility hypothesis.

The Mediating Effect of Adopting the Street Code

The next step in our analysis was to examine the extent to which street code meditated the effects of gene-environment interaction on aggression. We began with a simple mediating model without the genetic variables. As shown in Figure 3, this model provides a good fit to the data. Hostile/demoralizing environment is related to adopting the street code ($\gamma = .19$, $p < .05$) which, in turn, is related to aggression ($\beta = .20$, $p < .05$). Using SEM with the bootstrap procedure for small samples, the indirect effect of hostile/demoralizing environment on aggression through adopting the street code is significant (indirect effect = .04, $p < .05$) and accounts for 21% of the total variance. This is consistent with previous studies demonstrating that street code is an important mediator of the effects of structural conditions on violent and deviant behavior (see Stewart and Simons, 2006).

Next, we expanded the model to include the main effect of cumulative genetic plasticity and the interaction of cumulative genetic plasticity with hostile/demoralizing environment. This mediated-moderation model is designed to test the extent to which the interaction of cumulative genetic plasticity and hostile/demoralizing environment on aggression is mediated by adoption of the street code. As shown in Figure 4, the model fit the data well by all criteria, χ^2 (2, n=190) = 3.74, $p > .05$, CFI = .96, and SRMR = .02.

The model shows that the association between cumulative genetic plasticity and hostile/demoralizing environment is not significant, indicating no gene-environment correlation. Turning to the paths in the model, cumulative genetic plasticity, as expected, is not related to either street code or aggression. However, also as expected, the interaction of cumulative plasticity with hostile/demoralizing environment is significantly associated with adopting

the street code (γ = .32). Adding this GxE effect increases the variance explained in commitment to street code by 20% (R^2 increases from .15 to .18). Further, including this GxE path reduces the GxE effect on aggression from .31 to .26 and the indirect effect of GxE on aggression through street code is significant (indirect effect=.06) and accounts for 18% of the total variance. These findings support our mediated-moderator hypothesis as a significant proportion of the moderating influence of cumulative genetic plasticity on the association between hostile/demoralizing environment and aggression is mediated by the moderating effect of cumulative genetic plasticity on the association between hostile/demoralizing environment and adoption of the street code.

DISCUSSION

Most gene by environment studies employ a stress-diathesis perspective where a genetic risk is either amplified by an adverse environment or muted by a supportive environment (Shanahan and Hofer 2005). In contrast, the present study was informed by the recently articulated differential susceptibility perspective (Belsky and Pluess 2009). Rather than treating alleles as diatheses, this model posits that some people are genetically predisposed to be more susceptible to environment influence (i.e., they are more plastic) than others. Thus those persons most vulnerable to adverse social environments are also those who reap the most benefit from environmental support (Belsky, Bakermans-Kranenburg, and van IJzendoorn 2007). Focusing upon three genotypes – s-allele 5HTTLPR, l-allele DRD4 and l-allele MAOA - we found strong support for the perspective.

We found that exposure to hostile, demoralizing environmental conditions interacted with our index of cumulative genetic plasticity to predict involvement in aggressive behavior. The graph of this interaction showed the expected cross-over pattern. Individuals high on genetic plasticity showed greater violence than other genotypes in response to an adverse environment, whereas they showed less violence than other genotypes when the social environment was more benign.

Further, our results support the idea that the combination environmental adversity and genetic plasticity increase the probability of aggression because of the cognitive schemas that it engenders. If some individuals are genetically predisposed to be more sensitive to their environment than others, they would be expected to learn the lessons implicit in recurrent environmental events more quickly than less environmentally sensitive individuals. Consistent with this idea, respondents with high cumulative genetic plasticity scored higher than others on adoption of the street code when exposure to maltreatment was extreme. On the other hand, individuals with this genotype scored lower than others on adoption of the street code when adversity was minimal. Further, our results indicated that the interaction of genotype and environmental adversity on aggression is mediated by adoption of the street code. With the exception of Simons et al. (2010), we are not aware of any prior studies that have investigated the extent to which cognitive and emotional factors mediate the effect of gene-environment interaction on aggression.

Overall, these results support that idea that individuals with the s-allele 5HTTLPR, l-allele DRD4, and l-allele MAOA are more sensitive to their social environment than those with other genotypes. As a consequence, these individuals at are high risk for developing a hostile view of people and a concern with toughness and aggression in response to an neighborhood violence, racial discrimination, and harsh, antisocial caregivers. On the other hand, these are the persons who are most likely to develop a peaceful, sanguine orientation in response to a kinder environment. These findings suggest a more optimistic view of aggressive and antisocial individuals. Whereas the stress-diathesis perspective paints such persons as difficult to change given their genetic tendency to be hyper-responsive to

adversity, the differential susceptibility model argues that that their environmental sensitivity makes them good candidates for intervention. They are more likely than those with differing genotypes to learn the lessons being taught by a new, more positive environment. Consistent with this view, Bakermans-Kranenburg et al. (2008) found that children with the l-allele DRD4 showed the largest decline in conduct problems in response to parent training, and Brody et al. (2009) recently reported that a family based-intervention with African American teens was most effective in reducing risky behavior for those with s-allele 5HTTLPR.

Although our study improved upon many of the limitations of past gene x environment studies of aggression, it was not without shortcomings. Perhaps the biggest weakness was the homogeneity of our sample; all of the respondents in our sample were African American. Use of an all African American sample had the benefit, however, of allowing us to investigate the manner in which environment and genotype combine to produce aggression among a racial/ethnic group at high risk for social adversity, adoption of the street code, and involvement in violent behavior (Unnever et al. 2009). Although we cannot think of any reason as to why our results would be specific to African Americans, our findings clearly need to be replicated with more diverse samples.

In conclusion, criminology is largely concerned with the effect of social context on people's behavior. Importantly, genetic variability is a factor that has been shown to influence a person's response to his or her social environment (Freeze 2008). In recent years, a wide variety of perspectives have emerged regarding the complex manner in which genes and the social environment might interact over the life course (Shanahan and Hofer 2005, 2011). Our results support the recently articulated differential susceptibility model which posits that a substantial proportion of any population is genetically predisposed to be more responsive to their social environment than those with other genotypes. The fact that genetic data is now available in many of the large-scale social scientific data sets (e.g., Adolescent Health) means that criminologists are now able to test the differential susceptibility model, as well as a variety of other perspectives, regarding the complex interplay of genes and social context. The consequence will most certainly be a more precise and comprehensive understanding of human behavior.

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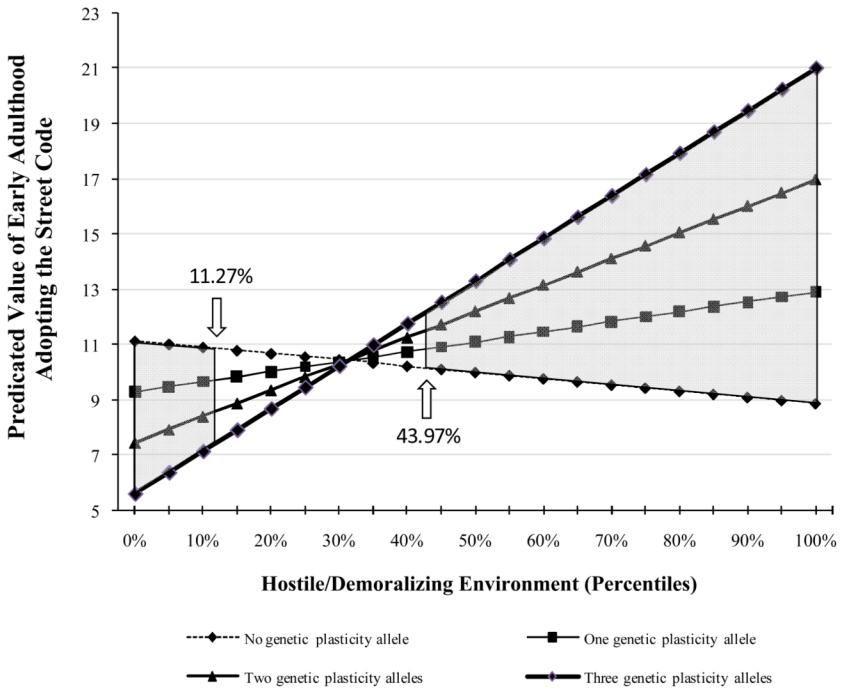

Figure 1.

The Effect of Hostile/Demoralizing Environment on Adopting the Street code by Number of Genetic Plasticity Alleles with Johnson-Neyman 95% Confidence Bands. The gray areas are significant confidence regions.

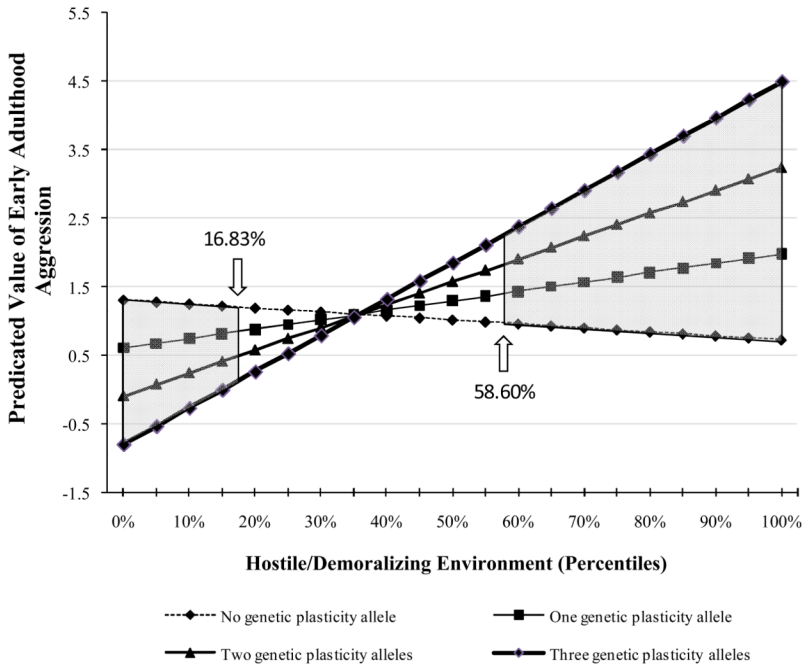

Figure 2.

The Effect of Hostile/Demoralizing Environment on Delinquency by Number of Genetic Plasticity Alleles with Johnson-Neyman Confidence Bands. The gray areas are significant confidence regions.

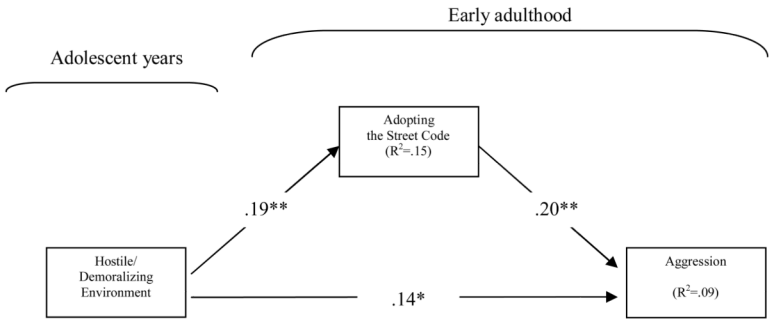

Figure 3.

Adopting the Street Code as Mediator of the Effect of Hostile/Demoralizing Environment on Aggression.

Note: χ^2=4.63, *df*=2, *p*=.10. SRMR=.02 and CFI=.95. The values presented are standardized parameter estimates. Previous delinquency, pervious commitment to the street code, region, family SES, and family structure are controlled.

**p≤.01; *p≤.05, †p<.10 (two-tailed tests), n=216

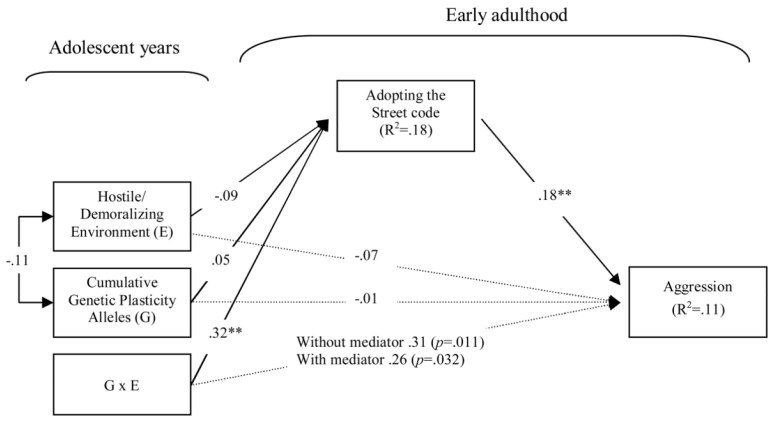

Figure 4.

Mediated Moderation Model.

Note: χ^2=3.11, *df*=2, *p*=.22, SRMR=.01 and CFI=.98. The values presented are standardized parameter estimates. Previous delinquency, pervious commitment to the street code, region, family SES, and family structure are controlled.

**$p \leq .01$; *$p \leq .05$ (two-tailed tests), n=216

Table 1

Correlation Matrix for the Study Variables.

	1	2	3	4	5	6	7
1. Hostile/demoralizing environment W2–W4	——						
2. Adopting the stree code W5	.22**	——					
3. Aggression W5	.21**	.23**	——				
4. Cumulative genetic plasticity alleles (0–3)	−.11	−.01	−.02	——			
5. Area (1 = South)	−.22**	.10	−.03	.05	——		
6. Family SES	−.02	.06	−.04	−.09	−.16*	——	
7. Family structure (1 = Single families)	−.01	.00	.01	.03	.03	−.28**	——
Mean	0	17.49	1.05	1.38	.56	.11	.48
SD	1	4.47	1.58	.87	.50	1.50	.50

** $p \leq .01$;

* $p \leq .05$ (two-tailed tests); n=216

Table 2

Hostile/Demoralizing Environment and Genetic Diversity as Predictors of Adopting the Street Code and Aggression (n=216).

	Adopting the street code				Aggression			
	Model 1a		Model 1b		Model 2a		Model 2b	
	b	β	b	β	b	β	b	β
Environment and gene								
Hostile/Demoralizing Environment (E)	.86** (.30)	.19	−.38 (.53)	−.08	.30** (.11)	.19	−.10 (.20)	−.06
Cumulative plasticity alleles (0–3)	.22 (.33)	.04	.23 (.33)	.05	−.02 (.13)	−.01	−.01 (.12)	−.01
Two-way interaction								
Cumulative plasticity alleles x (E)			.98** (.35)	.32			.33* (.13)	.31
Control variables								
Controlling early adolescent status	.32** (.08)	.28	.34** (.08)	.30	.21 (.15)	.10	.15 (.15)	.07
region (1 = South)	1.45* (.60)	.16	1.42* (.59)	.16	.02 (.22)	.01	.02 (.22)	.01
Family SES	.32 (.20)	.11	.28 (.20)	.09	−.04 (.08)	−.04	−.05 (.07)	−.05
Famuly structure (1 = Single Families)	.24 (.59)	.03	.03 (.59)	.00	.00 (.22)	.00	−.07 (.22)	−.02
Constant	10.49** (1.55)		10.32** (1.53)		1.03** (.26)		1.10 (.26)	
Adjusted R^2	.13		.16		.03		.05	
R^2 increase due to interaction			.03**				.03**	

Note: Standard errors are in parentheses;

** $p \leq .01$;

* $p \leq .05$,

† $p < .10$ (two-tailed tests).