Published in final edited form as:
Am Sociol Rev. 2011 December ; 76(6): 833–912. doi:10.1177/0003122411427580.

Social Environmental Variation, Plasticity Genes, and Aggression: Evidence for the Differential Susceptibility Hypothesis

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Abstract

Although G×E studies are typically based on the assumption that some individuals possess genetic variants that enhance their vulnerability to environmental adversity, the differential susceptibility perspective posits that these individuals are simply more susceptible to environmental influence than others. An important implication of this model is that those persons most vulnerable to adverse social environments are the same ones who reap the most benefit from environmental support. The present study tested several implications of this proposition. Using longitudinal data from a sample of several hundred African Americans, we found that relatively common variants of the dopamine receptor gene and the serotonin transporter gene interact with social environmental conditions to predict aggression in a manner consonant with differential susceptibility. When the social environment was adverse, individuals with these genetic variants manifested more aggression than other genotypes, whereas when the environment was supportive they demonstrated less aggression than other genotypes. Further, we found that these genetic variants interact with environmental conditions to foster various cognitive schemas and emotions in a manner consistent with differential susceptibility and that a latent construct formed by these schemas and emotions mediated the effect of gene by environment interaction on aggression.

In recent years genetic determinism has died a quiet death. The evidence is overwhelming that human beings are never simply instructed by their genes to engage in a particular trait or behavior. Rather, we live in variable environments and the sets of genes that are turned on (i.e., expressed) and the messages they transcribe vary depending upon environmental circumstances (Kandel 2006; Pennisi 2001; Shanahan and Hofer 2011). This new perspective on genetics underscores the importance of environmental context and of

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formulating models of human behavior that take into account the interplay of sociocultural and genetic variables (Shanahan and Boardman 2009).

This approach is perhaps most evident in the explosion of recent articles reporting gene by environment interactions (G×E). These studies find that genetic variation often interacts with environmental context to influence the probability of particular behaviors (see Rutter, Moffitt, and Caspi 2006; Shanahan and Hofer 2011). Interestingly enough, in most of these studies the genetic variable, unlike the environmental variable, has little if any main effect on the outcome of interest. Rather, its influence is largely through its moderation of the environmental variable of interest (Rutter et al. 2006). Thus such research does not challenge the importance of environmental factors in determining human behavior; rather it shows how social scientific explanations might be made more precise by incorporating genetic information (Guo et al. 2008, 2009; Shanahan et al. 2008).

Genetically informed social science requires models of the manner in which genetic variables combine with environmental context to influence behavioral outcomes (Freeze 2008; Shanahan and Hofer 2005, 2011). The model utilized in the vast majority of G×E studies focuses upon the extent to which particular alleles (variants of a gene) amplify the probability that exposure to some adverse social condition (e.g., abusive parenting, stressful life events) will lead to a problem behavior (e.g., crime, depression, substance abuse, school dropout). In psychology and psychiatry, this is labeled the diathesis-stress perspective. This approach assumes that some individuals are by nature more vulnerable than others as they possess dysfunctional “risk alleles” that foster maladjustment in the face of deleterious environmental conditions. This assumption is contradicted, however, by the fact that over the past several thousand years evolution seems to have conserved these various alleles (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & Van Ijzendoorn 2011). While truly dysfunctional genetic variants should largely disappear over time, most of the so called risk alleles studied by behavioral science researchers are highly prevalent, often being present in 40 to 50 percent of the members of the populations being investigated (Ellis et al. 2011). Thus contrary to the negative view usually taken of these alleles, this suggests that, at least in certain contexts, these genetic variants must provide advantages over other genotypes. This idea is an essential component of the alternative model of gene by environment interaction recently proposed by Jay Belsky and his colleagues (Belsky, Bakermans-Kranenburg, and von IJzendoorn 2007; Belsky and Pluess 2009; Ellis et al. 2011).

After reviewing scores of studies that purported to show evidence of a diathesis-stress effect, Belsky and company (Belsky et al., 2007; Belsky and Pluess 2009) concluded that a careful inspection of the data actually points to a different interpretation. Rather than showing that some individuals are more vulnerable to stress than others, they asserted that the data supports the idea that some people are simply genetically predisposed to be more susceptible to environment influence than others. This suggests 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 et al. 2007).

The idea that some persons are genetically predisposed to be more responsive to their environment than others would seem to have particular relevance to sociologists. The present study tests several implications of this proposition. Using longitudinal data from a sample of several hundred African Americans, we examine the manner in which functional polymorphisms in the dopamine receptor gene (DRD4) and the serotonin transporter gene (5-HTT) moderate the effects of both positive (supportive parenting, religious participation, neighborhood informal social control, school involvement) and negative (harsh parenting,

racial discrimination, neighborhood victimization, violent peers) social conditions on involvement in aggressive behavior.

FOCUS OF THE PRESENT STUDY

Data from a variety of sources indicate that aggression and violence are much more prevalent in the United States than in other wealthy countries (Messner and Rosenfeld 2007; Wikinson and Pickett 2009). For instance, the incidence of homicide, assault, and rape is several times higher in the United States than in most European countries. These findings underscore the importance of research on the causes of aggression and antisocial behavior. In the past few years, researchers concerned with aggression, like social scientists working in other areas, have attempted to make their theories more precise by incorporating molecular genetic variables (Guo et al. 2008; Rutter et al. 2006). Much of this research has focused upon functional polymorphisms in the dopamine receptor gene (DRD4) and the serotonin transporter gene (5-HTT). Although results are mixed regarding the main effects of these genes, several studies have reported that these genes interact with adverse social environments to increase the probability of aggressive and antisocial behavior. These findings are almost always interpreted within a diathesis-stress framework.

Our research extends prior gene by environment investigations of aggression in several ways. First, we test the differential susceptibility hypothesis by examining the extent to which individuals with particular variants of the dopamine receptor gene and the serotonin transporter gene show higher rates of aggression than the comparison group when they grow up in an adverse social environment but lower rates of aggression than the comparison group when the social environment is favorable. In performing these analyses, we utilize a more comprehensive measure of the social environmental than has been used in most G×E research. In most cases the focus has been upon childhood aggression and the only environmental condition considered has been abusive parenting. In those studies where adult behavior is the outcome, retrospective reports have often been utilized to assess past exposure to adversity. In contrast, the present study employs a composite measure of both environmental adversity and environmental support assessed across the adolescent years to predict aggression during early adulthood.

Further, we test models that incorporate cognitive schemas and emotional states that past research has shown to mediate the effect of the social environment on increased involvement in aggression. We investigate whether social environmental differences interact with genetic variability to influence development of these schemas and emotional states in a manner predicted by the differential susceptibility hypothesis. Our analyses also examine the extent to which the effect of the interaction of social environment and genotype on aggression is fully mediated by these variables. We are not aware of any studies that have investigated the extent to which cognitive and emotional factors mediate gene by environment effects on aggression.

We begin by using extant theory and research to formulate a general model of aggression. The model posits that various social environmental conditions give rise to emotional traits and cognitive schemas that encourage aggression. We expect this model to be corroborated by the data, but that is not the primary concern of the study. Rather, our purpose is to investigate the extent to which genetic polymorphisms moderate the effect of social environmental variability on emotional traits, social schemas, and aggression in a manner consistent with the differential susceptibility hypothesis.

A GENERAL MODEL OF AGGRESSION

Much of sociology assumes a life course paradigm wherein social experiences give rise to schemas and sentiments that, in turn, influence one's interpretation and response to subsequent situations (Shanahan and Macmillan 2008). We use this basic perspective in our attempt to integrate the extant research on aggression into a general model that might be used to test the differential susceptibility approach. A test of the differential susceptibility hypothesis requires that one consider the impact of the full range of social environmental conditions, from very negative to very positive. Turning to the literature on aggression, the various strain theories (Agnew 2006) identify adverse social environments that cause aggression and violence, whereas social control theories (Hirschi 1969; Sampson & Laub 1993) specify positive social conditions that discourage aggression and violence. We briefly review these two theoretical perspectives and show how they can be integrated into a general model of aggression that takes into account the full range of environmental conditions, from very favorable to very adverse.

Strain Explanations

Strain theories of aggression and violence identify recurrent exposure to adverse circumstances, especially persistent social interactions involving exploitation or mistreatment, as a root cause of aggressive actions. These theories differ, however, regarding the schemas and sentiments that link mistreatment to aggression. In general, the theories emphasize either feelings of anger and frustration, a hostile view of people and relationships, or concern with projecting an image of toughness. We briefly discuss each of these traditions.

Feelings of anger and frustration are a central component of Agnew's (2006) General Strain Theory and Berkowitz's (1990) revision of the frustration-aggression hypothesis. These theorists argue that aversive social relations engender anger and irritability, and that these feelings increase the risk of aggression because they foster belligerence and explosiveness, lower inhibition and concern with negative consequences, and create a desire for retaliation and revenge. Several studies have demonstrated that feelings of anger increase the probability that an individual will engage in aggressive behavior (Agnew 2006; Berkowitz 1990) and that anger mediates a significant proportion of the association between aversive social relationships and aggression (Jang and Johnson 2003; Mazerolle, Piquero, and Capowich 2003; Simons, Chen, Stewart, and Brody 2003; Simons et al. 2006). In most cases the adverse social relationships assessed in these studies involved abusive parenting, difficulties with peers, criminal victimization, or racial discrimination.

A second group of researchers has focused upon the linkages between mistreatment, a hostile attribution bias, and engaging in aggression (Dodge, 1986; Slaby and Guerra 1988). Persons with a hostile attribution bias possess a cynical, distrusting view of people and relationships. They expect to be treated unfairly and believe that they must be prepared to defend themselves in order to avoid mistreatment. Research has shown that this view of relationships is strongly held by aggressive children and adolescents. Indeed, a meta-analysis of over 100 studies reported a robust association between a hostile view of others and youth aggression (Orbio de Castro et al. 2002); moreover, antisocial adults also demonstrate this cognitive bias (Bailey and Ostrov 2007). Research on the factors that give rise to this view of relationships has tended to focused upon abusive parenting and difficulties with peers (Dodge et al. 1990). However, racial discrimination has also been shown to increase the chances of developing this cognitive schema (Simons et al. 2003, 2006).

Finally, several researchers have argued that individuals who experience chronic exploitation and mistreatment tend to develop a concern with projecting an image of toughness (Anderson 1999; Gilligan 2001; Jacobs and Wright 2006). In most contexts, people are rewarded for reputations that involve being trustworthy, kind, and generous (Hauser 2006). In an oppressive environment characterized by mistreatment and abuse, however, such an identity generates little respect and may even invite exploitation. In these milieus, respect and cooperation are obtained by projecting an image of toughness, by communicating a willingness to fight if there is any indication of unfair treatment (Anderson 1999). To let transgressions go unchallenged, even small ones, demonstrates that one is soft and weak and exposes one to future predation and exploitation. Consonant with these arguments, a variety of studies have reported an association between persistent exposure to violence and maltreatment and concern with a tough reputation (Anderson 1999; Jacobs and Wright 2006; Stewart and Simons 2006). Indeed, Anderson (1999) observed that adolescents often create altercations with the intention of building respect and letting others know they are not a chump. Similarly, Wilkinson (2001) found that young men committed robberies as a way to build or maintain a tough reputation, and Kubrin and Weitzer (2003) reported that offenders feel that they have to retaliate in response to insults or risk losing the respect of their peers.

Summarizing, the three most widely accepted strain perspectives on aggression agree that persistent mistreatment is a primary cause of aggressive behavior. Consonant with this view, a multitude of studies have reported that persistent exposure to social conditions such as harsh parenting, racial discrimination, criminal victimization, and violent peers increases the probability that an individual will engage in violence. Further, there is considerable empirical support for the mediating psychological mechanisms emphasized by each of these frameworks. Research has shown that anger, hostile view of relationships, and concern with tough reputation mediate much of the effect of mistreatment on perpetration of aggression and violence.

Social Control Perspectives

While strain theories are concerned with the social circumstances that promote violence, social control theories strive to identify social factors that reduce the probability of aggression and other forms of social deviance (Kornhauser, 1978). Control theories are endorsed by a higher proportion of criminologists and have generated more research than any other type of criminological theory (Ellis and Walsh, 1999). The perspective asserts that social bonds in the form of attachments to prosocial people and involvement in conventional activities decrease the probability of deviant behaviors such as aggression and violence (Hirschi, 1969; Sampson and Laub, 1993). The theory contends that this is because engaging in such deviant behavior jeopardizes future involvement in these valued relationships and activities. Consistent with this view, past research has demonstrated that factors such as supportive parenting, religious participation, involvement in school activities, and informal community social control operate to deter involvement in aggression as well as other deviant behaviors (see Akers & Sellers, 2010; Sampson, 2006).

Social control theorists have largely ignored the attitudinal and emotional consequences that are likely to be fostered by involvement in conventional relationships and activities. These social bonds might be expected, however, to color an individual’s relational schemas and sentiments. Indeed, such psychological outcomes might be expected to mediate much of the impact of conventional relationships and activities on the probability of aggression. Consonant with this view, Simons and Burt (2011) recently presented data showing that social control processes deter involvement in deviant behavior by discouraging the same cognitive schemas and emotions that are promoted by adverse social circumstances. Thus conventional relationships and activities foster a positive view of people and relationships

(rather than a hostile view of others), emphasize the importance of prosocial strategies for influencing others (rather than acting tough), and promote positive emotions (rather than anger).

If this is the case, we can combine the strain and social control perspectives to form an integrated model of aggression. This model is depicted in Figure 1. The model focuses upon a range of both supportive and adverse environmental conditions that might be experienced during the adolescent years. Informed by social control theory, we operationalize favorable environmental conditions as supportive parenting, religious participation, school involvement, and informal community control, whereas our measures of adverse environmental conditions involve variables emphasized by strain theories such as harsh parenting, criminal victimization, racial discrimination, and violent peers. Our integrated model suggests that persistent exposure to either favorable or adverse social environmental conditions predicts the extent to which an individual develops the psychological characteristics that encourage aggression and violence

In the interest of parsimony, but also because we believe that it makes good theoretical sense, the model treats these three psychological characteristics as indicators of a latent construct that we have labeled hostile orientation. Our rationale for this decision is as follows. First, there is good reason to believe that these psychological factors are connected and intercorrelated. They are a function of the same set of social conditions and, more importantly, they are likely to be mutually reinforcing. A hostile view of relationships, for example, is apt to foster anger and concern with a tough reputation, and anger is liable to encourage a concern with toughness and suspicion regarding the motives of others. Second, it is not any one belief or feeling that predicts an individual's actions in a situation; rather it is the dynamic interplay of the constellation of relevant schemas and emotions that is important (Bourdieu 1984; Mischel and Shoda 1995). Thus it is the combination of the three psychological mechanisms, and not any one element by itself, that is likely to be important in predicting aggression. Our model indicates that persistent exposure during the adolescent years to an environment that is high on adversity and low on social control fosters development of a hostile orientation which, in turn, increases the probability that situations will be defined in a manner conducive to aggression. While we expect our analyses to corroborate this model, our more fundamental goal is to examine the extent to which genetic variation moderates the paths in the model in the manner predicted by the differential susceptibility hypothesis.

GENETIC MODOERATION: VULNERABILITY VERSUS DIFFERENTIAL SUSCEPTABILITY TO CONTEXT

The genetic code is composed of nucleotide base pairs (bps) that are organized into genes. Genes represent segments of the genome that contribute to particular phenotypes or functions. Many genes are polymorphic in that their structure varies somewhat across individuals. Each variant is labeled an "allele." One type of variation involves the number of times that a particular set of base pairs is repeated. This type of variability is referred to as a Variable Number Tandem Repeat (VNRT). VNTRs are important as they often alter the product of the gene if they occur in the coding region or they may influence the amount of the product if they occur in the promoter region.

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. Studies of the serotonergic system have concentrated on polymorphisms in the serotonin transporter gene (5-HTT). 5HTT is a key regulator of serotonergic neurotransmission, localized to 17p13 and consisting of 14 exons

and a single promoter. A polymorphism in the promoter region of the gene results in two major variants, a short and a long allele, with the short allele resulting in lower serotonin transporter availability (Lesch et al. 1996). Individuals inherit two copies of a gene, one from their mother and one from their father. Most studies of the serotonin transporter gene distinguish between those carrying at least one short allele and those with two copies of the longer allele.

Although much of the research on 5-HTTLPR alleles has concentrated on depression and anxiety, several investigations have also examined associations with aggression and conduct problems. Some of these studies have found a relationship between the s-allele and antisocial behavior (Lyons-Ruth et al. 2007) whereas others have failed to find this relation (Sakai et al. 2007). 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. For example, 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. (2010) found the longitudinal association between racial discrimination and adolescent conduct problems to be strongest among males with the s-allele of 5-HTTLPR.

Studies of aggression concerned with the dopaminergic system have concentrated on the dopamine D4 receptor (DRD4). This gene contains a 48bp repeat polymorphism in exon III of chromosome 11. The number of tandem repeats range from 2 to 11, and studies usually distinguish between a long (l-DRD4; 6–8 repeats) and a short polymorphism group (s-DRD4; 2–5 repeats). Evidence suggests that the longer allele codes less efficiently 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 indicating that the long allele interacts with aversive environmental conditions such as non-optimal parenting to increase the risk of conduct problems (Bakermans-Kranenburg and van IJzendoorn 2006; Propper et al. 2007).

Summarizing, several studies indicate that individuals with either the 5-HTTLPR s-allele or the DRD4 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 et al., 2007; Belsky and Pluess 2009) 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 and DRD4 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 et al., 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 and Pluess 2009; Ellis et al., 2011). Recently, Belsky and Pleuess (2009) reviewed scores of G×E 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 5HTT or DRD4.

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 sensation seeking and the serotonergic system which has been linked to sensitivity to punishment and displeasure (see Carver et al., 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 papers have reported support for this idea (Belsky & Beaver, 2011; Belsky, Pluess, Comings, and MacMuray 2011; Simons et al., in press).

The present study provides a stringent test of the susceptibility hypothesis by examining several of its implications as they apply to the model of aggression proffered in the previous section. First, past gene by environment studies of aggression have largely limited their focus to one type of mistreatment – abusive parenting during the childhood years. We extend this research by examining adolescent exposure to composite measures of both an unfavorable (harsh parenting, racial discrimination, criminal victimization, and violent peers) and favorable (supportive parenting, school involvement, religious participation, informal community control) social environment conditions. We investigate the extent to which the effect of our composite measures on adult aggression is moderated by genotype in the manner predicted by the environmental susceptibility hypothesis. By focusing upon social environmental conditions across the adolescent years, we explore whether genetic differences in environmental sensitivity extend beyond childhood.

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 and s-allele 5HTTLPR. Since these two 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. A few studies have reported that l-allele DRD4 and s-allele 5HTTLPR interact to increase the probability of antisocial behavior (Hohmann et al. 2009) whereas others have failed to find this effect (Oades et al. 2008). Such contradictory findings would be expected if the consequence of possessing this genotype varies by environmental context. 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 recurrent maltreatment, but lower levels of aggression than the comparison group when the environment is benign or supportive. The present study tests this idea.

Third, we go beyond prior studies by testing models that include cognitive schemas and emotional states that have 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. Consonant with this view, Simons et al. (in press) recently reported that young African American males with various "risk alleles" were more likely than other genotypes to

adopt the code of the street when they grew up in a dangerous social environment but were less likely than other genotypes to adopt the street code when they were raised in a more conventional social milieu. Applying this reasoning to the general model of aggression presented in Figure 1, one would expect individuals wit h a combination of l-allele DRD4 and s-allele 5HTTLPR to score higher than others on anger, hostile view of relationships, and concern with toughness, as well as on the latent construct hostile orientation, when they grow up in an adverse social environment. On the other hand, individuals with this genotype would be expected to score lower than others on these variables when they are raise in a favorable social environment. 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 social environment and genotype on aggression is mediated by the latent construct hostile orientation. As just noted, the differential susceptibility perspective would suggest that genotype moderates the probability that social environmental variability will result in the development of a hostile orientation. As shown in Figure 1, our general model posits that a hostile orientation mediates the effect of adolescent exposure to low social control/high adversity on perpetration of aggression during early adulthood. Elaborating this model to include genetic effects, we expect that the interaction of genotype and social environment on aggression will be fully mediated by hostile orientation. In other words, we expect hostile orientation to operate as a mediated moderator. We are not aware of any studies that have investigated the extent to which cognitive and emotional factors mediate the effect of G×E on aggression. Such effects would be expected if the differential susceptibility argument is correct.

RESEARCH DESIGN

Sample

Our research utilizes the five waves of data that have been collected for the Family and Community Health Study (FACHS), a multi-site (Georgia and Iowa) investigation of neighborhood and family processes that contribute to African American children's development in families living in a wide variety of community settings (see Gibbons et al. 2004; Simons et al. 2002). The FACHS sample consists of several hundred African American families living in Georgia and Iowa at the initiation of the study but the children. Each family included a child who was in 5^{th} grade at the time of recruitment. Details regarding recruitment of the sample are described by Gibbons et al. (2004), Simons et al. (2002), and are available in the ASR Supplemental Materials website.

The first wave of the FACHS data were collected in 1997–1998 from 889 African American, fifth-grade children (411 boys and 478 girls). 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. By wave 5, the respondents were scattered throughout the United States. Of the 889 targets interviewed at Wave 1, 779 were reinterviewed at Wave 2, 767 at Wave 3, 714 at Wave 4, and 689 at Wave 5 (78% of the original sample).

Analyses indicated that those individuals who did not participate in waves 2, 3, and 4 did not differ significantly from those who participated with regard to youths' age, sex, or participation in delinquency or primary caregivers' education, household income, or neighborhood characteristics. Respondents who dropped out after the fourth wave, however, differed in a few ways from those in the first 3 waves. A higher percentage of those interviewed at wave 5 were female, and, not surprisingly, engaged in slightly less delinquency (diff = −.51, t =−1.97) on average than those not re-interviewed at Wave 5.

There were no differences between those remaining in the panel and those dropping out with regard to community measures, family structure, or parenting practices.

As part of wave 5 data collection, targets were asked to provide DNA (saliva sample) for purposes of genetic analyses. Of the 689 participants, 549 (80%) agreed to do so. Successful genotyping for both 5-HTTLPR and *DRD4* was achieved for 505 individuals. Analyses did not identify any significant differences in terms of caregivers' education, household income, family structure, neighborhood characteristics, age, gender, or antisocial behavior between those who provided DNA samples and those who chose not to participate.

Procedures

The questions 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. Many of the instruments administered in wave 5 include questions regarding illegal or potentially embarrassing sexual activities. Hence, in an effort to further enhance anonymity, we used 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

Aggression, the outcome used in our analyses, as well the three mediators - hostile view of relationships, anger, and reputation for toughness – were assessed at both waves 1 and 5. We used data obtained at waves 1 – 4 to assess favorable and adverse social environmental conditions. The DNA samples used to genotype DRD4 and 5-HTTLPR were obtained at wave 5. The specific measures are described below.

Aggression

Aggression 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; Shaffer et al., 1993; 2000). Respondents reported whether in the past year they had engaged in 11 aggressive behaviors (1 = yes; 0 = no) such as cruelty to animals, damaging property, fighting with weapons, and hurting another. 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 or she had engaged in all of the different acts. Coefficient alpha for the instrument was .76.

Favorable Social Environment

Our measure of favorable social environment focused upon social ties and activities emphasized by social control theory. This construct was assessed using a composite measure consisting of four components: supportive parenting, school involvement, religious participation, and neighborhood informal social control.

a. *Supportive parenting* was measured at waves 1 – 4 using target responses to nine items concerned with caregiver warmth, monitoring, and problem-solving during the previous 12 months. Response categories ranged from 1 (never) to 4 (always). In addition, based upon prior work (Guo et al. 2008), we included two items that asked how often (1=never, 4=every day) the family eats a meal together, and how often the caregiver helps the target youth with his or her homework. Coefficient alpha for this scale was .72 at wave 1, .76 at wave 2, .80 at wave 3, and .85 at wave 4. A composite measure of supportive parenting was created by standardizing and averaging the scores across waves.

b. *School involvement* was assessed using nine items (Brody et al. 2006) that asked the target youths to indicate how much they agree (1=strongly disagree, 4=strongly agree) that various statements describe themselves. The statements referred to having positive relationships with teachers, completing homework, interest in school, involvement in school activities, and academic performance. Coefficient alpha for the scale was .74 at wave 1, .73 at wave 2, .74 at wave 3, and .75 at wave 4. A composite measure of school involvement was obtained by standardizing and averaging scores across waves.

c. *Religious participation* was assessed using four items that asked respondents to report how often (1=never, 5=daily) during the past month they participated in religious activities, including church services, social events at church, Sunday school or discussion groups on religion, and go places with friends from church. Coefficient alpha for the scale was .76 at wave 1, .79 at wave 2, .85 at wave 3, and . 87 at wave 4. Scores were standardized and then averaged across waves to form a composite measure of religious involvement.

d. The final measure of favorable social environment, *neighborhood informal social control*, was assessed with a revised version of neighborhood monitoring from the Project on Human Development in Chicago Neighborhoods (PHDCD; Sampson et al. 1997). The respondents were asked to rate (1 = very unlikely, 4 = very likely) the probability that neighborhood residents would intervene (issue a verbal reprimand, call the police) if teens spray-painted graffiti on a building, showed disrespect to an adult, or skipped school and were hanging out on a street corner. Scores were standardized and then averaged across waves to form a composite measure of neighborhood informal social control. Coefficient alpha for this four-item scale was .54 at wave 1, .51 at wave 2, .63 at wave 3, and .73 at wave 4.

Confirmatory Factor Analysis utilizing the four composite measures used to assess favorable social environment resulted in factor loadings that ranged from .52 for neighborhood social control to .76 for supportive parenting. The factor scores were used to form a global assessment of persistent exposure to a favorable social environment across the adolescent years.

Adverse Social Environment

Based upon research on strain theory, our measure of social environmental adversity included four components: harsh parenting, racial discrimination, neighborhood victimization, and violent peers.

a. The *harsh parenting* scale consisted of 10 questions regarding how often during the past year that the primary caregiver engaged in physical (e.g., How often did your mother push, grab, hit or shove you? How often did your mother slap or hit you with her hands?) and verbal hostility (How often did your mom insult or swear at you? How often did your mom yell at you?) when disciplining the respondent. The response format for the items ranged from 1 (never) to 4 (always). This scale has been shown to have high validity and reliability (see Simons et al. 2006, 2007). Scores were standardized and then averaged across waves 1 – 4 to form a composite measure of persistent exposure to harsh parenting. Coefficient alpha for the measure was .70 at wave 1, .76 at wave 2, .76 at wave 3, and .83 at wave 4.

b. *Racial discrimination* was assessed at waves 1–4 using 13 items from the Schedule of Racist Events (Landrine and Klonoff 1996). 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 above .90 at each wave. Scores were standardized and then averaged across waves to form a composite measure of persistent exposure to discrimination.

c. *Neighborhood victimization* was assessed at waves 1 – 4 using the following questions (see Stewart, Schreck, and Simons 2006): During the past year, has anyone in the neighborhood surrounding your house ever used violence, such as mugging, physical attack, or sexual assault, against you? During the past year, has anyone in the neighborhood surrounding your house ever used violence, such as mugging, physical attack, or sexual assault, against one of your friends? Respondents who responded affirmatively were asked to report how many times such events had occurred. At each wave, 10 to 17 percent of the respondents reported that they or their friends had been the victim of at least one violent act in their neighborhood during the preceding year. Scores were standardized and then summed across waves to form a composite measure of persistent exposure to violent victimization.

d. Involvement with *violent peers* was measured using a six-item scale (Stewart and Simons 2010). The items asked respondents to report how many (1=none; 5=all) of their close friends had engaged in various violent behaviors in the past year. The items focused upon acts such as fights, robbery, and threatening someone with a weapon. Scores were standardized and then averaged across waves. Cronbach's alpha, was .59 at wave 1, .50 at wave 2, .62 at wave 3, and .72 at wave 4.

Confirmatory Factor Analysis utilizing the four composite measures used to assess adverse social environment resulted in factor loadings that ranged from .54 for neighborhood victimization to .74 for violent peers. The factor scores were used to form a global assessment of persistent exposure to social environmental adversity across the adolescent years.

Favorable/Adverse Social Environment

In order to obtain a global measure of the social environment that ranged from very favorable to very adverse, we reverse coded the favorable social environment measures, and then standardized and summed the favorable and adverse social environmental scales. Low

scores on this measure indicated low adversity in a favorable social environment whereas the high end equals high adversity in an unfavorable social environment. The Nunnally (1978) reliability formula was used to assess the reliability of this composite measure. Building on classical testing theory, the Nunnally technique utilizes information regarding the internal consistency of each of the scales being combined to determine the reliability of the new aggregate measure. Using this procedure, the reliability of our composite measure of favorable/adverse social environment was .809.

Hostile View of Relationships

At waves 1 and 5, a 6-item scale developed for the current study (see Simons et al. 2003, 2006) was used to assess this construct (e.g., You have to use physical force or violence to defend your rights; Behaving aggressively is often an effective way of dealing with someone who is taking advantage of you; You have often been lied to). The response format ranged from 1 (mostly true) to 0 (mostly false). Coefficient alpha for the scale was .72 at wave 5.

Anger

At wave 5, this construct was assessed using seven items from the Spielberg Trait Anger Scale (Spielberg 1983) which is designed to measure relatively enduring feelings of anger and frustration. The items focus on how often the respondent is hotheaded, shows a quick or fiery temper, flies off the handle when criticized, is easily frustrated, and becomes irritated or mad. The response format for the items ranged from 1 (almost never) to 4 (almost always). Responses to seven items were summed to form the measure of anger. The coefficient alpha for this measure was .87. At wave 1, anger was assessed using 4 items from the DISC-IV (Simons et al., 2003, 2006). The items ask the respondent to report how often (1=less than once per week; 4=nearly every day) he or she loses his or her temper, feels grouchy or annoyed, gets mad, or feels unfairly treated. Coefficient alpha was .65.

Reputation for Toughness

A five item scale developed for the project (Stewart and Simons 2006, 2010) was used to assess this construct in waves 1 and 5. Respondents indicated how much they agreed (1= strongly disagree, 4=strongly agree) with the following statements: 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; Being viewed as tough and aggressive is important for gaining respect; It is important not to back down from a fight or challenge because people will not respect you; and, It is important to show courage and heart and not be a coward in a fight in order to gain or maintain respect. Coefficient alpha for the mean scale was .84 at wave 5. Only the first two of the five items were available in the wave 1 instrument. Thus, the wave 1 measure of reputation for toughness was the mean of those two items.

Hostile orientation

In the current study, hostile orientation is an unobservable latent construct assessed by the three indicators: hostile view of relationships, anger, and reputation for toughness. The intercorrelation of these three observed variables was quite high (r =.32 to .60, p<.001) and confirmatory factor analysis indicated that they loaded on a common factor with loadings above .65.

Control variables

To avoid overestimated results, we controlled for previous status of our outcomes. In addition, we included controls for gender, region, family SES, and family structure. Target gender was coded 1 = males (40.8%) and 0 = females (59.2%). Region was coded 1 for respondents living in the South (53.9%) and 0 or those living in other areas of the country.

Family SES was a composite measure based upon the primary caregiver's education and family income. Family structure was coded as a dummy variable: 1=single parent families (52.1%); 0= other family types (47.9%).

Genotyping

Genotype at DRD4 contains a 48bp repeat polymorphism in exon III of chromosome 11 and was determined for each youth as described by Lichter et al. (1993). Following genotyping of DRD4, individuals were classified into one of two groups (Dreber et al., 2009): 1= at least one 7 repeat allele (l-allele); 0 = no 7 repeats allele (s-allele). Using this criterion, 301 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). Based on previous studies, individuals were classified into two groups based upon 5-HTTLPR genotype: 1 = at least one s-allele (s-allele); 0 = both alleles long (16 or 18 repeats). Using this criterion, 231 individuals were classified as s-allele.

Finally, the two genotypes (DRD4, 5-HTTLPR) were summed to form a measure of cumulative plasticity alleles. Respondents with the s-allele DRD4 and l-allele 5HTTLPR received a score of 0, those with either the l-allele DRD4 or s-allele 5HTTLPR received a score of 1, and those with both the l-allele DRD4 and the s-allele 5HTTLPR received a score of 2. This resulted in 140 individuals receiving a score of 2. The frequency distribution for the various combinations of the polymorphisms of 5-HTTLRP and DRD4 are shown in Table 1.

Among the 505 respondents in the sample, 6.9% were homozygous for the short allele (*ss*) at 5HTTLPR, 38.8% were heterozygous (*sl*), and 54.3% were homozygous for the long allele (*ll*). The distribution of the DRD4 polymorphisms were: 3.4% (*ll*), 36.9% (*sl*), and 59.7% (*ss*). Using the Hardy-Weinberg equilibrium test, the observed distribution of *DRD4* and 5HTTLPR did not differ significantly from that predicted on the basis of simple Mendelian inheritance.

ANALYTIC STRATEGY

Hierarchal regression models were run using Mplus 6.01 (Muthen and Muthen 2010) statistical software in order to test for the main and interactive effects of the social environment and genotype on aggression, as well as on the three potential mediators - hostile view of relationships, anger, and toughness. Because missing data might influence our findings, we used a multiple imputation (MI) techniques for missing data at the item level. Two-way interaction terms were used to test for gender differences in the effect of the social environment and of genotype. In order to have a common scale, this study employs standardized regression weights in which all independent variables were standardized (a mean of 0 and a standard deviation of 1) before the interaction terms were calculated. Some benefits of the standardized weights in the interaction model include making coefficients easier to interpret, reducing multicollinearity, and making the simple slope easier to test (Dawson & Richter, 2006). Post hoc analyses of significant interaction terms were conducted using the Johnson-Neyman (J-N) technique (Hayes and Matthes 2009). This procedure identifies regions of significance for interactions between continuous (social environment) and categorical variables (genotypes).

Next, we employed the mediated-moderation model available in Mplus 6.01 (Muthen and Muthen 2010) to examine the extent to which the latent construct hostile orientation mediates the main effect of social environment and, more importantly, the interaction of

social environment and genotype, on aggression. The mediated-moderation model simultaneously combines traditional moderation and mediation models (Muller et al. 2005). In this context, the interaction effect between two independent variables (G × E) is indirectly related to a dependent variable (aggression) through a mediating variable (a hostile orientation), and the effect of the interaction term on the dependent variable is significantly reduced. The logic of the mediated-moderation model is similar to traditional mediation models, but this model focuses only on the relationship among an interaction term, a mediator and an outcome, rather than considering the effects of multiple independent variables (Muller et al. 2005). Unlike traditional mediating theory using three steps to test for mediating effects (Barron and Kenny 1986), the bootstrapping option in Mplus determines the significance of mediation effects (see Mallinckrodt et al. 2006) and enables one to examine all direct and indirect effects (Preacher et al. 2007).

Given that aggression 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 present results that utilize 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. It should be noted, however, that we obtained an identical pattern of results when we employed the negative binomial option.

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). An RMSEA smaller than .05 indicates a close fit, whereas an RMSEA between .05 and .08 suggests a reasonable fit (Browne and Cudeck 1992).1

RESULTS

Table 2 presents the means, standard deviations, and zero-order correlations for the study variables. Gene-environment correlation (rGE) refers to a non-random distribution of environments among different genotypes. Importantly, rGE is likely to confound genotype × environment effects (Caspi & Moffitt 2006; Guo et al. 2008). Table 2 shows that there are no significant correlations between favorable/adverse social environment and either of our measures of genotype. This suggests an absence of rGE effects in the current study.2 As expected, favorable/adverse social environment is significantly associated with hostile view of relationships, chronic anger, belief in toughness, and aggression. The genotype variables, however, are not significantly related to any of these variables. Further, the table reveals that the three mediator variables – hostile view of relationships, chronic anger, and belief and toughness - are highly intercorrelated and show significant associations with aggression.

The Effect of G × E on Hostile View of Relationships, Anger, Toughness, and Aggression

Table 2 presents hierarchal regression models using four outcomes: aggression and the three variables identified in aggression theories as mediators. For each of these outcomes, Model

[1]The data utilized in the analyses, coding syntax, and statistical commands are available upon request.

[2]We used three approaches to rule out potential confounds between G-E interaction (G×E) and G-E correlation (rGE). First, there was no significant relationship between parental genotype (DRD4+5HTT) and child aggression ($r = .03$, $p > .05$). This finding suggests the absence of an evocative rGE effect on youth aggression. Second, the relationship between child genotype and the measure of social environment was not statistically significant. Thus there is no indication that a respondent's genotype influenced his or her selection of social environment. Finally, there is no evidence for passive rGE effects as parental genotype was not associated with our measure of social environment.

A examines the effect of favorable/adverse social environment (E) and cumulative plasticity alleles, while controlling for various factors including earlier assessments of the outcome variable. Thus our focus is upon change in the outcome variable. These models show that E is significantly related to increases in all four of the outcomes whereas cumulative plasticity is not. Model B, for each of the outcomes, adds the interaction of E with cumulative plasticity alleles. As expected, these models show that that E interacts with cumulative plasticity alleles to predict increases in the three mediators – hostile view of relationships, chronic anger, and belief in toughness – as well as aggression. In the case of the three mediators, E continues to show a main effect after the interaction of E by cumulative plasticity alleles is entered into the equation.3

Finally, for each of the four outcomes, Model C provides a further test of the cumulative plasticity idea. This hypothesis posits that individuals with the combination of the s-allele 5-HTTLPR and l-allele DRD4 will show a stronger response to the environment than those possessing only one of these alleles. Three dummy variables were formed to test this contention. These variables were defined as follows: two plasticity alleles = l-allele DRD4 and s-allele 5-HTTLPR; DRD4 only = l-allele DRD4 without s-allele 5-HTTLPR; 5-HTTLPR only = s-allele 5-HTTLPR without l-allele DRD4. The reference category consisted of individuals with neither the l-allele DRD4 nor the s-allele 5HTTLPR.

As seen in Table 2, none of these dummy variables has a main effect on either aggression or the three mediators. Further, with only one exception, neither the l-allele DRD4 by itself nor the s-allele 5HTTLPR by itself shows a significant interaction with our composite measure of social environment (E). The exception is the interaction of s-allele 5HTTLPR with E in predicting belief in toughness. More importantly, however, for all four outcomes there is a significant interaction between E and having both the l-allele DRD4 and the s-allele 5HTTLPR alleles. Consonant with the argument of cumulative plasticity, this finding indicates that individuals with both of these alleles respond more strongly to variations in the social environment than those with only one of these alleles.

Having established interactions between cumulative genetic plasticity and E, the next step was to graph these interactions to see if there is evidence of the cross-over pattern predicted by the differential susceptibility argument. Figure 2a depicts this interaction when aggression is the outcome. The figure shows that the effect of E on aggression is strongest for persons with two genetic plasticity alleles, somewhat weaker for those with only one allele, and weakest for those with neither of the alleles. Analysis using the simple slope procedure (Aiken and West 1991) indicated that the slopes for respondents with either one or two genetic plasticity alleles are significantly different from zero, whereas the slope is not significantly different from zero for those with neither of the genetic plasticity alleles. More importantly given the focus of the present study, the graph demonstrates the crossing pattern predicted by the differential susceptibility hypothesis. When the social environment is highly favorable, individuals with two plasticity alleles report less aggression than persons with the other two genotypes; when the social environment is adverse, those with two plasticity alleles report more aggression than persons with the other two genotypes.

The Johnson-Neyman (J-N) technique (Preacher et al., 2006) was used to assess the significance of these differences. This approach has the advantage of identifying turning points and confidence bands. The shaded area in each graph shows the area of significance. The graph show that cumulative genetic plasticity significantly ($p<.05$) increases an individual's aggression when E is greater than 1.70 standard deviations above the mean, and

[3]We also included a three-way interaction term that was utilized to examine gender or area differences in G × E effects. The findings showed that the interaction of social environment with cumulative plasticity does not differ by gender (1 = male) or area (1 = South).

it significantly decreases aggression when E is less than .830 standard deviations below the mean. Figures 2c – 2f show the results for hostile view of relationships, chronic anger, and belief in toughness, respectively. The expected crossing pattern is present in all of these graphs as individuals with two plasticity alleles are lower on hostile view of relationships, anger, and toughness than persons with the other two genotypes when E is roughly one standard deviation below the mean (i.e., the environment is high on social control and low on adversity) whereas those with two plasticity alleles are significantly higher on these outcomes than persons with the other two genotypes when E is approximately one standard deviation above the mean (i.e., the social environment is high on adversity and low on social control).

The Mediating Effect of Hostile Orientation

Our analyses to this point suggest that cumulative plasticity interacts with the social environment to predict aggression as well as the mediators of aggression identified by several widely accepted theories in the field. Further, the nature of this interaction is in keeping with the differential susceptibility hypothesis. Our last set of analyses was designed to test the extent to which the interaction of cumulative genetic plasticity and the social environment on aggression is mediated by the variables specified by the aggression theories. To perform these analyses, the three mediators – hostile view of relationships, toughness, and anger – were treated as indicators of the latent construct hostile orientation. In addition to simplifying the analysis, combining the variables into a single latent construct provided a measure of the cognitive and emotional consequences of social environmental experiences that was more comprehensive and had greater variance than that of the individual indicators.

Figure 3 shows the results of a simple mediator model without the genetic variables. The factor loadings for the latent construct indicate good construct validity and the fit indices indicate that the model fits the data well. The model shows that our composite measure of the social environment is associated with hostile orientation ($\gamma = .512$) which, in turn, is associated with aggression ($\beta = .406$). The indirect effect of the social environment on aggression through hostile orientation is significant ($p < .05$). Indeed, hostile orientation almost completely mediates the effect of the social environment, reducing the relationship between these two variables from .200 to .017.

Prior to adding the genetic variable to our model, we ran two reciprocal effects models using MPLUS in order to evaluate the causal priorities inherent in our theoretical model. First, we assessed our assumption that adverse environmental conditions cause aggression rather than aggressive individuals either selecting or fostering hostile environmental circumstances. The results (see Appendix A, online supplement), based upon analyses using data from waves 1 and 2, indicated that our composite measure of the social environment predicted aggression ($\beta = .562$) whereas the reverse was not the case ($\beta = -.04$). Second, we evaluated our assumption that a hostile orientation leads to aggression rather than aggression leading to a hostile orientation. The results (see Appendix B, online supplement) indicated that a hostile orientation predicts aggression ($\beta = .299$) whereas aggression has little impact upon hostile orientation ($\beta = .123$).

Having established support for the causal priorities assumed by our theoretical model, we ran a mediated moderator model in an effort to determine the extent to which the interaction of cumulative genetic plasticity and our composite measure of the social environment on aggression is mediated by hostile orientation. As shown in Figure 4, the model provided a good fit to the data. Examining the structural coefficients, favorable/adverse social environment has a significant main effect on hostile orientation ($\gamma = .295$), but cumulative genetic plasticity does not. The interaction of cumulative plasticity with favorable/adverse social environment is significantly related to hostile orientation ($\gamma = .279$). And, including

hostile orientation in the model reduces the association of the G×E interaction term with aggression from a significant .173 to an insignificant .059. Further, the indirect effect of this interaction term on aggression through hostile view of relationships is significant. This pattern of findings supports the hypothesis that the moderating effect of cumulative genetic plasticity on the association between favorable/adverse social environment and aggression is mediated by hostile orientation.

Lastly, as shown in Figure 5, we employed the J-N procedure to graph the effect of cumulative genetic plasticity × favorable/adverse environment on hostile orientation. The graph shows that cumulative genetic plasticity significantly ($p<.05$) increases an individual's hostile orientation when favorable/adverse social environment is greater than .076 standard deviations above the mean, whereas it decreases hostile orientation when favorable/adverse social environment is less than .547 standard deviations below the mean. These findings provide strong support for the differential susceptibility hypothesis.

DISCUSSION

In recent years, sociologists and other behavioral scientists have begun to incorporate genetic variables into their models. Shanahan and Boardman (2009: 234) describe the challenge as that of identifying "constellations of environmental factors that, through time, act in concert with genetic factors to make specific behaviors more or less likely." Such research requires models of the manner in which environments and genes "act in concert." 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 et al., 2007). Focusing upon two genotypes – s-allele 5HTTLPR and l-allele DRD4 - we tested various implications of the differential susceptibility model as they apply to the etiology of aggressive behavior. The results provided strong support for the perspective.

First, the differential susceptibility perspective asserts that the more plasticity alleles one carries, the more susceptible he or she will be to environmental context (Belsky & Pleuss 2009). Consistent with this expectation, we found that it was persistent exposure to a social environmental low on social control and high on adversity combined with having both s-allele 5HTTLPR and l-allele DRD4 that best predicted aggression, aggression related cognitive schemas, and chronic anger. Indeed, neither the l-allele DRD4 by itself nor the s-allele 5HTTLPR by itself showed a significant interaction with quality of social environment. Although a larger sample might have detected significant interactions for each of the individual alleles, our results clearly demonstrate that individuals with both alleles respond more strongly to social environmental influence than those with only one.

Second, graphs of the interaction between cumulative genetic plasticity and variability of social environment showed the crossover pattern indicative of differential susceptibility. Persons with both s-allele 5HTTLPR and l-allele DRD4 showed higher levels of aggression, anger, hostile view of relationships, and concern with toughness than those with other genotypes when exposed to a social environment low on social control and high on adversity. On the other hand, they showed lower scores on these variables than those with other geneotypes when they had experienced high social control and low adversity. This pattern of findings provides strong support for the idea that some individuals are

programmed by their genes to be more sensitive than others to environmental context, whether that influence be for better or worse (Belsky et al., 2007).

Finally, our results support the idea that the combination of social environment and genetic plasticity increases the probability of aggression because of the schemas and emotions that it engenders. Various theories of aggression identify chronic anger, hostile view of relationships, and concern with toughness as mediators of the association between maltreatment and 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 implicit in recurrent environmental events more quickly than less environmentally sensitive individuals. Consistent with this idea, respondents with a combination of l-allele DRD4 and s-allele 5HTTLPR scored higher than others on anger, hostile view of relationships, and concern with toughness, as well as on the latent construct hostile orientation formed from these three variables, when the social environment was high on adversity and low on social control. On the other hand, individuals with this genotype scored lower than others on these variables when the social environment was high on social control and low on adversity. Further, our results indicated that the interaction of genotype and social environment on aggression is mediated by the latent construct hostile orientation. 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 and the l-allele DRD4 are more sensitive to adverse treatment than those with other genotypes. These findings provide a fuller understanding of why some people respond to mistreatment with aggression whereas many individuals do not. Past research has shown, for example, that abusive parenting dramatically increases the chances that a child will grow up to be aggressive with their own children and yet the majority of abused individuals do not show this pattern (Kaufman and Zigler 1989). Findings from the present study suggest that it may be persons with the s-allele 5HTTLRP and l-allele DRD4 who are most like to show this intergenerational effect.

Importantly, however, our results indicate that this is only part of the story. Individuals at genetic risk for the highest rates of aggression in response to a biography of mistreatment are also genetically predisposed to show the lowest rates of aggression when they grow up in a benign environment. Those who are most likely to develop anger, a hostile view of people, a concern with toughness, and aggression in reaction to criminal victimization, parental mistreatment, racial discrimination, and violent peers are the same persons who are most likely to develop a peaceful, sanguine orientation in response to a kinder social environment characterized by social support, school involvement, religious participation, and informal social control. 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.

This idea is supported by recent intervention studies. Bakermans-Kranenburg et al. (2008) found, for example, that children with the l-allele DRD4 showed the largest decline in conduct problems in response to parent training. 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, and Beach, Brody, Lei, and Philibert (2010) reported similar findings for l-allele DRD4 and substance use. These interventions lasted

only a few months and provide support for the differential susceptibility hypothesis whereas they are contrary to the diathesis-stress perspective.

Of course, these interventions all focused upon children and adolescents. The schemas and behavior patterns of adults are apt to be much more obdurate and resistant to change. The schemas investigated in the present study developed slowly over the teen years and one would expect that it would take persistent exposure to new information over a rather protracted period of time to foster a change in these cognitive structures. Still, there is compelling evidence, including studies of previously incarcerated individuals, indicating that antisocial adults often adopt a more conventional outlook and life style in response to life changes such as marriage and employment (Laub and Sampson, 2003; Savolainen, 2009). The differential susceptibility perspective suggests that it is those with plasticity alleles who most likely to change in response to such new circumstances. We are aware of only one study that has provided evidence bearing on this idea. Consistent with the differential susceptibility perspective, Beaver, Wright, DeLisi, and Vaughn (2007) found that men with s-allele 5-HTTLPR and with l-allele DRD4 showed greater desistance from crime following marriage than other genotypes. Given the large number of longitudinal studies that have begun to collect genetic data, much more research regarding this issue is likely to be published in the near future.

Although our study improved upon many of the limitations of past gene by 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 African American sample had the benefit, however, of allowing us to investigate the manner in which social environmental variability and genotype combine to produce aggression among a racial/ethnic group who often experiences high levels of abuse and unfair treatment and manifests high rates of violence and antisocial behavior (Unnever et al. 2009). Further, while several past studies of differential susceptibility have focused on European and European American samples, there has been scant investigation of the hypothesis among African Americans. Given the strength of our findings, however, there is a need to examine the extent to which differential susceptibility influences the development of cognitions, emotions, and aggression in a similar fashion among other racial and ethnic groups.

In conclusion, sociology 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; Shanahan, 2010). 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. We focused upon two widely studied genes associated with the serotonergic and dopaminergic neurotransmitter systems. However, several other genes often included in gene × environment studies (e.g., DRD2, MAOA) are also involved in these systems. It may well be the case that what these genes have in common is that they influence thresholds for experiencing pleasure or displeasure and thereby enhance responsiveness to environmental events (Belsky and Pleuss 2009; Ellis et al., 2011). The fact that genetic data is now available in many of the large-scale social scientific data sets (e.g., Adolescent Health) means that sociologists 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.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research was supported by the National Institute of Mental Health (MH48165, MH62669), the Center for Disease Control (U01CD001645), the National Institute on Drug Abuse (DA021898, 1P30DA027827), the National Institute on Alcohol Abuse and Alcoholism (2R01AA012768, 3R01AA012768-09S1), and both the Center for Contextual Genetics and Prevention Science and the Center for Gene-Social Environment Transaction at the University of Georgia

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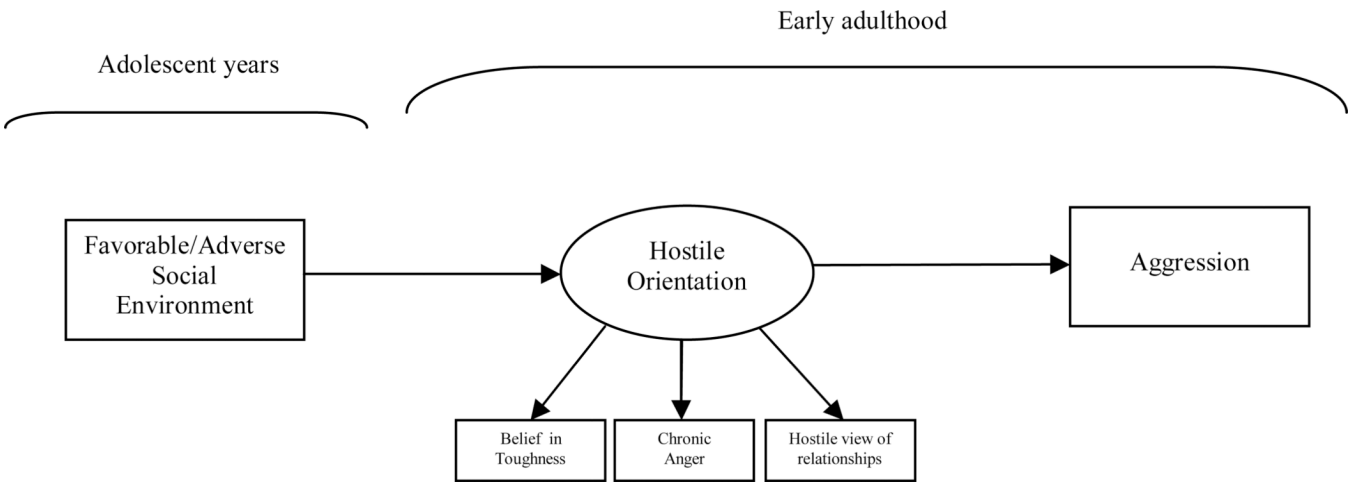
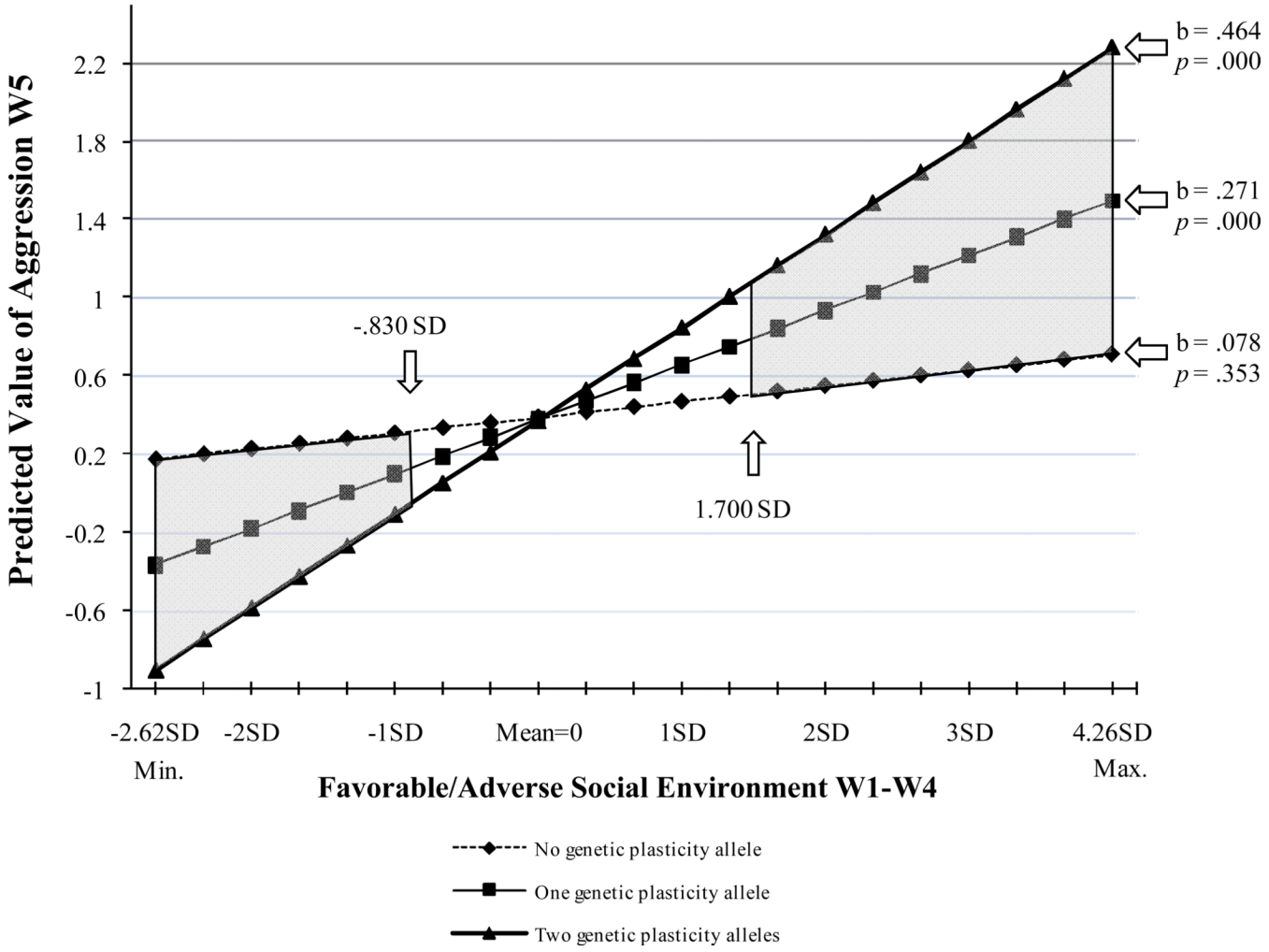


Figure 1.
Integrated Model of Aggression.

2A



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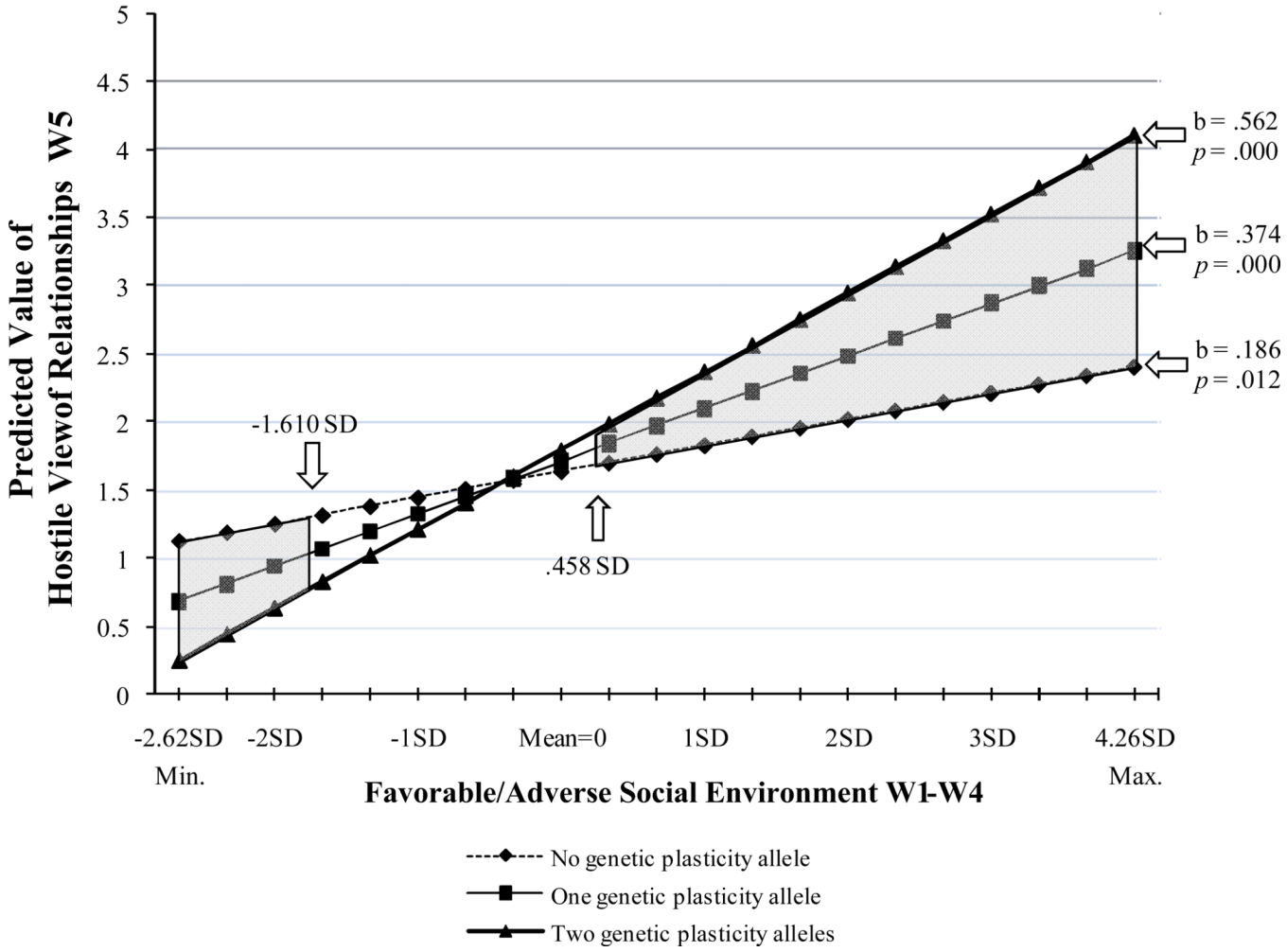

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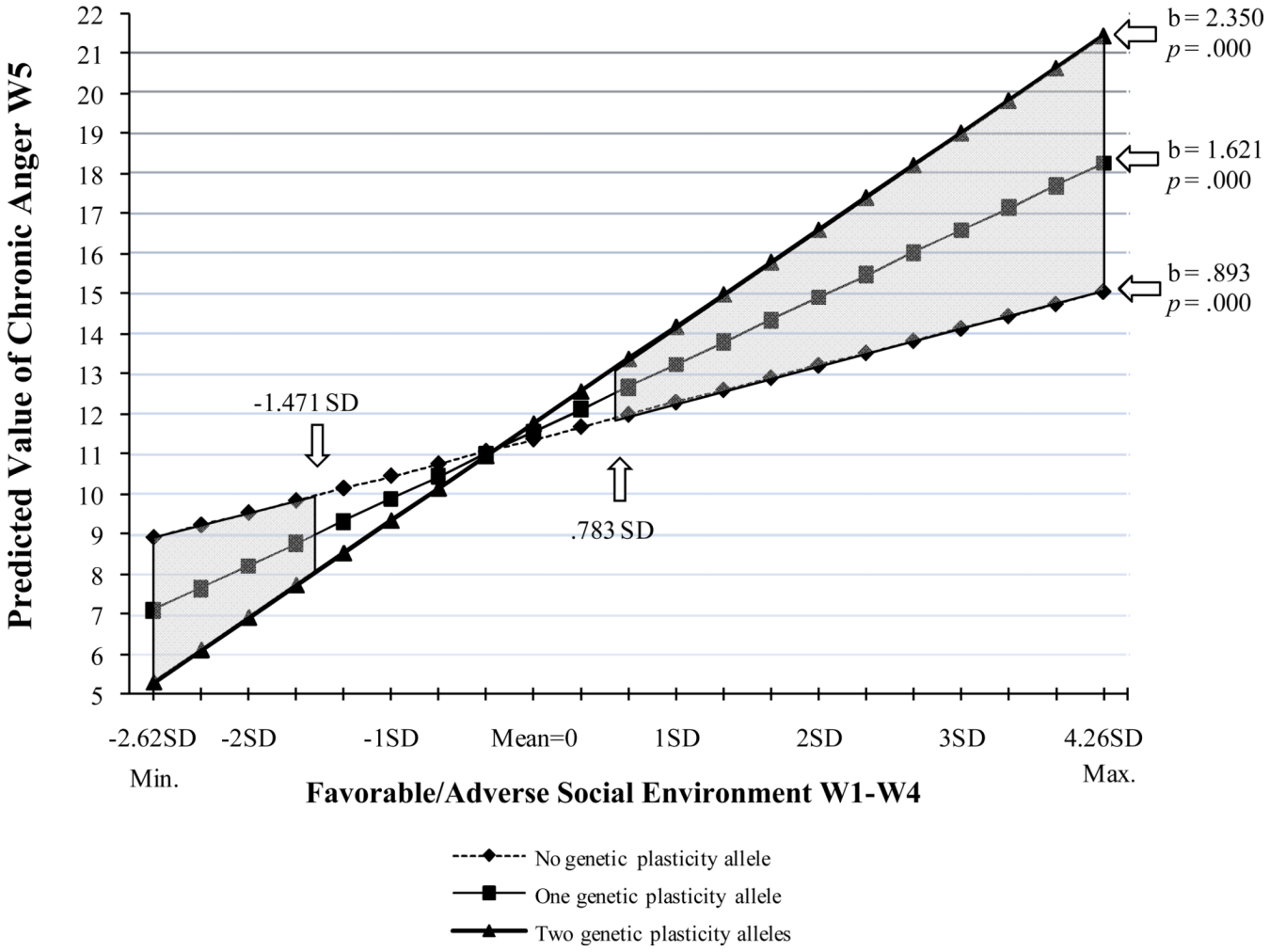

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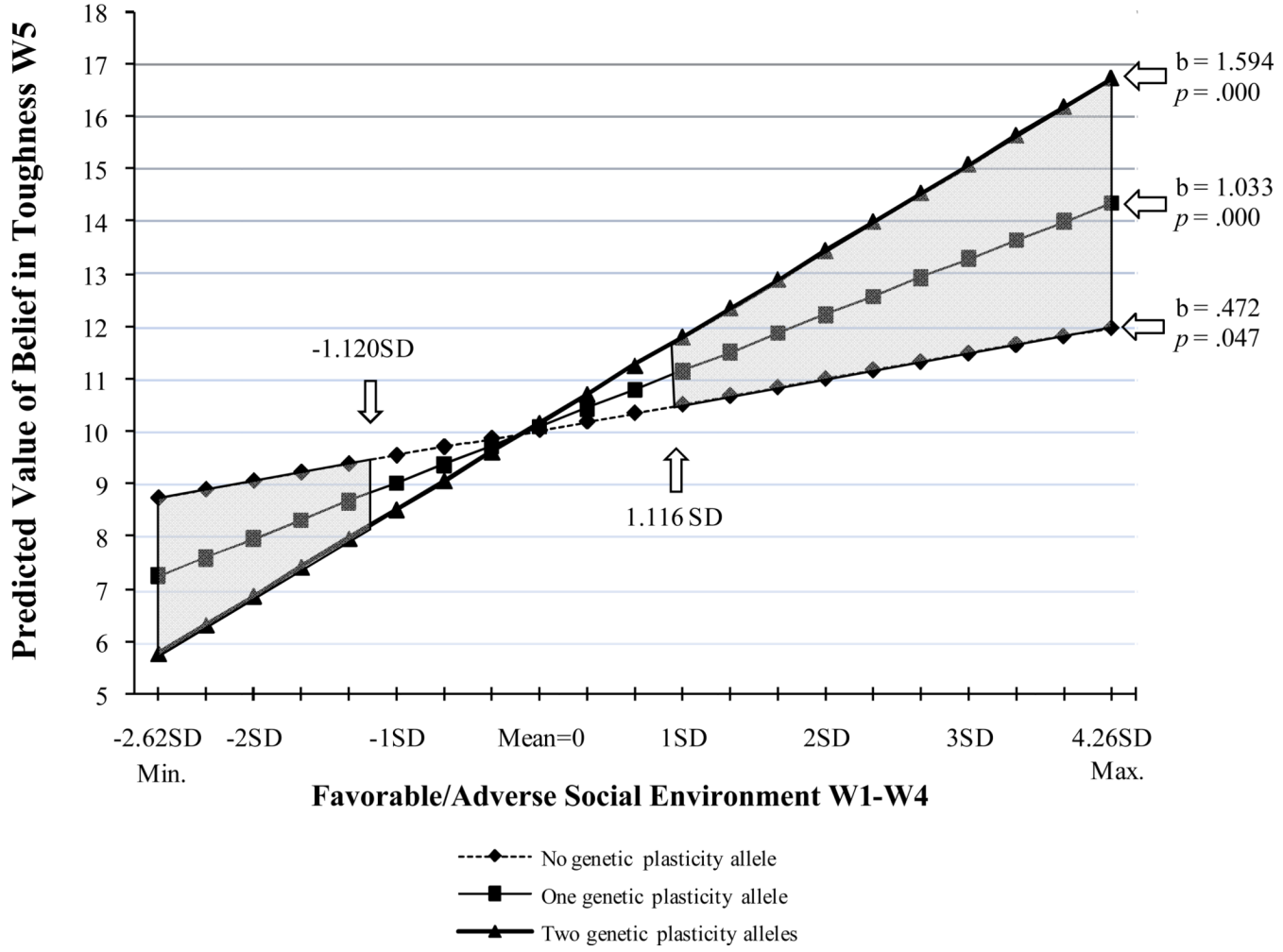

Figure 2.

A. The Effect of Favorable/Adverse Social Environment on Aggression by Number of Genetic Plasticity Alleles with the Johnson-Neyman 95% Confidence Bands. The gray areas are significant confidence regions.

B. The Effect of Favorable/Adverse Social Environment on Hostile View of Relationships by Number of Genetic Plasticity Alleles with the Johnson-Neyman 95% Confidence Bands. The gray areas are significant confidence regions.

C. The Effect of Favorable/Adverse Social Environment on Chronic Anger by the Number of Genetic Plasticity Alleles the Johnson-Neyman 95% Confidence Bands. The gray areas are significant confidence regions.

D. The Effect of Favorable/Adverse Social Environment on Toughness by Number of Genetic Plasticity Alleles with the Johnson-Neyman 95% Confidence Bands. The gray areas are significant confidence regions.

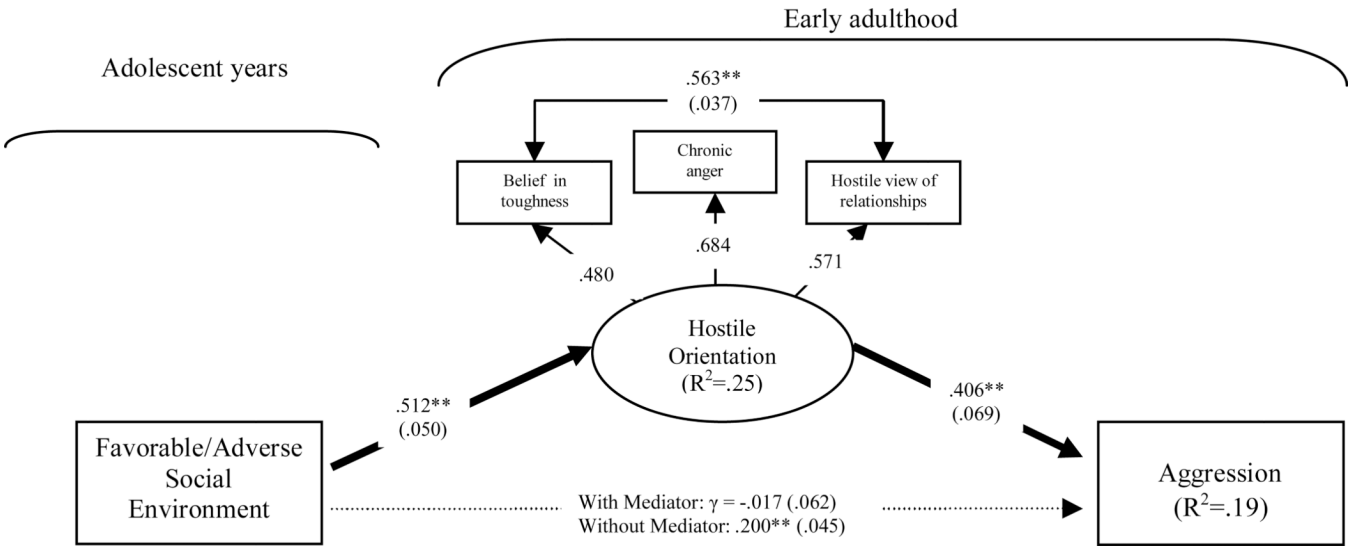

Figure 3.

Hostile Orientation as Mediator of the Effect of Favorable/Adverse Social Environment on Aggression.

Note: χ^2=59.544, *df*=18, *p*=.000. SRMR=.041 and CFI=.929. The values presented are standardized parameter estimates and the standard errors are in parentheses. Previous aggression, gender, area, family SES and family structure are controlled. Using bootstrap methods with 1000 replications, the indirect effect is significant [indirect effect = .208 (the 92% portion of the total variance), $p < .001$].

**$p \leq .01$; *$p \leq .05$, †$p < .10$ (two-tailed tests), n=505

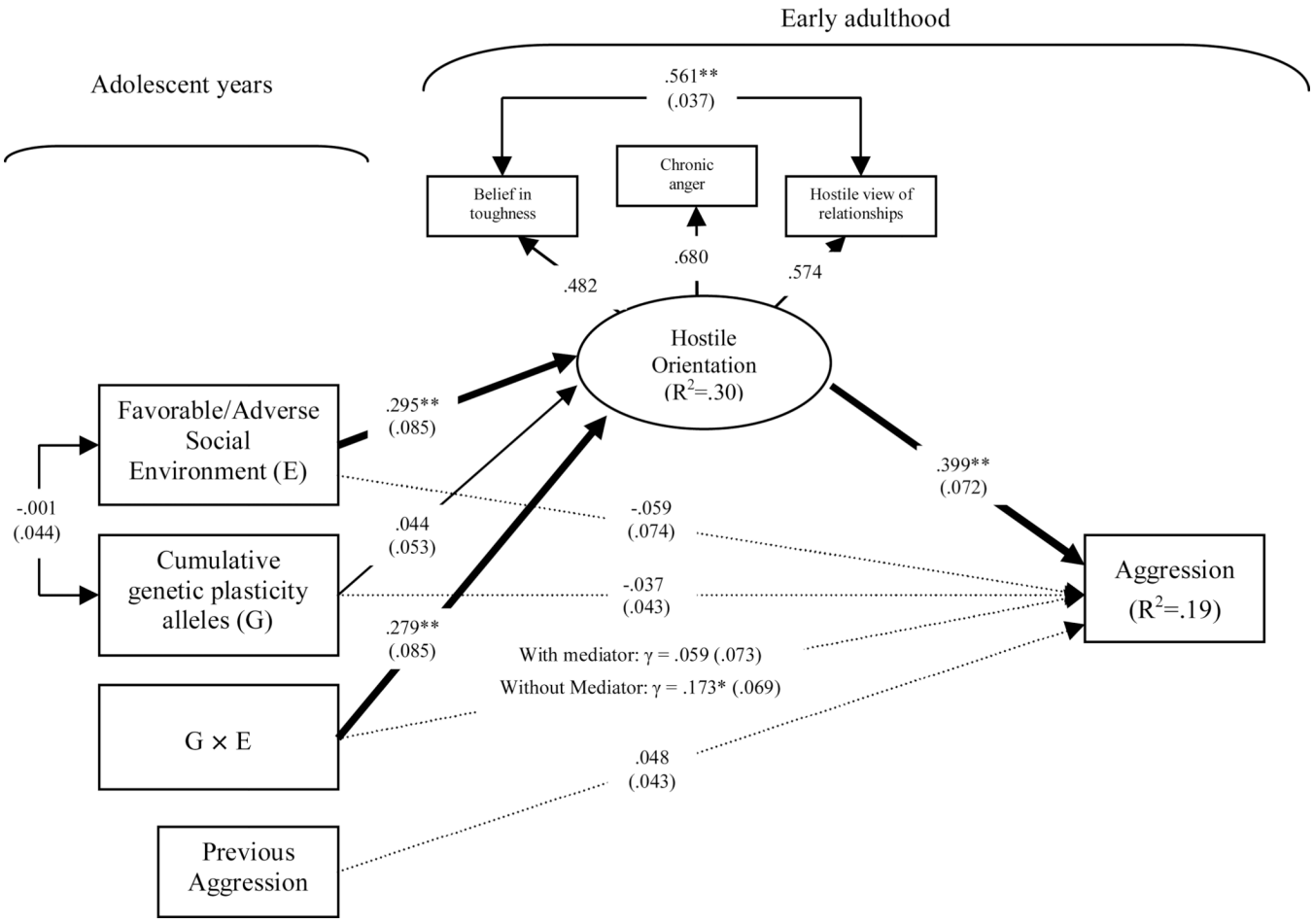

Figure 4.

Mediated Moderation Model.

Note: χ^2=61.854, *df*=22, *p*=.000. SRMR=.036 and CFI=.933. The values presented are standardized parameter estimates and the standard errors are in parentheses. Using bootstrap methods with 1000 replications, the bold lines indicate that the test of the indirect effect of interaction term is significant [indirect effect = .111 (the 65% portion of the total variance), $p<.05$]. Gender, area, family SES and family structure are controlled in these analyses. $^{**}p \leq .01$; $^{*}p \leq .05$, $\dagger p < .10$ (two-tailed tests), n=505

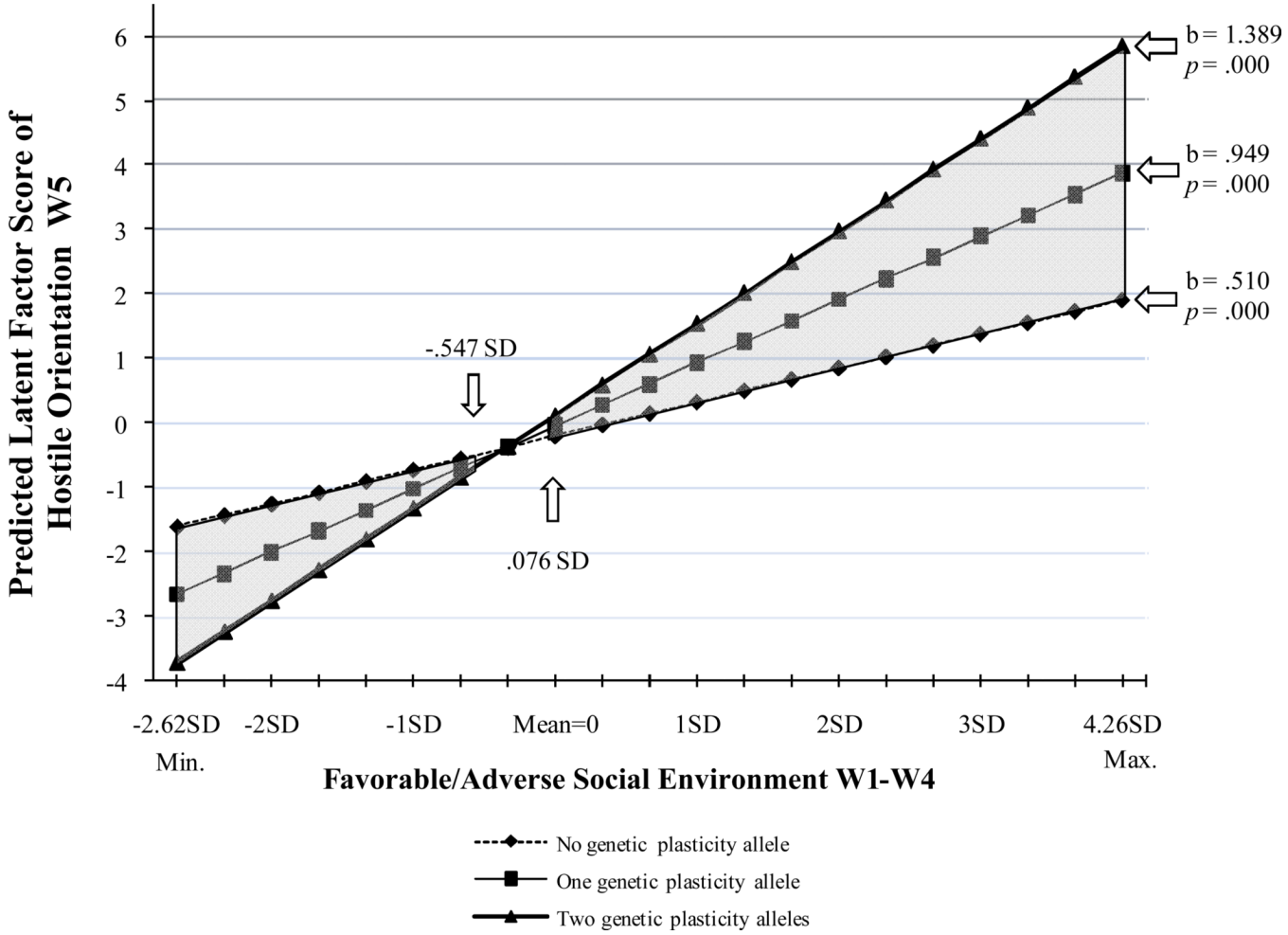

Figure 5.

The Effect of Favorable/Adverse Social Environment on Hostile Orientation by Number of Genetic Plasticity Alleles with the Johnson-Neyman 95% Confidence Bands. The gray areas are significant confidence regions.

Table 1

Alleles Frequency Distributions of the *DRD4* and 5-HTTLPR Gene Polymorphism

		5-HTTLPR		
		Any 5HTT transporter short alleles	**No 5HTT transporter short alleles**	**Total**
DRD4	Any D4 long (7R+ alleles)	91 (18%)	113 (22.4%)	204 (40.4%)
	No D4 long alleles (both alleles less than 7R)	140 (27.7%)	161 (31.9%)	301 (59.6%)
	Total	231 (45.7%)	274 (54.3%)	505 (100%)

Table 2

Correlation Matrix for the Study Variables.

	1	2	3	4	5	6	7	8	9	10	11	12
1. Gender (1 = Male)	——											
2. Area (1 = South)	.025	——										
3. Family SES	.035	−.131 **	——									
4. Family structure (1 = Single-parent families)	−.090 *	030	−.319 **	——								
5. Favorable/adverse social environment (W1-W4)	.060	−.234 **	−.096 *	−.044	——							
6. Hostile view of relationships W5	.106 *	.067	−.049	.029	.321 **	——						
7. Chronic anger W5	−.011	−.009	−.116 **	.084 †	.333 **	.394 **	——					
8. Belief in toughness W5	.183 **	.056	.031	.038	.253 **	.679 **	.318 **	——				
9. Aggression W1	.047	−.116 **	−.012	−.009	.232 **	.048	.084 †	−.027	——			
10. Aggression W5	.137 **	−.047	−.079 †	.033	.224 **	.192 **	.306 **	.226 **	.103 *	——		
11. Cumulative genetic plasticity alleles (0–2)	.015	−.002	−.087 *	.071	−.001	.039	.033	.001	.065	−.005	——	
12. Two genetic plasticity alleles (any 7R and any S)	.041	−.021	−.074 †	.102 *	.021	.024	.028	.004	.045	.008	.771 **	——
Mean	.410	.539	.080	.525	.000	2.370	12.063	11.978	.200	.529	.861	.180
SD	.492	.499	1.554	.498	1.000	1.073	4.398	3.405	.665	1.180	.693	.385

** $p \leq .01$;

* $p \leq .05$,

† $p < .10$ (two-tailed tests); n=505

Table 3

c Diversity as Predictors of Hostile View of Relationships, Chronic Anger, Concern with Toughness and Aggression.

Hostile View of Relationships						Chronic Anger						Belief in Toughness						Aggression					
odel 1a		Model 1b		Model 1c		Model 2a		Model 2b		Model 2c		Model 3a		Model 3b		Model 3c		Model 4a		Model 4b		Model 4c	
b	*β*	b	*β*	b	*β*	b	*β*	b	*β*	b	*β*	b	*β*	b	*β*	b	*β*	b	*β*	b	*β*	b	*β*
**		1.608 **		1.565 **		11.225 **		11.235 **		11.224 **		10.020 **		9.958 **		9.956 **		.373 **		.375 **		.369 **	
)		(.173)		(.175)		(.425)		(.420)		(.454)		(.656)		(.655)		(.673)		(.113)		(.112)		(.129)	
*	.319	.186 *	.173	.211 *	.196	1.492 **	.339	.893 *	.203	.944 *	.215	.940 **	.276	.471 †	.138	.469	.138	.235 **	.199	.078	.066	.126	.107
)		(.075)		(.083)		(.221)		(.403)		(.463)		(.154)		(.255)		(.287)		(.070)		(.103)		(.111)	
	.037	.051	.033			.118	.019	.096	.015			.009	.002	−.011	−.002			−.029	−.017	−.035	−.021		
)		(.059)				(.267)		(.260)				(.191)		(.188)				(.076)		(.075)			
		.188 **	.185					.728 *	.175					.561 *	.174					.193 *	.173		
		(.065)						(.336)						(.220)						(.089)			
				.084	.030					.175	.015					−.054	−.006					−.072	−.023
				(.120)						(.528)						(.375)						(.150)	
				.012	.005					−.203	−.019					−.174	−.021					−.145	−.051
				(.126)						(.477)						(.410)						(.119)	
				.184 †	.077					.457	.047					.305	.040					.047	.018
				(.111)						(.492)						(.370)						(.147)	
				.408 **	.144					1.511 *	.130					1.112 *	.124					.439 *	.141
				(.128)						(.648)						(.436)						(.174)	
				.133	.061					.712	.080					.318	.046					−.037	−.015
				(.123)						(.548)						(.387)						(.129)	
				.137	.069					.564	.070					.799 *	.128					.193	.089

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Hostile View of Relationships						Chronic Anger						Belief in Toughness						Aggression					
Model 1a		Model 1b		Model 1c		Model 2a		Model 2b		Model 2c		Model 3a		Model 3b		Model 3c		Model 4a		Model 4b		Model 4c	
b	β	b	β	b	β	b	β	b	β	b	β	b	β	b	β	b	β	b	β	b	β	b	β
				(.116)						(.608)						(.397)						(.196)	
* .130		.158 **	.134	.166 **	.140	.130 †	.070	.117	.062	.116	.062	.122	.052	.133	.056	.124	.052	.089	.050	.073	.041	.093	.052
		(.052)		(.051)		(.078)		(.078)		(.079)		(.103)		(.102)		(.103)		(.087)		(.085)		(.080)	
* .085		.200 *	.092	.195 *	.089	−.231	−.026	−.172	−.019	−.202	−.023	1.156 **	.167	1.201 **	.173	1.193 **	.172	.308 **	.128	.324 **	.135	.316 **	.132
		(.089)		(.089)		(.380)		(.378)		(.378)		(.288)		(.286)		(.286)		(.110)		(.110)		(.109)	
* .140		.303 **	.141	.307 **	.143	.639 †	.072	.645 †	.073	.654 †	.074	.887 **	.130	.897 **	.132	.915 **	.134	−.013	−.006	−.011	−.005	.008	.003
		(.088)		(.089)		(.383)		(.385)		(.385)		(.289)		(.289)		(.291)		(.100)		(.100)		(.100)	
.021		.014	.020	.017	.025	−.132	−.047	−.134	−.047	−.122	−.043	.216 *	.099	.214 *	.098	.218 *	.099	−.041	−.054	−.042	−.055	−.038	−.051
		(.029)		(.029)		(.103)		(.100)		(.098)		(.098)		(.098)		(.097)		(.035)		(.034)		(.034)	
.055		.116	.054	.112	.052	.667 †	.076	.661 †	.075	.633	.072	.648 *	.095	.642 *	.094	.643 *	.094	.091	.038	.089	.037	.080	.034
		(.093)		(.094)		(.386)		(.382)		(.391)		(.311)		(.308)		(.309)		(.114)		(.113)		(.116)	
		.150		.148		.121		.131		.128		.107		.117		.116		.060		.070		.073	
		.013 **						.011 *						.012 **						.009 *			

efficients are shown with Robust standard errors are in parentheses; environment variable is standardized by z-transformation (mean =0 and SD = 1); no plasticity

Am Sociol Rev. Author manuscript; available in PMC 2012 January 1.