Published in final edited form as:

J Marriage Fam. 2013 April ; 75(2): 325–341.

Genetic Moderation of the Impact of Parenting on Hostility toward Romantic Partners

Ronald L. Simons,
University of Georgia

Leslie Gordon Simons,
University of Georgia

Man Kit Lei,
University of Georgia

Steven R.H. Beach,
University of Georgia

Gene H. Brody,
University of Georgia

Frederick X. Gibbons, and
Dartmouth College

Robert A. Philibert
University of Iowa

Abstract

Although GxE studies are typically based on the assumption that some individuals possess genetic variants that enhance their vulnerability to environmental adversity, the differential susceptibility model posits that these individuals are simply more sensitive to social context, whether that context be adverse or supportive. Thus those persons most vulnerable to adversity are the same ones who reap the most benefit from support. This idea was tested using longitudinal data from a sample of several hundred African Americans. Findings indicated that relatively common variants of the GABRA2 gene interact with parenting to predict hostility toward romantic partners in a manner consonant with the differential susceptibility hypothesis. Individuals with these genetic variants displayed more aggression toward their partner than those with other genotypes when they had been subjected to harsh parenting, but exhibited less aggression toward their partner than other genotypes if their parents avoided harsh parenting practices.

Keywords

sociobiology; intimate partner/marital abuse; parenting styles; intergenerational transmission

In the past decade, there has been an explosion of articles reporting that genetic variation 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 gene by environment (GxE) 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; Simons et al., 2011).

Genetically informed behavioral science requires models of the manner in which genetic variables combine with environmental context to influence behavioral outcomes (Shanahan and Hofer 2005, 2011). Most GxE research has been guided by the diathesis-stress framework which views some individuals as carrying risk alleles (genetic variants) that make them more vulnerable to adverse social conditions than others. Recently, however, a group of researchers has proffered an alternative perspective. They argue that those individuals thought to be at genetic risk for problem behavior in the face of adversity are simply programmed by their genes to be more sensitive than others to environmental context (Belsky & Pluess, 2009; Ellis et al., 2011). Thus those most vulnerable to environmental adversity are the same ones who reap the most benefit from environmental support. Using this framework, known as differential susceptibility, genetic variants considered to be risk alleles within the diathesis-stress perspective are redefined as plasticity alleles that enhance the extent to which an individual is responsive to environmental influence.

Most of the support for the differential susceptibility approach comes from studies that investigated the impact of parenting on child and adolescent development. These studies found that genetic variation interacts with parental behavior in a differential susceptibility fashion to influence outcomes such as conduct problems and depression (Belsky & Pluess, 2009), negative arousal (Beach et al., in press), hostile attribution bias, chronic anger, and aggression (Simons et al., 2011, 2012), self-regulation (Belsky & Beaver, 2011), prosocial behavior (Knafo et al., 2011), and attachment style (Bakermans-Kranenburg & van IJzendoorn, 2007). Most of these studies focused on variation in either the serotonin transporter gene (5-HTT) or dopamine receptor genes (D2 or D4). The present study extends this line of research by investigating the consequences of variation in GABRA2, a gene that codes for receptor sites for a subunit of the neurotransmitter GABA (gamma butyric acid). Based on neurobiological findings discussed below, we expect that that variation in this gene operates to enhance susceptibility to environmental influence. We test this idea by in examining the relationship between the quality of parenting received as a child and subsequent hostility toward adult romantic partners. While past research has documented a relationship between these two constructs (Black, Susman, & Unger, 2010; Carr & Van Deusen, 2002; Foshee, Bauman, & Linder, 1999; Hendy et al., 2003; Kwong, Bartholomew, Henderson, & Trike, 2003; Lewis & Fremouw, 2001; Simons, Simons, & Lie, in press; Tschann et al., 2009), the present study investigates the extent to which variations in the GABRA2 gene moderate this association in the manner predicted by the differential susceptibility perspective.

We investigate this moderating effect using a sample of young African American adults. This population is particularly relevant in examining the association between childhood experiences in the family of origin and subsequent hostility toward adult romantic partners as past research has as there is strong evidence that the romantic relationships of African Americans are more troubled than those of European Americans. Research by Anderson (1999) and Wilson (2003), for example, indicates that the romantic relationships of African American young adults are often fractious, antagonistic, and unstable, and Kurdek (2008) recently reported that Black dating couples exhibited more arguing and relationship hostility than White couples.

A Brief Introduction to Molecular Genetics

The genetic code is composed of nucleotide base pairs (bps) that are organized into genes. Genes represent segments of the genome that produce proteins 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.” Two common types of variation, length and sequence, are observed. Length type variation involves the number of times that a particular set of base pairs is repeated. Depending on the size of the repeat unit, this type of variability is referred to as a Variable Number Tandem Repeat (VNTR) or microsatellite/minisatellite repeat. VNTRs are important as they often alter the product (protein) 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. The other type of genetic variation is Single Nucleotide Polymorphisms, frequently called SNPs, which involve variation in a single nucleotide base pair. Like VNTRs, SNPs can influence the quality and amount of product produced by a gene. Much of the GxE research has focused upon VNTRs. Increasingly, however, studies have broadened their focus to include SNPs in genes such as the gamma-aminobutyric acid receptor gene (GABRA2). In the present study, we focus on SNPs in this gene.

The more common variants of a gene are labeled the major alleles whereas the less common variants are labeled minor alleles. Individuals carry two copies of a gene as they inherit a copy from each of their parents. Thus for any particular polymorphism, they may be homozygous with regard to the major allele (have two copies of the major allele), heterozygous (have a copy of both the major and minor allele), or be heterozygous with regard to the minor allele (have two copies of the minor allele). In most cases, GxE studies examine the way that having one or two copies of the minor allele of some gene changes the way that individuals respond to environmental conditions.

Models of GxE

As noted, genetically informed behavioral science requires models of the manner in which genetic variation combines with social context to influence behavioral outcomes. The diathesis-stress model utilized in the vast majority of GxE studies assumes that allelic variation in particular genes amplifies the probability that exposure to social adversity (e.g., abusive parenting, racial discrimination, economic hardship) will result in maladjustment or problem behavior. Thus the perspective presumes 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 et al., 2011; Homberg and Lesch, 2010). 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 et al., 2007; Belsky and Pluess 2009; Ellis et al. 2011).

Belsky and his colleagues (Belsky, Bakermans-Kranenburg, and von IJzendoorn 2007; Belsky and Pluess 2009) have proposed a GxE model that they label the differential susceptibility perspective. This model posits that the polymorphisms used in most GxE studies of child and adolescent adjustment exert their influence by augmenting susceptibility

to social context, whether that environment is adverse or supportive. These individuals are programmed by their genes to be more sensitive to environmental influence than others. Thus those persons most vulnerable to adverse social environments are the same ones who reap the most benefit from environmental support. In other words, they are more *plastic* than other genotypes. Boyce and Ellis (2005; Ellis et al., 2011) use the term *orchid children* in referring to these individuals as a way of signifying their special sensitivity to both highly stressful and highly nurturing environmental conditions. In contrast, they use the designation *dandelion children* in referring to those without these alleles as a way of indicating their relative ability to function adequately in a wide variety of circumstances.

How would genes cause some individuals to be more susceptible than others to environmental influence? Belsky and his colleagues (Belsky & Pluess, 2009) note that the genes analyzed in most studies of child and adolescent adjustment involve neurotransmitters concerned with either the dopaminergic system which has been implicated in reward sensitivity and sensation seeking or the serotonergic system which has been linked to sensitivity to punishment and displeasure. Further, the low activity or minor alleles associated with these genes tend to increase the activity of the brain's limbic system, especially the amygdala, thereby increasing emotional responsiveness to environmental events. Thus the differential susceptibility model assumes that persons with plasticity alleles are more responsive to environmental rewards and punishments than those with other genotypes.

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. 2007; Belsky and Pluess 2009). In a recent article, Belsky and Pluess (2009) reviewed scores of studies reporting a G×E effect on child or adolescent adjustment. Although these studies appeared to support a stress-diathesis model, Belsky and Pluess concluded that a careful inspection of the results pointed to a different interpretation. All of the studies included in the review showed a cross-over effect.

Since Belsky and Pluess published their review article, a number of additional papers supporting the differential susceptibility perspective have been published. This includes a meta-analysis (Bakermans-Kranenburg & Van Ijzendoorn, 2011) of G×E studies focusing on youth externalizing problems and a recent issue of *Development and Psychopathology* (February, 2011) that focused entirely upon research supporting the differential susceptibility perspective. The vast majority of these studies have focused upon the moderating effect of genetic variation on the relationship between parenting behavior and child or adolescent adjustment.

The Present Study

The present study extends prior research on the differential susceptibility perspective by examining the effects of variation in the GABRA2 gene. This gene has been linked to a variety of externalizing behaviors such as conduct problems (Dick et al., 2006; Dick et al., 2009), illicit drug use (Agrawal et al., 2006; Dick et al., 2006), and alcohol dependence (Covault et al., 2004; Fehr et al., 2006; Soyka et al., 2008). Although most of these studies did not test for a G×E effect, Dick et al. (2009) recently reported that GABRA2 interacts with parental monitoring to predict trajectories of externalizing behavior. Like most G×E studies, the analyses reported in this study assumed a diathesis-stress perspective. In the present study, we explore the possibility that GABRA2 is a plasticity gene. Given GABRA2's effect on the neurotransmitter Gama-Aminobutyric Acid (GABA), there is good

reason to believe that variability in this gene influences susceptibility to social environmental events.

GABA is the major inhibitory neurotransmitter in the mammalian central nervous system and it plays a major role in regulating neuronal excitability. GABA antagonists enhance activity in brain limbic structures such as the amygdala, insula and striatum (Dunsmoor et al., 2011; Phillips et al., 2003). Increased activation of these structures is associated with elevated vigilance and emotional responsiveness to environmental events. The GABRA2 gene influences receptor sites for GABA and therefore might be expected to affect vigilance and emotional responsiveness to environmental events through its impact on amygdala, insula, and striatum activity. Consistent with this hypothesis, the minor alleles in GABRA2 have been associated with greater disinhibition and excitability in the brain (Edenberg et al., 2004), with neuroticism (Penelope et al., 2008), and with impulsiveness and fMRI changes in the insula and amygdala in response to anticipated rewards or costs (Villafuerte et al., 2011).

These findings indicate that variability at GABRA2 is a viable individual difference variable that may enhance responsiveness to environmental events, especially emotionally significant experiences such as those that occur in the course of family interaction. In support of this idea, Nelson et al. (2009) recently reported that variation in GABRA2 interacted with childhood maltreatment to influence risk for adult post-traumatic stress. This finding was interpreted within a diathesis-stress framework, viz., minor alleles in GABRA2 enhance responsiveness to adversity. The evidence reviewed above indicates, however, that these alleles are associated with fMRI changes in the limbic system in response to anticipated costs *and* rewards (Villafuerte et al., 2011). This suggests that the neurological consequence of these minor alleles in GABRA2 is to enhance responsiveness to the full range of environmental events, whether adverse or favorable. We test this idea by investigating the extent to which variations in GABRA2 moderate the often reported link between quality of parenting received in childhood and adult hostility toward romantic partners (Black et al., 2010; Carr & Van Deusen, 2002; Foshee et al., 1999; Hendy et al., 2003; Kwong et al., 2003; Lewis & Fremouw, 2001; Tschann et al., 2009).

Our investigation utilizes two single nucleotide polymorphisms (SNPs) that have been included in much of the past research on GABRA2. Each of these SNPs represents a commonly studied haplotype. A haplotype is a set of SNPs that are highly statistically associated ($r = .98$), suggesting that they tend to be transmitted together. We examine the extent to which these two SNPs moderate the impact of parenting received at ages 10–12 on the probability of engaging in verbal and physical aggression toward a romantic partner during early adulthood. If these SNPs operate as plasticity alleles, individuals with the minor alleles should display more verbal and physical aggression toward their partner than those with the major alleles when subjected to harsh parenting as a child, but exhibit less verbal and physical aggression toward their partner than those with the major alleles when they were the recipient of supportive parenting

METHOD

Sample

We tested our hypotheses using data from waves 1, 2 and 5 of the Family and Community Health Study (FACHS), a multi-site investigation of neighborhood and family effects on health and development. FACHS was designed to identify neighborhood and family processes that contribute to school-age African American children's development in families living in a wide variety of community settings outside the inner-city core. Each family included a child who was in 5th grade at the time of recruitment. At wave 1, the FACHS

sample consisted of 889 African American children (411 boys and 478 girls) and their primary caregivers. At study inception, about half of the sample resided in Georgia and the other half in Iowa. The children averaged 10 years of age (5th grade) at Wave 1 (1997–1998) and 20 years of age at wave 5 (2007–2008). Of the 889 targets interviewed at Wave 1, 779 were reinterviewed at Wave 2, and 689 at Wave 5 (78% of the original sample). At wave 1, the sample had an average family per capita income of \$6956/year. Thirty six percent of the families were below the poverty line, and fifty one percent of the respondents lived in a single-parent family. Additional details regarding sampling procedures and the demographic characteristics of the participants can be found in Gibbons et al. (2004) and Simons et al. (2011). As part of wave 5 data collection, targets were asked to provide DNA for genotype analysis. Of the 689 participants, 549 (80%) agreed to DNA collection. Successful genotyping for GABRA2 SNPs (single nucleotide polymorphisms) was achieved for 542 individuals.

Current study participants

The present study focused on the 46% of respondents who reported at wave 5 that they were involved in a committed romantic relationship ($n = 319$ individuals, 129 men and 190 women). Analyses indicated that those individuals with a romantic partner did not differ significantly from those without a romantic partner regarding exposure to harsh parenting and demographic variables. Of the respondents with a romantic partner, 253 (79%) were successfully genotyped. Finally, GABRA2 SNP assays and videotaped family interaction data were available for 242 of these individuals ($n = 94$ men and 148 women). Comparison of those participants excluded from the present study with those retained did not identify any significant differences with regard to age, gender, household income, or parent-child observed interactions at waves 1 or 2. Further, including Heckman's Lambda (Heckman, 1979) in our analyses did not change the findings, suggesting an absence of selection bias.

Procedures

At each wave, computer assisted interviews were administered in the respondent's home and took on average about 2 hours to complete. 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.

A 20-minute videotape of parent-child interaction was obtained at both waves 1 and 2. Parent and child were seated at a table (or a couch) facing the camera and were given a set of 16 cards with trigger questions about topics such as what they enjoy doing together, parental rules and expectations, and the child's biggest accomplishment or disappointment in the past year. They were asked to read and respond to the questions. Interviewers provided instructions, set up and started the video equipment, and left the room so as not to hear the recorded discussion.

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 manufacturer's specifications.

Measures

Hostility toward Intimate Partner was assessed at wave 5 using five items from the Conflict Tactics Scale (Straus & Gelles, 1990). The items asked how often during the past year that respondents had engaged in the following acts when they had had a disagreement with their

romantic partner: shout or yell at him/her, insult or swear at him/her, slap or hit him/her, throw things at him/her, and hit him/her with an object. The response format for the items ranged from 1 = *never* to 4 = *always*. Scores were summed across items to form a measure of hostility toward one's intimate partner. Coefficient α for the measure was .73.

Harsh Parenting was assessed using the videotapes of parent-child interaction collected at waves 1 and 2. Observers trained in the use of the Iowa Family Interaction Rating Scales (Melby et al., 1990; McGruder, Lorenz, Hoyt, Ge, & Montague, 1992) rated six dimensions of parental behavior: hostility, verbal attack, physical attack, reciprocate hostile, prosocial behavior, and communication. Before observing tapes, coders had to independently rate precoded interaction tasks and achieve at least 90% agreement with the standard for the task. Twenty-five percent of the tapes were randomly selected to be independently observed and rated by a second observer. The intraclass correlation for these comparisons averaged from .60 to .70. The rating format for each scale ranged from 1 to 9, with higher scores indicating greater intensity and/or frequency of the dimension being rated. We reverse coded the positive dimensions, and then summed the six rating scales to form a measure of harsh parenting for each wave. Coefficient alpha for the scale was .79 at wave 1 and .73 at wave 2. Low scores indicated positive interaction with little or no hostility whereas high scores indicated hostility with little or no positive interaction. Scores for the two waves were standardized and averaged to form a composite measure of harsh parenting.

Control variables. The analyses controlled for four demographic measures. Respondent gender was coded 1 = *males* (38.8 percent) and 0 = *females* (61.2 percent). Region was coded 1 for respondents living in the South (52.1 percent) and 0 for those living in other areas of the country. Family SES was a composite measure based on the primary caregiver's education and family income ($M = .04$, $SD = 1.44$). Family structure was coded as a dummy variable: 1 = *single-parent family* (52.1 percent), 0 = *other family type*.

Genotyping. All participants were genotyped using Taqman[®] MGB assays (Applied Biosystems, Foster City, CA) and Fluidigm Biomark Genetic Analysis System (Fluidigm, South San Francisco, CA). In our dataset, the GABRA2 (GABAA receptor subunits alpha-2) was genotyped using a panel of 11 SNPs.

Analytic Strategy

Hierarchical regression models were employed to test the differential susceptibility hypothesis using SAS software, version 9.1 (SAS Institute Inc., Cary, NC). Coefficients are presented with robust standard errors. Item-level multiple imputation (MI) techniques were used to correct for missing data. Independent variables were standardized (mean of 0 and a standard deviation of 1) before interaction terms were calculated. Standardization makes for easier interpretation of coefficients, reduces multicollinearity, and makes the simple slope easier to test (Dawson & Richter, 2006). When gene-environment interaction effects were significant, post hoc analyses of interaction terms were conducted using the Johnson-Neyman (J-N) technique (Johnson & Neyman, 1936; Hayes & Matthes, 2009). This procedure identifies regions of significance for interactions between continuous (quality of parenting) and categorical variables (GABRA2). In addition to hierarchical regression models, a two-way ANCOVA was used to assess the effects of harsh parenting and GABRA2 on hostility toward romantic partner. The model allows us to estimate a nonlinear combination of gene-environment interactions. To control for the inflated probability of Type I error in multiple tests, a false discovery rates (FDR) p -value was used (SAS, PROC MULTTEST).

RESULTS

Initial Findings Regarding the GABRA2 SNPs

A haplotype is a set of adjacent SNPs that are highly statistically associated, suggesting that they tend to be transmitted together. Using our 242 respondents, the program Haploview through the “strong LD Spine” algorithm (Barrette et al., 2005) indicated that the 11 GABRA2 SNPs available in our data set comprised two haplotype blocks. As shown in Figure 1, these two haplotype blocks are outlined by a solid line, and the R-square (LD) between two SNPs is shown in the cross cells. Block 1 (104000 base pairs in length) included three SNPs: rs531460, rs567926, and rs279858; and Block 2 (6000 base pairs in length) consisted of two SNPs: rs1440130 and rs279837.

We conducted our analyses using the five SNPs from these two haplotype blocks. These SNPs have been used in studies examining the effect of GABRA2 on various behavioral and neurological outcomes (e.g., Dick et al., 2009; Philibert et al., 2009; Ittiwut et al., 2011). For example, rs279858 was the SNP used by Villafuerte et al. (2011) in their fMRI study of responses to anticipated costs and rewards. Past research has reported that it is the G allele for these SNPs that is associated with increased risk for problem behavior.

Among the 242 respondents used in our analysis, in Block 1, 8.8% were homozygous for the minor allele A at rs531460, 34.6% were heterozygous (A/G), and 56.7% were homozygous for the major allele G; 8.3% were homozygous for the minor allele G at rs567926, 38.2% were heterozygous (A/G), and 53.5% were homozygous for the major allele A; 8.3% were homozygous for the minor allele G at rs279858, 36.8% were heterozygous (A/G), and 55% were homozygous for the major allele A. Similarly, in Block 2, 11.2% of respondents were homozygous for the minor allele G of rs1440130, 42.3% were heterozygous (A/G), and 46.5% were homozygous for the major allele A; 9.9% of respondents were homozygous for the minor allele G of rs279837, 38% were heterozygous (A/G), and 52.1% were homozygous for the major allele A. The observed distribution of these SNPs did not differ significantly from that predicted by the Hardy-Weinberg equilibrium law.

In the analyses presented below, we treat each of the SNPs as a dichotomous variable where individuals receive a score of 0 if they are homozygous for the major allele and a score of 1 if they have at least one copy of the minor allele (i.e., they are either homozygous or heterozygous for the minor allele). According to the differential susceptibility perspective, the more plasticity alleles one carries, the more susceptible one will be to environmental influence (Belsky and Pluess 2009). Therefore, in addition to analyzing their individual effects, we selected rs279858 to represent Block 1 and rs27837 to represent Block 2 and summed the two SNPs to assess the consequences of cumulative plasticity. Respondents with the major allele A of rs279858 and rs279837 received a score of 0, those with the G-allele on either rs279858 or rs279837 received a score of 1, and those with the G-allele on both rs279858 and rs279837 received a score of 2. Results showed that 102 individuals (42.1%) received a score of 2.

Descriptive and Association analysis

A substantial proportion of respondents indicated that they had perpetrated acts of physical and verbal aggression toward their partner. For instance, 64% of respondents reported that they had shouted or yelled at their partner and 46.7% indicated they had insulted or swore at their partner. Roughly 12% reported that they had hit, pushed, or grabbed their partner, 6.2% that they had thrown things at their partner, and 4% that they had struck their partner with an object.

The means, standard deviations, and zero-order correlations among the study variables are presented in Table 1. The table shows that low family SES is associated with greater childhood exposure to harsh parenting. As expected, harsh parenting is significantly correlated with hostility toward intimate partner. There is no significant correlation between either of the two GABRA2 SNPs and hostility toward partner. This finding is consistent with previous molecular genetic studies indicating that so-called risk alleles generally have little main effect on problem behavior (Caspi et al., 2003; Moffitt et al., 2005).

Before testing for G×E effects, we examined our data for evidence of gene-environment correlation (rGE), i.e., the extent to which genetic variation is associated with individual differences in exposure to environments (Shanahan & Hofer, 2005). Importantly, rGE is likely to confound G×E effects (Caspi & Moffitt 2006). Table 1 shows that there are no significant correlations between harsh parenting and the GABRA2 SNPs. Thus there is no evidence of an active rGE effect where people seeking out or evoke environments that are compatible with their genetic predispositions. Furthermore, in analyses not shown, there was no significant relationship between parental genotype and either harsh parenting or hostility toward partner. Thus there is no evidence of a passive rGE where parents both pass on their genes to their children and share their environment. These findings suggest that rGE effects are not a factor in our analysis.

The Effect of Gene-Environment Interaction on Hostility toward Partner: Regression Analyses

Table 2 presents the results of regression analyses where hostility toward partner is predicted from the five SNPs of GABRA2 and the observers' ratings of parental hostility. All of the results reported are for the total sample as our analyses indicated that none of the models presented differ by gender of respondent. Controlling for the demographic measures, results from Model 1a through Model 5a show that the main effects of harsh parenting are significantly associated with perpetration, whereas the five SNPs of GABRA2 are not. Model 1b through Model 5b add the interaction of harsh parenting with the GABRA2 SNPs to the regression equation. Neither harsh parenting nor the GABRA2 SNPs are significant in this model, but the interaction of these two variables is a significant predictor of hostility toward partner. For example, Model 5a shows that the main effect of harsh parenting on hostility toward partner is significant ($b = .265, p = .034, \text{Adjusted FDR } p = .039$) whereas SNP rs279837 is not; Model 5b adds the multiplicative interaction term and shows that there is a significant interaction of harsh parenting and rs279837 in predicting hostility toward partner ($b = .691, p = .002, \text{Adjusted FDR } p = .010$).

Having established the significance of the interactions between GABRA2 genotype and harsh parenting, the next step was to examine and graph these interactions to see if there is evidence of the cross-over pattern predicted by the differential susceptibility argument. First, the bottom of Table 2 shows that the effect of harsh parenting on hostility toward partner is strongest for respondents with at least one copy of the minor allele and weakest for those homozygous for the major allele. Indeed, based on the simple slope test (Aiken & West, 1991), the slopes for respondents with at least one copy of the minor allele are significantly different from zero, whereas the slopes are not significantly different from zero for those homozygous for the major allele. For example, Figure 2 shows that for SNP rs279837 the slope is .602 ($p = .0002, \text{Adjusted FDR } p = .0010$) for those with at least one copy of the minor allele versus an insignificant $-.089$ for those homozygous for the major allele.

More importantly, given our concern with testing the differential susceptibility perspective, the pattern of interaction in Figure 2 is that of a cross-over effect. This indicates that when exposure to harsh parenting was high those with at least one copy of the minor alleles of GABRA2 show significantly more hostility toward their romantic partner than those with no

copies, whereas those with at least one copy demonstrate significant less hostility toward their romantic partner than those with no copies if their parents engaged in little or no harsh parenting. The J-N technique was used to investigate the significance of this pattern of findings (Hayes & Matthes, 2009). Consonant with the differential susceptibility argument, Figure 2 shows that carriers of the minor allele on rs279837 show more hostility toward their partner than those homogenous for the major allele when level of harsh parenting is greater than 1.193 standard deviations above the mean, whereas they display significantly less hostility toward their partner than those with the major allele when harsh parenting is less than $-.485$ standard deviations below the mean. Approximately 16% of respondents in our sample scored above 1.19 standard deviations on harsh parenting and 38% of respondents scored below $-.485$ standard deviation on our outcome. Although not shown for reasons of parsimony, the graphs for all interactions in Table 2 indicate a pattern virtually identical to those depicted in Figure 2.

The Effect of Gene-Environment Interaction on Hostility toward Partner: ANCOVA Analyses

It is possible that the crossing pattern shown in Figure 2 is consequence of constraining the effect of harsh parenting to be linear. To further explore this possibility, we grouped our respondents based on whether they had experienced high (above one standard deviation, 18.6%), medium (within ± 1 standard deviation, 69.8%) or low (below one standard deviation, 11.6%) exposure to harsh parenting. Using a 3×2 ANCOVA with harsh parenting and the GABRA2 SNPs as factors, Table 3 reveals that there are no significant main effects of harsh parenting and the GABRA2 gene, but the interaction effects are statistically significant predictors of hostility toward partner.

For example, the interaction effect between SNP rs279837 and harsh parenting is a significant predictor of hostility toward partner with $F(2, 232) = 18.402, p = .002$, and Adjusted FDR $p = .005$. The bar graph in Figure 3 shows the results of graphing this interaction. The figure indicates that carriers of the minor allele on rs279858 show more hostility toward their partner than those homogenous for the major allele when their parents engaged in high levels of harsh parenting [simple effect test: $F(1, 232) = 4.955, p = .027$]. By contrast, when exposure to harsh parenting was low those with at least one copy of the minor alleles on rs279858 show significant less hostility toward their romantic partner than those with no copies [simple effect test: $F(1, 232) = 7.867, p = .005$]. Although not presented for the purpose of brevity, the graphs of the interaction of harsh parenting and the other GABRA2 SNPs are almost identical to the pattern in Figure 3. In other words, both regression and ANCOVA analyses tell a similar story. GABRA2 operates as a plasticity gene that influences the extent to which individuals are responsive to harsh parenting.

The Effect of Gene-Environment Interaction on Hostility toward Partner: Cumulative Plasticity

Finally, the index of cumulative plasticity alleles was assessed using two representative SNPs from Blocks 1 and 2: rs279858 and rs27837. Similar to using a single SNP, Figure 4 shows a similar pattern of findings when the index of cumulative plasticity is used. The slope does not differ from zero for individuals homogenous for the major allele on both SNPs, whereas it is significantly different from zero for individuals that carry copies of the minor alleles. Consistent with the differential susceptibility perspective, the slopes are steeper the greater the number of plasticity alleles and present the expected crossing pattern. Using the J-N technique, cumulative plasticity significantly increases hostility toward the romantic partner when harsh parenting is greater than .966 standard deviations above the mean while it significantly decreases hostility toward the partner when it is less than $-.834$

standard deviations below the mean. These results provide strong support for the differential susceptibility perspective.

DISCUSSION

In recent years, there has been a proliferation of studies investigating the manner in which genetic variation combines with environmental context to influence various behavioral outcomes. Most of this research has been informed by the diathesis-stress perspective which assumes that some individuals possess genetic liabilities that make them more vulnerable to adverse environments than other genotypes. Recently, some researchers have begun to investigate an alternative model of G×E labeled the differential susceptibility perspective (Belsky & Pluess, 2007; Ellis et al., 2011). This model posits that many of the genes included in past G×E studies do not simply enhance sensitivity to adversity; rather, they amplify responsiveness to context more generally, whether that context is positive or negative.

The present study extended past research by examining the extent to which variants of the GABRA2 gene interact with parental behavior in a manner predicted by the differential susceptibility perspective. The candidate genes utilized in any G×E study need to be selected based upon neuroscientific findings regarding their effects (Belsky & Pluess, 2009; Caspi & Moffitt, 2006). Based upon neurobiological findings for GABRA2, we expected that this gene likely influences an individual's responsiveness to environmental events. We also extended past research on the differential susceptibility perspective by focusing upon an adult outcome. The vast majority of differential susceptibility studies have concentrated on the way that parental behavior interacts with a particular gene to influence child or adolescent adjustment. The present study expanded this focus by examining the way that parental behavior interacts with GABRA2 to influence interaction with adult romantic partners.

Our results provided rather strong support for the differential susceptibility perspective. Individuals with GABRA2 minor alleles displayed more verbal and physical aggression toward their partner than those with the major alleles when they had been subjected to harsh parenting as a child, but exhibited less verbal and physical aggression toward their partner than those with the major alleles when their parents had engaged in supportive parenting practices. Further, this pattern became stronger as the number of plasticity alleles increased. The diathesis-stress perspective would predict the first of these findings but not the second. This pattern of results is consistent with the argument that GABRA2 influences the extent to which youth are responsive to the influences exerted by their social environment, whether these influences are favorable or adverse. In the absence of information about genetics, our study would have both over- and under-estimated the effects of the rearing experience under consideration by failing to make clear that the effect is bigger for those who are susceptible and smaller for those who are less so.

The present study included several strengths. Chief among them was the use of longitudinal data and the avoidance of shared methods bias by using observer ratings to assess childhood exposure to harsh parenting and questionnaire data to assess adult relationship hostility. Nevertheless, the study also contained certain limitations. First, self-reports were used to assess hostility toward romantic partners. Availability of partner reports or observer ratings would have increased the validity of this measure. Although self-reports undoubtedly contain a bias, it is not clear how this would have contributed to the pattern of findings obtained in our analyses. Second, there is the issue of the homogeneity of our sample. Although there is a need for research on the determinants of intimate partner violence among African Americans, we are left with the question of the extent to which our results

can be generalized to other racial/ethnic groups. Although we cannot think of any reasons why our findings regarding the moderating effects of GABRA2 would be specific to African Americans, the findings clearly need to be replicated with more diverse samples.

Finally, our analyses did not provide any information regarding the mechanisms whereby parenting behavior influences behavior toward romantic partners. One might speculate that GABRA2 is enhancing the learning of cognitive schemas involving intimate relationships. Recently, Simons and colleagues (Simons et al., 2011) argued that one of the implications of the differential susceptibility perspective is that persons with plasticity alleles should learn the norms, schemas, attitudes, and values subtly communicated by their environment more quickly than other genotypes. In support of this idea, Simons et al. (2011, 2012) reported results showing that young adults with various putative plasticity alleles are more likely than other genotypes to adopt the code of the street, a hostile view of relationships, and a cynical view of conventional norms when they grow up in a dangerous social environment but are less likely than other genotypes to adopt these deviant schemas when they are raised in a more favorable social milieu. Similarly, a recent study by Gibbons et al. (in press) found that these alleles moderated the impact of discrimination on future orientation and risk prototypes in a manner consonant with differential susceptibility. Applying these findings to the present study, it may well be the case that GABRA2 interacts with parenting behavior to influence the learning of models of relationships, attributional styles, and other cognitive schemas that serve to mediate the effect of G×E on behavior toward romantic partners. This hypothesis might be explored in subsequent research.

Recent studies indicate that genes such as 5HTT and DRD4 are plasticity genes in that they increase, for better or worse, sensitivity to environmental influence (Belksy & Pleuss, 2009). Our results suggest that GABRA2 may also be a plasticity gene. To the extent that this is the case, carriers of minor alleles are likely vulnerable to a wide variety of problem behaviors, and not simply relationship hostility, in response to adverse environments. On the other hand, they would be expected to flourish compared to other genotypes when environmental conditions are favorable. This increased responsiveness to a favorable environment suggests a more optimistic view of potential for change. Whereas the diathesis-stress perspective paints such persons as difficult to change given their genetic tendency to be hyper-responsive to adversity, the differential susceptibility model argues 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 7-repeat 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 7-repeat allele DRD4 and substance use. Together, these interventions provide strong support for the contention that those genotypes most likely to develop problem behaviors in response to adversity are also the ones most likely to benefit from intervention. Hopefully, future studies will examine the extent to which various putative plasticity genes, including GABRA2, enhance the learning of messages conveyed in relationship education and treatment programs.

In conclusion, a variety of studies have reported that exposure to harsh parenting as a child increases the probability of engaging in hostility toward adult romantic partners (Black et al., 2010; Carr & Van Deusen, 2002; Foshee et al., 1999; Hendy et al., 2003; Kwong et al., 2003; Lewis & Fremouw, 2001; Tschann et al., 2009). The present study indicates that this

relationship is conditioned by the gene GABRA2. Such findings suggest that family research can be made more precise by including genetic variables. G×E studies require, however, a model of the manner in which genetic variation and the social environment combine to influence behavior. 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 social circumstances than those with other genotypes (Belsky & Pluess, 2009; Ellis et al., 2011). The fact that genetic data is now available in many secondary data sets (e.g., Add Health) means that family scholars are now able to test the differential susceptibility model, as well as other G×E perspectives, regarding the complex interplay of genes and social context. The consequence is likely to be a more precise and comprehensive understanding of how family processes influence behavior.

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.

REFERENCES

- Agrawal A, Edenberg HJ, Foroud T, Bierut LJ, Dunne G, Hinrichs AL, Nurnberger JI, Crowe R, Kuperman S, Schuckit MA, Begleiter H, Porjesz B, Dick DM. Association of GABRA2 with drug dependence in the collaborative study of the genetics of alcoholism sample. *Behavior Genetics*. 2006; 36(5):640–650. [PubMed: 16622805]
- Aiken, LS.; West, SG. Multiple regression: Testing and interpreting interactions. Newbury Park, CA: Sage; 1991.
- Anderson, E. Code of the street: Decency, violence, and the moral life of the inner city. New York, NY: W. W. Norton & Company; 1999.
- Bakermans-Kranenburg MJ, van IJzendoorn MH. Oxytocin receptor (OXTR) and serotonin transporter (5-HTT) genes associated with observed parenting. *Social Cognitive and Affective Neuroscience*. 2008; 3:128–134. [PubMed: 19015103]
- Bakermans-Kranenburg MJ, Van IJzendoorn. Differential susceptibility to rearing environment depending on dopamine-related genes: New evidence and a meta-analysis. *Development and Psychopathology*. 2011; 23:39–52. [PubMed: 21262038]
- Barrett JC, Fry B, Maller J, Daly MJ. Haploview: Analysis and visualization of LD and haplotype maps. *Bioinformatics*. 2005; 21(2):263–265. [PubMed: 15297300]
- Beach SRH, Brody GH, Lei M-K, Philibert RA. Differential susceptibility to parenting among African American youths: Testing the DRD4 hypothesis. *Journal of Family Psychology*. 2010; 24:513–521. [PubMed: 20954761]
- Beach SRH, Lei MK, Brody GH, Simons RL, Cutrona C, Philibert RA. Genetic Moderation of Contextual Effects on Negative Arousal and Parenting in African-American Parents. *Journal of Family Psychology*. Nov 14.2011 Advance online publication.
- Belsky J, Bakermans-Kranenburg MJ, van IJzendoorn MH. For better and for worse: Differential susceptibility to environmental influences. *Current Directions in Psychological Science*. 2007; 16:300–304.
- Belsky J, Pluess M. Beyond diathesis-stress: Differential susceptibility to environmental influences. *Psychological Bulletin*. 2009; 135:885–908. [PubMed: 19883141]
- Belsky J, Beaver KM. Cumulative-genetic plasticity, parenting and adolescent self-regulation. *Journal of Child Psychology and Psychiatry*. 2010; 52:619–626. [PubMed: 21039487]
- Black DS, Sussman S, Unger JB. A further look at the intergenerational transmission of violence: Witnessing interparental violence in emerging adulthood. *Journal of Interpersonal Violence*. 2010; 25(6):1022–1042. [PubMed: 19801446]

- Boyce WT, Ellis BJ. Biological sensitivity to context: I. An evolutionary–developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology*. 2005; 17:271–301. [PubMed: 16761546]
- Brody GH, Beach SRH, Philibert RA, Chen Y-F, Lei M-K, Murry VM, Brown AC. Parenting moderates a genetic vulnerability factors in longitudinal increases in youths' substance use. *Journal of Consulting and Clinical Psychology*. 2009; 77:1–11. [PubMed: 19170449]
- Carr JL, VanDeusen KM. Risk factors for male sexual aggression on college campuses. *Journal of Family Violence*. 2004; 19(5):279–289.
- Caspi A, Sugden K, Moffitt TE, Taylor A, Craig LW, Harrington H, et al. Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT Gene. *Science*. 2003; 301:386–389. [PubMed: 12869766]
- Caspi A, Moffitt T. Gene-environment interactions in psychiatry. *Nature Reviews Neuroscience*. 2006; 7:583–590.
- Covault J, Gelernter J, Hesselbrock V, Nellissery M, Kranzler HR. Allelic and haplotypic association of GABRA2 with alcohol dependence. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*. 2004; 129B:104–109.
- Dawson JE, Richter AW. Probing three-way interactions in moderated multiple regression: Development and application of a slope difference test. *Journal of Applied Psychology*. 2006; 91(4):917–926. [PubMed: 16834514]
- Dick DM, Bierut LJ, Hinrichs AL, Fox L, Bucholz K, Kramer JR, Kuperman S, Hesselbrock M, et al. The role of GABRA2 in risk for conduct disorder and alcohol and drug dependence across developmental stages. *Behavior Genetics*. 2006; 36:577–590. [PubMed: 16557364]
- Dick DM, Latendresse SJ, Lansford JE, Budde J, Goate A, Dodge KA, Pettit GS, Bates JE. The role of GABRA2 in trajectories of externalizing behavior across development and evidence of moderation by parental monitoring. *Archives of General Psychiatry*. 2009; 66:649–657. [PubMed: 19487630]
- Edenberg HJ, Dick DM, Xuei X, Tian H, Almasy L, Bauer LO, Crowe RR, et al. Variations in GABRA2, encoding the $\alpha 2$ subunit of the GABAA receptor, are associated with alcohol dependence and with brain oscillations. *The American Journal of Human Genetics*. 2004; 74:705–714.
- Ellis BJ, Boyce WT, Belsky J, Bakermans-Kranenburg MJ, Van Ijzendoorn MH. Differential susceptibility to the environment: An evolutionary-neurodevelopmental theory. *Development and Psychopathology*. 2011; 23:8–28.
- Fehr C, Sander T, Tadic A, Lenzen KP, Anghelescu I, Klawe C, Dahmen N, Schmidt LG, Szegedi A. Confirmation of association of the GABRA2 gene with alcohol dependence by subtype-specific analysis. *Psychiatric Genetics*. 2006; 16:9–17. [PubMed: 16395124]
- Foshee VA, Bauman KE, Linder GF. Family violence and the perpetration of adolescent dating violence: Examining social learning and social control processes. *Journal of Marriage and the Family*. 1999; 61:331–342.
- Gibbons FX, Gerrard M, Cleveland MJ, Wills TA, Brody GH. Perceived discrimination and substance use in African American parents and their children: a panel study. *Journal of Personality and Social Psychology*. 2004; 86:517–529. [PubMed: 15053703]
- Gibbons FX, Roberts ME, Gerrard M, Zhigang L, Beach SRH, Simons RL, Weng C, Philibert RA. The impact of stress on the life history strategies of African American adolescents: Cognitions, genetic moderation, and the role of discrimination. *Developmental Psychology*. in press
- Guo G, Roettger ME, Cai T. The integration of genetic propensities into social-control models of delinquency and violence among male youths. *American Sociological Review*. 2008; 73:543–568.
- Guo G, Tillman KH. Trajectories of depressive symptoms, dopamine D2 and D4 receptors, family socioeconomic status and social support in adolescence and young adulthood. *Psychiatric Genetics*. 2009; 19:14–26. [PubMed: 19125104]
- Hayes AF, Matthes J. Computational procedures for probing interactions in OLS and logistic regression: SPSS and SAS implementations. *Behavior Research Methods*. 2009; 41:924–936. [PubMed: 19587209]
- Heckman J. Sample selection bias as a specification error. *Econometrica*. 1979; 47:153–161.

- Hendy HM, Weiner K, Bakerofskie J, Eggen D, Gustitus C, McLeod KC. Comparison of six models for violent romantic relationships in college men and women. *Journal of Interpersonal Violence*. 2003; 18:645–665.
- Homberg JR, Lesch K-P. Looking on the bright side of serotonin transporter gene variation. *Biological Psychiatry*. 2010; 69:513–519. [PubMed: 21047622]
- Johnson PO, Neyman J. Tests of certain linear hypotheses and their applications to some educational problems. *Statistical Research Memoirs*. 1936; 1:57–93.
- Knafo A, Israel A, Ebstein RP. Heritability of children's prosocial behavior and differential susceptibility to parenting by variation in the dopamine receptor D4 gene. *Development and Psychopathology*. 2011; 23:53–67. [PubMed: 21262039]
- Kwong MJ, Bartholomew K, Henderson AJZ, Trinkle SJ. The intergenerational transmission of relationship violence. *Journal of Family Psychology*. 2003; 17:288–301. [PubMed: 14562454]
- Lewis SF, Fremouw W. Dating violence: A critical review of the literature. *Clinical Psychology Review*. 2001; 21:105–127. [PubMed: 11148892]
- McGruder, B.; Lorenz, FO.; Hoyt, D.; Ge, XJ.; Montague, R. Dimensions of parenting: A technical report. Ames, IA: Iowa State University, Center for Family Research in Rural Mental Health; 1992.
- Melby, J.; Conger, R.; Book, R.; Rueter, M.; Lucy, L.; Repinski, D.; Ahrens, K.; Black, D., et al. The Iowa family interaction rating scales. 2nd. Unpublished manuscript, Iowa State University: Center for Family Research in Rural Mental Health; 1990.
- Moffitt TE. The new look of behavioral genetics in developmental psychopathology: Gene-environment interplay in antisocial behavior. *Psychological Bulletin*. 2005; 131:533–554. [PubMed: 16060801]
- Philibert RA, Gunter TD, Beach SRH, Brody GH, Hollenbeck N, Andersen A, Adams W. Role of GABRA2 on risk for alcohol, nicotine, and cannabis dependence in the Iowa Adoption Studies. *Psychiatric Genetics*. 2009; 19:91–98. [PubMed: 19672139]
- Rutter M, Moffitt TE, Caspi A. Gene-environment interplay and psychopathology: Multiple varieties but real effects. *Journal of Child Psychology and Psychiatry*. 2006; 47:226–251. [PubMed: 16492258]
- Shanahan MJ, Vaisey S, Erickson LD, Smolen A. Environmental contingencies and genetic propensities: Social capital, educational continuation, and Dopamine Receptor Gene DRD2. *American Journal of Sociology*. 2008; 114:S260–S286.
- Shanahan MJ, Hofer SM. Social context in gene-environment interactions: Retrospect and prospect. *Journals of Gerontology: SERIES B*. 2005; 60:65–76.
- Shanahan, MJ.; Hofer, SM. Molecular genetics, aging, and well-being: Sensitive period, accumulation, and pathway models. In: Binstock, RH.; George, LK., editors. *Handbook of Aging and the Social Sciences*. New York: Elsevier; 2011. p. 135-148.
- Simons LG, Simons RL, Lei M-K. Parental Warmth Amplifies the Negative Effect of Parental Hostility on Dating Violence. *Journal of Interpersonal Violence*. in press
- Simons RL, Lei M-K, Brody GH, Beach SRH, Philibert RA, Gibbons FX. Social environmental variation, plasticity genes, and aggression: Evidence for the differential susceptibility hypothesis. *American Sociological Review*. 2011; 76:883–912.
- Simons RL, Lei M-K, Stewart EA, Beach SRH, Philibert RA, Gibbons FX. Social adversity, genetic variation, street code, and aggression: A genetically informed model of violent behavior. *Youth Violence and Juvenile Justice*. 2012; 10:3–24. [PubMed: 23785260]
- Soyka M, Preuss UW, Hesselbrock V, Zill P, Koller G, Bondy B. GABA-A2 receptor subunit gene (GABRA2) polymorphisms and risk for alcohol dependence. *Journal of Psychiatric Research*. 2008; 42:184–191. [PubMed: 17207817]
- Straus, MA.; Gelles, RJ. *Physical violence in American families*. New Brunswick, NJ: Transaction; 1990.
- Tschann JM, Pasch LA, Flores E, Marin BV, Baisch EM, Wibbelsman CJ. Nonviolent aspects of interparental conflict and dating violence among adolescents. *Journal of Family Issues*. 2009; 30:295–319.

- Villafuerte S, Heitzeg MM, Foley S, Yau W-YW, Majczenko K, Zubieta J-K, Zucker RA, Burmeister M. Impulsiveness and insula activation during reward anticipation are associated with genetic variants in GABRA2 in a family sample enriched for alcoholism. *Molecular Psychiatry*. 2011 Advance online public.
- Wilson, WJ. The woes of the African American inner-city father. In: Clayton, O.; Mincy, RB.; Blankenhorn, D., editors. *Black fathers in contemporary American society*. New York, NY: Sage; 2003. p. 9-29.

Figure 1.
Linkage Disequilibrium (LD) of GABRA2 in the FACHS African American Respondents

Figure 2. The effect of observer ratings of harsh parenting on hostility toward romantic partner by rs279837 with Johnson-Neyman 95% confidence bands. The gray areas are significant confidence regions.

Figure 3.
The effect of observer ratings of harsh parenting on hostility toward romantic partner by rs279837

Figure 4.

The effect of observer ratings of harsh parenting on hostility toward romantic partner by number of genetic plasticity alleles with Johnson-Neyman 95% confidence bands. The gray areas are significant confidence regions.

Table 1

Correlation Matrix for the Study Variables.

	1	2	3	4	5	6	7	8	9	10	11
1. Observer ratings of harsh parenting	—										
2. Hostility toward romantic partner	.134 *	—									
3. GABRA2: rs531460	.077	.058	—								
4. GABRA2: rs567926	.065	.040	.943 **	—							
5. GABRA2: rs279858	.039	.038	.950 **	.942 **	—						
6. GABRA2: rs1440130	.097	-.012	.699 **	.718 **	.730 **	—					
7. GABRA2: rs279837	.082	-.029	.794 **	.784 **	.827 **	.864 **	—				
8. Male	.039	-.093	-.005	-.046	-.023	.012	-.052	—			
9. South	.259 **	-.071	-.029	.007	.004	.143 *	.060	.001	—		
10. Family SES	-.269 **	-.097	-.062	.005	-.019	-.115 †	-.040	.014	-.181 **	—	
11. Single family status	.042	.030	.079	.087	.075	.072	.097	-.079	.077	-.326 **	—
Mean	0	6.587	.433	.465	.450	.535	.479	.39	.521	.044	.523
SD	1	1.746	.497	.500	.499	.500	.501	.49	.501	1.437	.499

p .01

*
p .05 (two-tailed tests); n = 242

Table 2
Observer Ratings of Parental Hostility and GABRA2 SNPs as Predictors of Hostility toward Romantic Partner.

	Model 1a	Model 1b	Model 2a	Model 2b	Model 3a	Model 3b	Model 4a	Model 4b	Model 5a	Model 5b
	UnStand. b	UnStand. b	UnStand. b	UnStand. b	UnStand. b	UnStand. b	UnStand. b	UnStand. b	UnStand. b	UnStand. b
Intercept	6.920 *	6.924 *	6.907 *	6.903 *	6.908 *	6.912 *	6.977 *	6.977 *	7.015 *	7.007 *
Environment and Genetic Variables										
Observer ratings of harsh parenting (E)	.260 *	.026	.256 *	.019	.257 *	-.018	.261 *	-.081	.265 *	-.089
GABRA2-Block1: rs531460	.134	.133								
GABRA2-Block1: rs567926			.098	.097						
GABRA2-Block1: rs279858					.103	.105				
GABRA2-Block2: rs1440130							-.060	-.035		
GABRA2-Block2: rs279837									-.149	-.147
Gene-Environment Interaction										
E × rs531460		.497 *								
E × rs567926				.471 *						
E × rs279858						.576 *				
E × rs 1440130										
E × rs279837								.598 *		.691 *
R-Square	.049	.069	.046	.064	.046	.073	.046	.073	.047	.085
R-Square increase due to interaction		.020 *		.018 *		.027 *		.027 *		.038 *
Simple slope test										
Plasticity allele		.523 *		.489 *		.558 *		.517 *		.602 *
Non-plasticity allele		.026		.019		-.018		.645		-.089

* Adjusted FDR p .05

Note: Unstandardized coefficients are shown with robust standard errors in parentheses; parenting practices is standardized by z-transformation (mean = 0 and SD = 1); Gender, area family SES, and family structure are controlled in the analyses; n=242.

Table 3
Two-Way ANCOVA of Hostility toward Romantic Partners by Harsh Parenting $\times$ GABRA2 SNPs

Harsh Parenting	Main Effects				Estimated marginal means						Interaction Effects	
	Harsh Parenting (E)		Genes (G)		Major rallele						G $\times$ E	
	F (df)	FDR p-value	F (df)	FDR p-value	HP-L	HP-M	HP-H	HP-L	HP-M	HP-H	F (df)	FDR p-value
Block 1												
rs531460	2.879 (2)	.128	.465 (1)	.496	5.700	6.597	7.567	7.321	6.337	6.799	4.420 (2)	.013
rs567926	2.446 (2)	.128	1.081 (1)	.476	5.723	6.586	7.434	7.524	6.327	6.794	4.972 (2)	.010
rs279858	2.689 (2)	.128	.771 (1)	.476	5.657	6.566	7.560	7.468	6.347	6.724	5.459 (2)	.008
Block 2												
rs1440130	1.725 (2)	.181	1.110 (1)	.476	5.696	6.421	7.529	7.692	6.449	6.434	6.523 (2)	.005
rs279837	2.309 (2)	.128	1.048 (1)	.476	6.565	6.323	7.635	7.460	6.537	6.496	6.454 (2)	.005

Note: adjusted for gender, area, family SES, and family structure; n = 242. HP-L: Low harsh parenting; HP-M: middle harsh parenting; HP-H: high harsh parenting.