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The Association Between Genetic Predisposition and Parental Socialization: An Examination of Gene–Environment Correlations Using an Adoption-Based Design

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Abstract

An extensive body of research has examined the role that genetic influences play in the development of antisocial behavior. Even so, there remains much that is unknown regarding the intersections among antisocial behavior, environments, and genetic influences. The current study is designed to shed some light on this issue by examining whether gene–environment correlations are present in the lives of adopted adolescents. More specifically, this article seeks to contribute to scholarship efforts aimed at understanding whether biological parents' antisocial behavioral phenotypes—behaviors often attributed to an increased likelihood of receiving a genetic propensity for antisocial behaviors—predict variation in environments that are experienced by their adopted-away offspring. To do so, the biological parents of adoptees were assessed and used to identify ways in which children elicit certain responses from their adoptive parents based, in part, on their genotype. Correlational analyses were calculated on a sample of adoptees (the final analytic sample ranged between $n = 229$ and $n = 293$) drawn from the National Longitudinal Study of Adolescent to Adult Health (Add Health). The results of the study revealed very little evidence of gene–environment correlations. The implications of these findings are considered.

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Introduction

There has been a considerable amount of curiosity regarding the role that genetic influences play in the development of antisocial behavior during the past 20 years (Connolly, Schwartz, Nedelec, Beaver, & Barnes, 2015; Miller & Barnes, 2013). Much of this research has focused on the percentage of variance in antisocial behavior that is accounted for by genetic factors, the identification of specific genetic polymorphisms that might account for phenotypic variance, or the ways in which genes and environments might interact to produce behavioral variation (Barnes, Beaver, & Boutwell, 2011; Boisvert, Boutwell, Vaske, & Newsome, 2014; Cadoret et al., 2003; Harden, Hill, Turkheimer, & Emery, 2008). These studies have produced a significant number of findings that have added to the biosocial understanding of the development of criminal behavior (Ferguson, 2010; Polderman et al., 2015; Rhee & Waldman, 2002). Even so, there remains much that is unknown and unexplored regarding the biosocial influence on criminal behavior.

Therefore, there is much interest in determining the role—if any—that genes play in structuring exposure to certain environments; however, most of this interest is met with empirical uncertainty. Environmental variation is not typically modeled directly, at least not in most social science research. There is reason to believe, however, that genetic propensities might account for variation in most of the environments that are salient to human development (Moffitt, 2005). For example, Kendler & Baker (2007) examined 55 studies that estimated the heritability of different environments, including parenting behaviors, family environment, peer interactions, and stressful life events. Their review illustrated the significance of genetic influences on environments. They concluded that most, if not all, of the environments they surveyed were genetically influenced (Kendler & Baker, 2007). This is a particularly noteworthy possibility for two reasons. First, rather than assuming that environmental variation is random, understanding the genetic architecture to environments can provide insight into how and why environments are distributed across people. Second, if environmental variation is partially the result of genetic variation, then statistical models will have to account for this possibility when attempting to estimate the influence of environment effects on phenotypic variation. The current study seeks to address the link between genetic variation and environmental variation by analyzing data drawn from a longitudinal sample of adoptees.

Gene–Environment Correlations

Although there is a significant amount of research examining the extent to which genetic factors matter for phenotypic variance (Moffitt, 2005), there is comparatively less research exploring the genetic influences on environmental variation. The studies

that do exist tend to converge on the finding that genes have some influence on differential exposure to environments, pointing to the importance of examining both genetic and environmental influences to make sense of how they operate together (Barnes & Jacobs, 2013; Brendgen, 2014). To understand these findings, and the mechanisms that create and shape environments, researchers point to the concept of gene–environment correlations (*r*GEs). There are three types of *r*GEs: passive *r*GE, active *r*GE, and evocative *r*GE, each of which describes a different mechanism that can account for the covariance between genes and environments (Jaffee & Price, 2007).

Passive *r*GE captures the process that accounts for why a child's genotype is often correlated with the rearing environment into which they are born. All nonadopted children receive two sources of influence from their biological parents: a genotype and an environment. Thus, predispositions and early-life family environments are likely correlated because they derive from the same source—that is, biological parents. As an example, children born into homes with parents who are both highly aggressive and antisocial are significantly more likely to be exposed to an abusive and neglectful environment, and, at the same time, are at risk of possessing genetic predispositions for antisocial phenotypes. Given the association between a child's genotype and their rearing environment, it is expected that the child's environment (i.e., abuse and neglect) is correlated with genetic predisposition (i.e., displaying antisocial behaviors; Beaver, 2016).

There is a limited amount of research that has directly tested for passive *r*GE. In most cases, studies compare genetically related and genetically unrelated parent–child groups using an adoption-based approach. In doing so, they are able to determine whether the genetic risk for antisocial behavior is correlated with the rearing environment. For instance, Harold & colleagues (2013) used adoption-based research designs to test for the presence of passive *r*GE by identifying the individual roles that interparental conflict and parent-to-child hostility play in the development of negative child outcomes (e.g., violent behavior and destruction of property). They controlled for the effects of genetics by including biologically related and unrelated mother–child and father–child groups. The initial analysis suggested that there was a significant association between interparental conflict, hostile parenting, and behavioral outcomes among the genetically related group, but not for the genetically unrelated adoption sample. Further examination revealed that genetically unrelated parent–child groupings displayed similar significant associations, but that the relationships among the variables were stronger for genetically related and unrelated fathers compared with genetically related and unrelated mothers. Additional studies have been conducted to examine the contribution of passive *r*GE to the association between familial risk factors and negative child behavioral outcomes (Bornovalova et al., 2014; O'Connor, Caspi, DeFries, & Plomin, 2000). These findings indicate that a mix of environmental effects and passive *r*GE effects account for the relationships among family conflict, parenting behaviors, and child outcomes.

The second type of *r*GE is known as an active *r*GE. Active *r*GEs can be thought of as "genetic niche-picking" and occur when genotypes influence the selection of environments. Genes, of course, do not directly code for choosing one environment over

another, but rather the genetic effect on the selection of an environment operates indirectly via personality traits, such as sensation seeking, and impulse control problems. These traits, in turn, are partially responsible for selecting environments.

Previous research that has tested for active *r*GE has found some support in favor of it (Fu, Nowak, Christakis, & Fowler, 2012; McPherson, Smith-Lovin, & Cook, 2001). One of the more explored *r*GEs in criminological research is self-selection into adolescent peer networks. These networks tend to have relatively high levels of homophily (Beaver, Wright, & DeLisi, 2008). The question, of course, is whether the selection into environments (in this case, peer groups) is influenced by genetic variation. Multiple lines of evidence converge to suggest that this is indeed the case. For instance, twin-based studies have shown that measures of peer groups, including delinquent peer groups, are highly heritable (Beaver et al., 2008). In one study, for example, Cleveland, Wiebe, & Rowe (2005) analyzed a sample of twin pairs and found that 64% of the variance was due to genetics. In a more recent study, Schwartz, Solomon, & Valgardson (2019) reported a sizable *r*GE between peer deviance and self-reported delinquency ($r\text{GE} = .73$), thus indicating that a significant portion of the covariance between peer deviance and self-reported delinquency is explained by correlated genetic influences. Other studies have reported similar results (Loehlin, 2010; Willis & Carey, 2013).

A relatively small number of studies have also examined whether specific genetic polymorphisms are related to delinquent peer group affiliation (Lu & Menard, 2016; Vaughn, DeLisi, Beaver, & Wright, 2009; Yun, Cheong, & Walsh, 2011). In one study, Christakis & Fowler (2014) conducted one of the first genomewide analyses of correlations in genotypes between friends and identified certain patterns across the whole genome that can be used to explain assortment into friendships. To do this, they observed overall homophily within pairs of friends by calculating the probability that two alleles sampled at random from two individuals will be identical. Their results showed that pairs of friends are, on average, as genetically similar to one another as fourth cousins. More specifically, they found that friends significantly resemble each other genotypically, noting that the subtle process of genetic sorting in human social relationships might be the result of an active *r*GE (Christakis & Fowler, 2014). Similarly, in another study, Beaver & colleagues (2008) examined whether a polymorphism in the *DAT1* gene was related to exposure to delinquent peers. Their analysis revealed that the gene was associated with delinquent peer affiliation for male adolescents from high-risk environments. Taken together, genetic association studies and twin-based research converge to provide at least some evidence of an active *r*GE when it comes to the formation and selection of delinquent peer groups.

The final type of *r*GE is evocative *r*GE. According to the logic of evocative *r*GE, individuals elicit certain responses from their environments based, in part, on their genotype. These environmental responses are, in turn, correlated with their genotype. For example, a child with criminogenic traits—traits which have been found to be heritable (Beaver, 2011)—is more likely to evoke harsh discipline from their parents and be excluded by their peers when compared with a child who does not possess criminogenic traits (Gelhorn et al., 2005). This effect, wherein a genetically influenced phenotype produces an environmental reaction, is the same logic that is used in a

child-effects model (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990). The difference, however, is that child-effect models typically focus on whether certain personality traits or behaviors (not genes per se) create an environmental reaction. With evocative *r*GE, the focus is on identifying the genetic architecture that underlies these personalities and behaviors.

There is a limited, but growing, body of research testing for evocative *r*GEs. The research that does exist identifies the importance of genetically influenced behaviors and their influences on a child's environment (DiLalla, Bersted, & John, 2015; Scarr & McCartney, 1983). For example, in a recent study, DiLalla & DiLalla (2018) investigated whether preschoolers with a genetic risk for rule breaking would be more likely to elicit aggressive behaviors from their playmates and be ranked as more likely to engage in rule-breaking behaviors by their parents. In this study, 5-year-old twins were randomly paired with an unfamiliar, same-age, and same-sex children, and, in this way, the researchers were able to control for chosen environments (passive and active *r*GE). Their analyses showed that children with genetic risk for rule breaking were more likely to have partners who behaved aggressively, suggesting that the genetically influenced characteristics of rule breaking evoked aggressive responses in same-age peers. This finding indicates that genes and environments are not independent influences on development (DiLalla & DiLalla, 2018).

Other empirical studies have tested for evocative *r*GE and found that specific genes are correlated with both parenting and child outcomes (Beaver, Shutt, Vaughn, DeLisi, & Wright, 2012; Klahr et al., 2017). For instance, Kryski, Smith, Sheikh, Singh, & Hayden (2014) tested whether links between *OXTR* variation and parenting can be explained, in part, by genetically influenced child emotionality and behavior. Their analyses revealed that, relative to children with at least one G allele, children with two A alleles displayed significantly more negative behavior and, consequently, had caregivers who displayed lower parenting confidence. The child's behavior, then, mediated the relationship between genotype and parenting, suggesting that the effects of *OXTR* genotype on child behavior may be an evocative mechanism. In addition, Burt (2009) explored a possible link between the *–G1438A* gene and the social status of an adolescent. This gene is linked to serotonergic functioning (Knutson et al., 1998) and has been used to predict socially affiliative behaviors; therefore, Burt (2009) suggested that variations in this gene may predispose some adolescents to behave in ways that make them more or less likeable to other students. Analyses revealed that this gene is associated with a predisposition for rule-breaking behavior, but, more specifically, that the students were ranked as more likeable and more popular when they engaged in or reported the highest levels of rule breaking. As evocative *r*GE would predict, the presence of the $5HT_{2A}$ *–G1438A* gene contributed to popularity with others via rule-breaking behaviors (Burt, 2009).

The Current Study

One of the reasons why *r*GEs have not been assessed more consistently in the literature is because they can be difficult to test due to the substantial methodological challenges involved in measuring interactions between genetic and environmental

influences (Moffitt, 2005). To tease out *r*GE interactions, adoption-based research designs are one of the more suitable approaches because they allow researchers to separate the effects of genes and the environment in an effort to measure the heritability of different traits. Adoption-based research designs can broadly test for *r*GEs because adopted children are genetically unrelated to their adoptive parents, and, in this way, it is possible to see whether the phenotypes of the biological parents predict environmental variation in the adopted-away child. Adoptees with limited exposure to their biological parents should only bear resemblance to them as a result of shared genetic material. Conversely, adoptees will resemble their adoptive parents as a result of a shared environment, provided adoptees are not related to their adoptive parents (Beaver, 2011). Therefore, the only reason that the two should be correlated is due to genetic propensities that are found both within the biological parents and their biological (adopted-away) offspring.

Moreover, prior researchers have developed methods for measuring overall assessments of an individual's genetic risk by using the genetic polymorphisms present in their biological parents as indicators of genetic influence on the child's antisocial behaviors (Beaver, Sak, Vaske, & Nilsson, 2010). In this way, it is possible to explore whether evocative *r*GE effects on parenting (i.e., parenting behaviors from adoptive parents) may change depending on the characteristics (i.e., phenotypes) of the biological parent. Children born to parents with higher levels of antisocial behaviors, such as alcoholism or criminality, may display more antisocial phenotypes, as compared with children born to parents with low levels of antisocial behavior (DeLisi, Beaver, Vaughn, & Wright, 2009; Klahr et al., 2017).

Against this backdrop, this article seeks to contribute to scholarship efforts aimed at understanding whether biological parents' antisocial behavioral phenotypes—behaviors often attributed to an increased likelihood of receiving a genetic propensity for antisocial behaviors—predict variation in environments that are experienced by their adopted-away offspring (Beaver et al., 2010; DeLisi et al., 2009). In particular, the current study explores whether the relationship adopted-away children have with their adoptive parents may be genetically influenced via the evocative *r*GE. Moreover, the researchers seek to identify the ways in which children elicit certain responses from their adoptive parents based, in part, on their genotype.

Method

Data

The current study uses data drawn from the National Longitudinal Study of Adolescent to Adult Health (Add Health; Udry, 2003). The Add Health currently consists of four waves of data, with Wave V in progress. The sample originally consisted of students in seventh through 12th grades who were attending 132 schools during the 1994 to 1995 academic year. More than 90,000 students participated in Wave I, which included a wide range of self-reported questions about peers, personal behavior, and family life

(Harris et al., 2003). Approximately, 1.5 years after the in-school surveys were administered, Wave II data were collected, and responses were obtained from 14,197 youth. These adolescents were asked a variety of questions regarding their behavior and social activities, including participation in delinquent behaviors, peer groups, friendships, and romantic relationships. Wave III was completed in 2001 to 2002. Surveys were adjusted to include age-appropriate questions, such as marital status and educational history. In 2007 and 2008, Wave IV was collected from 15,701 respondents (Harris et al., 2003). Respondents, now ranging in ages from 24 to 32 years, were asked about topics pertaining to their mental, physical, and social health, including relationships with their parents, their sexual behaviors, and their involvement in risk-taking conduct.

A unique aspect of the Add Health data is that a subsample of adopted adolescents was nested within the nationally representative sample. During Wave I, youth were asked about their home life and the status of their current guardians. Youth were included in the final analytic subsample if they indicated that they were adopted and if they reported that they did not live with either of their biological parents. These criteria excluded adolescents who may be living with one biological parent or any other biological relative and resulted in a total of $n = 646$ participants. In addition, adoptees and their adopted parents were asked a series of questions regarding the adoptees' biological parents (discussed in more detail below). The final analytic sample was limited to adoptees with valid information on their biological parents and ranged between $n = 229$ (for estimates examining biological father arrest status) and $n = 293$ (for estimates examining biological mother alcoholic status).

We examined differences between the adopted sample and the nonadopted sample for the variables used in our analyses. We found significant differences between the two groups, including that adoptees experienced significantly (a) greater percentage of arrested biological fathers ($\chi^2 = 30.61$, $p < .001$), (b) greater percentage of alcoholic biological fathers ($\chi^2 = 57.32$, $p < .001$), (c) greater percentage of arrested biological mothers ($\chi^2 = 144.00$, $p < .001$), (d) greater percentage of alcoholic biological mothers ($\chi^2 = 310.38$, $p < .001$), (e) greater percentage of males ($\chi^2 = 5.68$, $p = .017$), (f) greater percentage of Caucasians ($\chi^2 = 7.25$, $p = .027$), (g) greater levels of Wave I adopted paternal involvement ($t = 2.59$, $p = .01$), and (h) greater levels of Wave I adopted maternal disengagement ($t = 2.58$, $p = .01$). Researchers generally recognize that being an adoptee is a unique experience (Eldred et al., 1976). Under these circumstances, it is quite possible that the differences observed between the adopted sample and the nonadopted sample are related to our findings and subsequent conclusions; however, these differences may also be valid predictors of our outcome measures simply because of the biological risk present among more of the adoptees in the sample. Given the potential methodological limitations of *r*GE designs, it is particularly important to understand the conditions that lead to such differences among adopted adolescents. Therefore, further research is needed to address the concern that accompanies the significant differences found between the two groups.

Measures

Genetic risk. Adoption studies allow for the analysis of both environmental and genetic effects on human behavior (Kendler et al., 2014). In this analysis, environmental effects are represented by parenting measures of adoptive parents and genetic effects are represented by antisocial behavior in biological parents. To measure variation in genetic risk, primary caregivers were asked questions regarding the health history of the adoptees' biological parents. During Wave I, primary caregivers were asked whether the biological mother or father had ever suffered from alcoholism. Responses were coded as 0 = no and 1 = yes. During Wave IV interviews, the interviewees were asked to indicate whether their biological mother or father had spent time in jail or prison. Responses were coded as 0 = no and 1 = yes. The mean, standard deviation, and range for the genetic risk measures, along with all other study measures, are presented in Table 1.

Maternal attachment. Children who are attached to their mothers are less likely to engage in delinquent behaviors than children who have strained relationships with their mothers (Gottfredson & Hirschi, 1990). To measure individual variation in maternal attachment, youth were asked a series of questions regarding their attachment to their adoptive mothers. Specifically, respondents were asked how close they feel to their mother and their perceptions of how much their mother cares about them. Responses to these items were summed together to create the Wave I maternal attachment index ($\alpha = .70$). For this index, higher scores on the index represent greater levels of maternal attachment. Importantly, this index has been used in previous research (Schreck, Fisher, & Miller, 2004).

Maternal involvement. Prior research has indicated a significant association between maternal–child involvement and antisocial behaviors (Loeber & Stouthamer-Loeber, 1986). The Add Health survey included a list of 10 activities that, when combined, can be used to determine how involved mothers were with their children. Adolescents were asked about their mother's participation in such activities as shopping together or playing sports in the 4 weeks prior to the survey. Responses were coded dichotomously where 0 = did not participate in the activity and 1 = participated in the activity. Responses to the questions were then summed together for the maternal involvement index ($\alpha = .60$). This index has been used in the past (Crosnoe & Elder, 2004).

Maternal disengagement. Children raised by withdrawn or disengaged parents are at an increased risk for delinquent involvement (Loeber & Stouthamer-Loeber, 1986). Therefore, a maternal disengagement index was developed (Beaver, 2008). During Wave I of the study, adolescents were asked to report on the overall quality of their relationship with their mother and the amount of warmth they felt from her. Responses to these questions were summed to form a 5-item maternal disengagement index ($\alpha = .85$), where the higher scores indicate greater maternal disengagement.

Table 1. Univariate Statistics for Study Measures.

	M/%	*SD*	Minimum-Maximum	*n*
Paternal measures				
Biological paternal measures (%)				
Biological father arrested			0-1	229
Yes	27.51	—		63
No	72.49			166
Biological father alcoholic			0-1	228
Yes	31.58	—		72
No	68.42	—		156
Adoptive paternal socialization measures (*M*)				
Paternal attachment	9.07	1.46	2-10	481
Paternal involvement	3.19	2.22	0-10	480
Maternal measures				
Biological maternal measures (%)				
Biological mother arrested			0-1	279
Yes	17.20	—		48
No	82.80	—		231
Biological mother alcoholic			0-1	293
Yes	19.80	—		58
No	80.20	—		235
Adoptive maternal socialization measures (*M*)				
Maternal disengagement	9.37	3.75	5-24	602
Maternal attachment	9.23	1.26	3-10	603
Maternal involvement	4.11	2.12	0-10	602
Adoptive parental socialization measures, mean				
Parental permissiveness	5.18	1.49	0-7	630
Demographic measures				
Age, mean	16.24	1.65	12-20	646
Sex (%)			0-1	646
Male	53.25	—		344
Female	46.75	—		302
Race (%)			1-3	646
Caucasian	65.94	—		426
African American	19.20	—		124
All other races	14.86	—		96

Parental permissiveness. Parental supervision has been found to be one of the most consistent predictors of antisocial behaviors (Gottfredson & Hirschi, 1990). To take this finding into account, a parental permissiveness index was used to analyze the degree of autonomy adolescents were allowed by their parents. At Wave I, adolescents indicated whether they had a say in such decisions as friend choice and bedtimes.

Responses to the 7-item index were coded dichotomously (0 = no, 1 = yes), and summed to (α = .58) where higher values indicate greater levels of parental permissiveness. Responses were summed together to create the parental permissiveness index (α = .57). This index has been used in previous research (Barnes & Morris, 2012).

Paternal attachment. Relatively low levels of paternal attachment have been found to be related to an increase in delinquent behavior (Higgins, Mahoney, & Jennings, 2010). As a result, a paternal attachment index was included in the analysis. This index was developed through responses to questions similar to those in the maternal attachment index. Youth, for instance, were asked how close they felt to their fathers and how much they think their father cares about them. Responses to these items were summed together to create the Wave I paternal attachment index (α = .77). Similar indexes have been used in previous research (Beaver et al., 2014).

Paternal involvement. Findings from research suggest that adolescents who have fathers who are less involved in their daily lives are at an increased risk for antisocial behavior when compared with youth whose fathers take an active role in their lives (Higgins et al., 2010). To incorporate this finding, we included a paternal involvement index in the current study. Much like the index for maternal involvement, a 10-item index was created to measure paternal involvement. Respondents indicated whether their father participated in any of the 10 activities such as shopping or working on school projects in the 4 weeks prior to the survey. Responses were coded dichotomously (0 = no, 1 = yes) and were summed together to create a paternal involvement index (α = .65). Similar indexes have been used in previous research (Beaver et al., 2014).

Analytical plan. In an effort to examine the presence of *r*GE spanning multiple parenting measures, we make use of the adoption subsample of the Add Health to estimate *r*GE in a novel way. More specifically, as the analytic sample is comprised solely of adoptees who were not adopted by a family member, the examined participants do not share any dissenting genetic material with their adoptive parents. In this way, the examined parental socialization measures capture environmental influences from the adoptive parents, net of any genetic influences. Alternatively, as the adoptee sample had no contact with their biological parents, the maternal/paternal arrest and alcoholism measures tap genetic predisposition and are free of environmental sources of influence. Collectively, any correlation between the adoptive parent socialization measures and biological parent predisposition measures would, therefore, represent an *r*GE. Based on this observation, we calculated bivariate correlations between these two sets of variables to more closely examine the presence of *r*GE. In an effort to more closely examine the extent to which maternal and paternal interactions may result in differences in *r*GE, we examined maternal and paternal measures separately. Finally, as the adoptive parent socialization measures were continuous and the biological parent predisposition measures were binary, we estimate point biserial correlation coefficients.

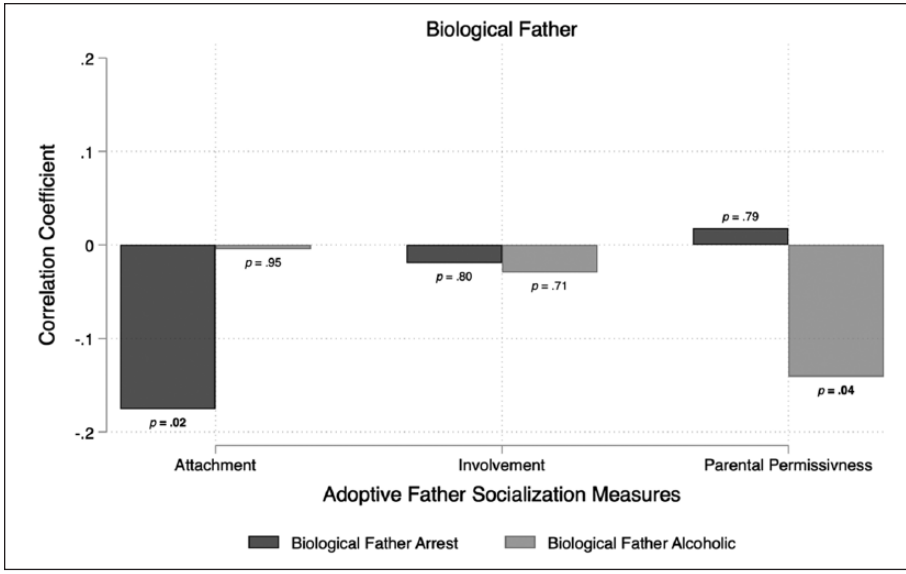

Figure 1. Biserial correlation coefficients for adoptive father socialization measures and biological father arrest and alcoholic measures.
Note. Presented bars represent biserial correlation coefficients and accompanying *p* values are presented.

Results

We begin our analysis by examining the potential *r*GE between the two biological paternal predisposition measures and the three adoptive paternal socialization measures from Wave I, with the results presented in Figure 1.[1] The figure presents correlations between the biological paternal arrest measure and the adoptive paternal socialization measures. As can be seen in the figure, lower levels of paternal attachment at Wave I were significantly correlated with paternal arrest ($r = –.18$, $p = .02$). Lower levels of paternal involvement at Wave I were also correlated with paternal arrest, but the resulting association was nonsignificant ($r = –.02$, $p = .80$). Greater levels of paternal permissiveness at Wave I was correlated with paternal arrest, but, again, the correlation was nonsignificant ($r = .02$, $p = .79$).

Figure 1 also presents the results of the correlations between the paternal socialization measures and the paternal alcoholism measure. Lower levels of paternal attachment ($r = –.00$, $p = .95$) and paternal involvement ($r = –.03$, $p = .71$) at Wave I were associated with paternal alcoholism, but only the association involving paternal permissiveness was statistically significant. In addition, lower levels of parental permissiveness ($r = –.14$, $p = .04$) was significantly associated with paternal alcoholism.

We also estimated the influence that the two biological paternal predisposition measures have on the three adoptive maternal socialization measures from Wave I, with the results presented in Figure 2. The figure first presents correlations between

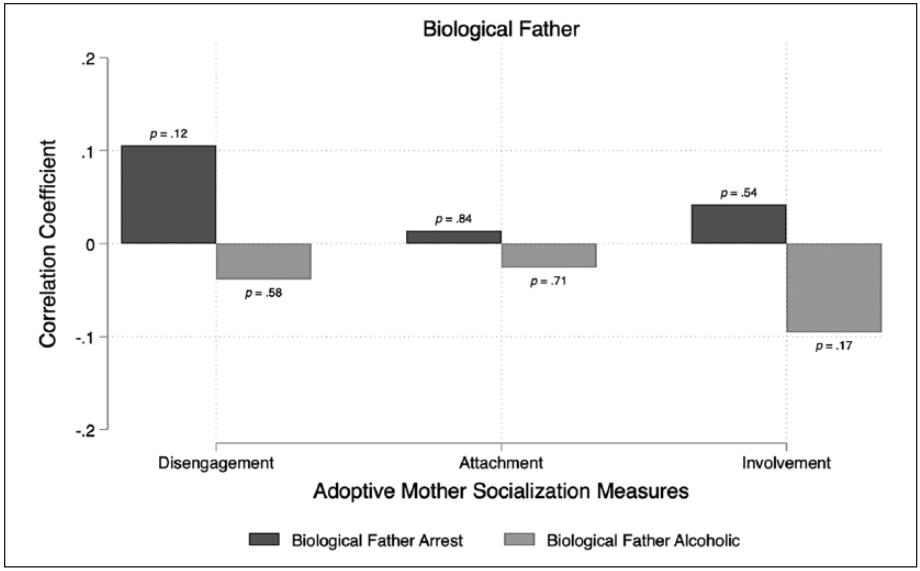

Figure 2. Biserial correlation coefficients for adoptive mother socialization measures and biological father arrest and alcoholic measures.
Note. Presented bars represent biserial correlation coefficients and accompanying *p* values are presented.

the biological paternal arrest measures and the adoptive maternal socialization measures. Overall, higher levels of maternal disengagement ($r = .11$, $p = .12$), attachment ($r = .01$, $p = .84$), and involvement ($r = .04$, $p = .54$) at Wave I were correlated with paternal arrest, but the resulting associations were nonsignificant.

Figure 2 also presents the correlations between the maternal socialization measures and the paternal alcoholism measures. The analysis reveals that lower levels of maternal disengagement ($r = –.03$, $p = .58$), attachment ($r = –.03$, $p = .71$), and involvement ($r = –.10$, $p = .17$) at Wave I were correlated with paternal alcoholism, but the resulting associations were nonsignificant.

The results for *r*GEs involving adoptive maternal socialization measures and biological maternal predisposition measures are presented in Figure 3. First, the figure presents the results of *r*GEs involving the maternal socialization measures and the maternal arrest measures. As can be seen in the figure, lower levels of maternal disengagement ($r = –.01$, $p = .89$) and lower levels of maternal attachment ($r = –.04$, $p = .55$) at Wave I were associated with an increased likelihood of maternal arrest, but none of the examined associations were significant. However, we also observed that greater levels of maternal involvement ($r = .01$, $p = .91$) at Wave I were correlated with maternal arrest, but again, the resulting correlation was not significant. In addition, lower levels of parental permissiveness ($r = –.07$, $p = .24$) was not significantly associated with maternal arrest.

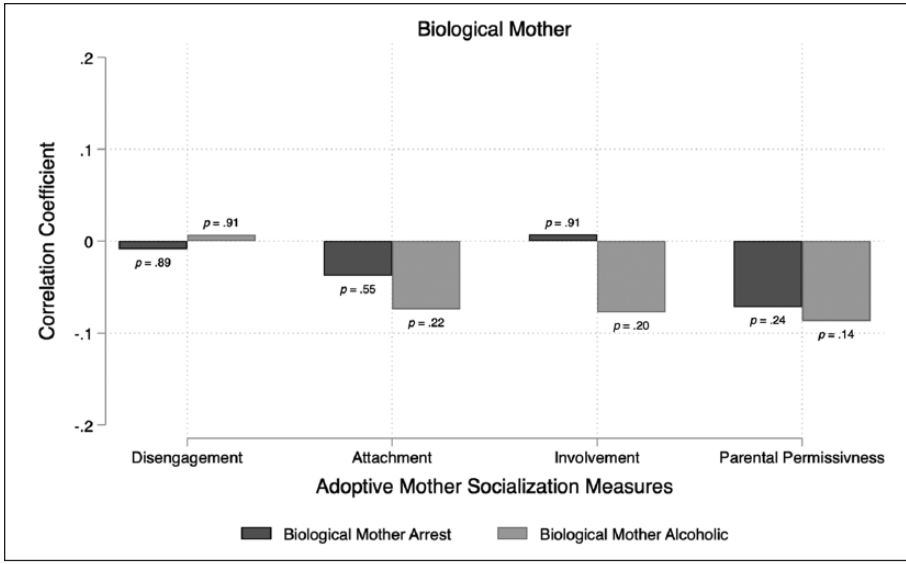

Figure 3. Biserial correlation coefficients for adoptive mother socialization measures and biological mother arrest and alcoholic measures.
Note. Presented bars represent biserial correlation coefficients and accompanying *p* values are presented.

The results for the *r*GEs involving the adoptive maternal socialization measures and the biological maternal alcoholism predisposition measures are also presented in Figure 2. Among the Wave I maternal socialization measures, greater levels of maternal disengagement ($r = .01$, $p = .91$), lower levels of maternal attachment ($r = –.07$, $p = .22$), and lower levels of maternal involvement ($r = –.08$, $p = .20$) were associated with maternal alcoholism, but the resulting correlation coefficients were nonsignificant. We also observed that lower levels of parental permissiveness ($r = –.09$, $p = .14$) were associated with maternal alcoholism, but the resulting correlation coefficient was nonsignificant.

Finally, we estimated the influence that the two biological maternal predisposition measures have on the three adoptive paternal socialization measures from Wave I, with the results presented in Figure 4. The figure first presents correlations between the biological maternal arrest measure and the adoptive paternal socialization measures. The figure demonstrates that greater levels of paternal attachment ($r = .00$, $p = .99$) and greater levels of paternal involvement ($r = .01$, $p = .87$) were associated with maternal arrest, but the resulting correlation coefficients were nonsignificant.

Figure 4 also presents the correlations between the paternal socialization measures and the maternal alcoholism measures. The analysis reveals that greater levels of paternal attachment ($r = .07$ $p = .31$) and involvement ($r = .01$, $p = .87$) at Wave I were correlated with maternal alcoholism, but the resulting associations were nonsignificant.[2]

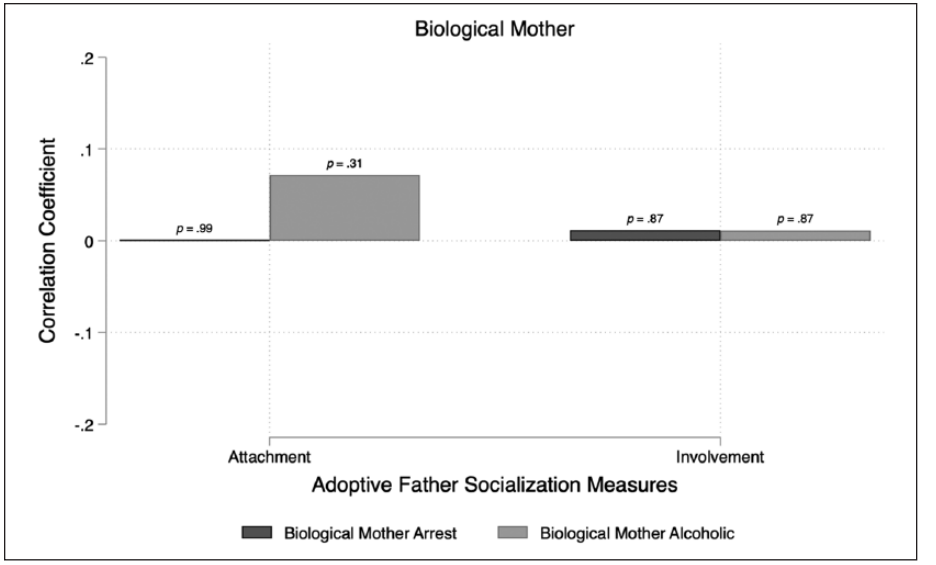

Figure 4. Biserial correlation coefficients for adoptive father socialization measures and biological mother arrest and alcoholic measures.
Note. Presented bars represent biserial correlation coefficients and accompanying *p* values are presented.

Discussion

During the past 20 years, there has been a considerable amount of research dedicated to identifying the role that genetic influences play in the development of antisocial behavior (Connolly et al., 2015; Miller & Barnes, 2013). Despite the methodological challenges involved in *r*GE research, it is worth testing theories that utilize *r*GE perspectives, for where measured *r*GE interactions are found to influence behavior outcomes, both specific genes and environments have more significant effects, as opposed to the traditional smaller effects from individual environmental interactions (Moffitt, 2005). Research that examines how environments work together with genetic factors reveals that genetic influences can explain approximately half of the variance in human phenotypes, including antisocial behavior (Ferguson, 2010; Rhee & Waldman, 2002). Much less emphasis has been placed on the potential role that genes play in creating environmental variance, including criminogenic environments. The limited research in this area has found that virtually every environment ever studied is partially shaped by genetic influences (Jaffee & Price, 2007; Kendler & Baker, 2007).

The current study added to this existing body of literature by analyzing a sample of adoptees to estimate the influence of genetic factors on a range of parenting measures. In contrast to much of the existing literature, most of the analyses revealed relatively little support for *r*GEs. Although it was demonstrated that both genetic and environmental influences from a child's biological father should be considered when examining the etiology of antisocial behaviors, interestingly, the presence of antisocial behaviors in the

biological mother was shown to be unrelated to the relationship that the genetically at-risk children had with their adoptive parents. Although the findings did not provide additional support for the role that genes inherited from a child's biological mother play in structuring exposure to certain environments, this finding is instead in line with research that suggests that having an antisocial biological father has more of an influence on the development of antisocial personality traits (Beaver, Rowland, Schwartz, & Nedelec, 2011). The question, of course, is why the findings from the current study are so different from those generated in other *r*GE studies.

While purely speculative, we do point out that much of the evidence for *r*GEs has been derived from studies that decompose environmental variance using a twin-based methodology (Beaver, 2016; Kendler & Baker, 2007). The current findings were based on an adoption research design wherein subjects were asked to report on their biological parents' criminality (in Wave IV) and their adoptive caregivers were asked to report on the biological parents' alcoholism (in Wave I). Given that the interviewees might not be knowledgeable about either of the biological parents' criminality or their alcoholism, using the approach may result in increased levels of error in the genetic measures of criminality. If that is the case, then this error would likely attenuate the *r*GE estimates. Future research is needed to more fully determine whether this is the reason for the null findings or whether, in fact, in these data the *r*GEs are relatively sparse.

Moreover, our analyses are limited, in that they are only capturing the effect of genetic influences on environments among our sample of adolescents and young adults. From what is known about child-driven effects on parenting, in that children may evoke reactions from others that are consistent with their genetic predispositions, it is possible that younger children may be more likely to evoke harsh reactions simply because they are at home more than they are out with their peers or participating in extracurricular activities (Klahr et al., 2017; Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983). To illustrate, when researchers explored the hypothesis that younger children with genetic risk evoke more negative parenting than older children with genetic risk, O'Connor, Deater-Deckard, Fulker, Rutter, and Plomin (1998) found a significant age effect. Their analyses revealed that parents reported significantly more negative control when the children were at age 7 than at ages 9, 10, 11, and 12 (O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998). As a result, our finding may be null because of this suggested age effect. Therefore, future research is needed to address this possibility.

Even though most of the analyses revealed null effects for the *r*GEs, there are two statistically significant results that are worthy of additional consideration. First, adoptees who were at genetic risk based on their biological father's criminality were more likely to feel less attached to their adoptive father. Therefore, the criminogenic nature of an adopted child's biological father may have influenced their relationship with their adoptive father. This child-driven effect on parenting may function via evocative *r*GE based on the child's genetic predispositions. It is unclear how parent characteristics and child genetic risk interact, but this finding suggests that adoptive fathers' behaviors toward their children can be in reaction to their child's genetically influenced antisocial

behaviors. It is possible that the genetic risk associated with criminal biological fathers of adoptees is eliciting responses from their adoptive environment and affecting the relationship they have with their adopted father in a way that supports prior research on evocative *r*GE (Scarr & McCartney, 1983). However, as with all correlational findings, it may also be the case that the relationship the child has with the adoptive father can work to both suppress and reveal genetic effects. For example, adoptees with genetic risk placed with exceptionally caring adoptive parents may be less likely to be affected by their genetic risk for antisocial behaviors in their adoptive socialization environment. Moreover, because we found evidence of evocative *r*GE, wherein the relationship adoptees felt with their adoptive father was significantly influenced by the criminality of their biological father, further empirical exploration could glean valuable information about possible gender effects, as well as child-driven effects. To illustrate, DeLisi & colleagues (2009) analyzed the precise ways that genetic and environmental pathogens interact to predict antisocial behavior among sons with criminal fathers by demonstrating a significant interaction between genetic predispositions for antisocial behaviors (i.e., biological criminal father) and violent, delinquent behaviors in their sons.

The second main finding to emerge from the analyses was that adoptees who were at genetic risk based on their biological father's alcoholism felt that their adoptive parents allowed for more autonomous decision-making. Therefore, the child's birth father may have affected the relationship they reported with their adoptive parents, thus revealing some evidence for evocative *r*GE interactions. With regard to parenting, the adoptive parents of children with genetic risk factors may find it more difficult to discipline or maintain enforced rules around their child, which may allow the child to feel that they are more in charge of their curfew or peer groups because the child is eliciting certain responses from their parents based, in part, on their antisocial genotype. These findings are consistent with prior *r*GE research that has found that genes are correlated with parenting outcomes (Beaver et al., 2012; Klahr et al., 2017).

The results generated from the current study need to be interpreted with caution due to a number of shortcomings with the analyses. First, this study focused on a sample of adoptees and, therefore, the results may not necessarily generalize to nonadoptees. Future studies are needed that use different methodologies and samples of nonadoptees to determine the robustness and the generalizability of these findings. Second, and relatedly, information regarding adoption was based on self-reports. This necessarily raises the question of whether some adoptees were not included in the final analytic sample because they were unaware that they had been adopted. Unfortunately, the Add Health data do not include any other information regarding adoption. Additional research is needed to more fully determine whether the measurement of adoption in the Add Health would bias the overall results in any significant way. Third, all of the parenting measures were based on self-reports of the adolescent. This measurement strategy obviously raises potential concerns regarding whether these reports are accurate assessments of the type of parenting that they are receiving. Ideally, independent observers would have rated different aspects of parenting or, at the very least, multiple reporting sources would have been used. Unfortunately, the Add Health did not employ these measurement approaches for

parenting. Finally, this study is limited as the analysis was only correlational in nature. Future research is needed to examine whether these findings would be replicated using different measures of parenting, different reporting sources, or different methods of analysis.[3]

Despite these limitations, the current findings indicate that research that explores the interplay between genes and environments, via *r*GEs, can begin to unravel the important influences that genetics have on a child's perceptions, attitudes, and behaviors. Although the literature is limited, the studies that do exist continue to find support for the effect that individual biological differences have on the environment. By advancing the range of variables that are studied, not only through the adoption-based methodology but also through the *r*GE perspective, we will know more about how genetic and environmental influences intersect with parenting, family environments, and child outcomes.

Appendix A

Results From Multivariate Linear Regression Models Examining the Association Between Adoptive Father Socialization Measures and Biological Father Arrest and Alcoholic Measures.

	Paternal attachment			Paternal involvement			Permissiveness		
	b	*SE*	*n*	*b*	*SE*	*n*	*b*	*SE*	*n*
Arrest									
Biological father	−0.47†	0.26	167	0.11	0.42	167	−0.04	0.23	224
Age	−0.16*	0.07		−0.27*	0.11		0.30*	0.06	
Sex	0.27	0.23		0.26	0.36		−0.15	0.21	
Race									
Caucasian		ref.			ref.			ref.	
African American	−0.25	0.30		−0.60	0.47		−0.41†	0.24	
All other races	0.13	0.34		0.56	0.55		−1.34**	0.33	
Alcoholic			164			163			221
Biological father	−0.08	0.29		−0.19	0.41		−0.39†	0.23	
Age	−0.13	0.09		−0.19	0.12		0.25**	0.07	
Sex	−0.13	0.28		0.48	0.40		0.16	0.22	
Race									
Caucasian		ref.			ref.			ref.	
African American	−0.56	0.37		−0.46	0.52		−0.20	0.27	
All other races	−0.07	0.46		0.62	0.65		−0.73*	0.37	

Note. Unstandardized regression coefficients and accompanying standard errors (*SE*) presented.
†$p < .10$. *$p < .05$. **$p < .001$.

Appendix B

Results From Multivariate Linear Regression Models Examining the Association Between Adoptive Mother Socialization Measures and Biological Mother Arrest and Alcoholic Measures.

	Maternal disengagement			Maternal attachment			Maternal involvement			Permissiveness		
	b	*SE*	*n*	*b*	*SE*	*n*	*b*	*SE*	*n*	*b*	*SE*	*n*
Arrest												
Biological mother	0.21	0.63	272	−0.22	0.22	273	0.07	0.36	272	−0.16	0.24	284
Age	0.51**	0.14		−0.14*	0.05		−0.14†	0.08		0.28**	0.05	
Sex	−0.71	0.47		0.12	0.16		−0.63*	0.27		−0.01	0.18	
Race												
Caucasian		ref.			ref.			ref.			ref.	
African American	−0.38	0.57		0.10	0.20		−0.30	0.33		−0.39	0.22	
All other races	1.39†	0.73		−0.61*	0.25		−0.21	0.42		−1.14**	0.28	
Alcoholic												
Biological mother	0.24	0.50	257	−0.25	0.17	257	−0.53	0.34	257	−0.25	0.23	273
Age	0.40**	0.12		−0.11**	0.04		−0.13	0.08		0.25**	0.06	
Sex	−0.27	0.40		0.08	0.13		−0.43	0.27		−0.11	0.19	
Race												
Caucasian		ref.			ref.			ref.			ref.	
African American	0.06	0.48		−0.03	0.16		−0.24	0.32		−0.30	0.22	
All other races	−0.72	0.72		−0.07	0.24		0.14	0.49		−0.72**	0.32	

Note. Unstandardized regression coefficients and accompanying standard errors (*SE*) presented.
†$p < .10$. *$p < .05$. **$p < .001$.

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Notes

1. We reestimated all of our models with the same measures from Wave II.
2. Pairwise correlation was used for correlations. Listwise deletion was used for regression models. We reestimated all of the models using full information maximum likelihood (FIML) estimation, which takes all available information into account to address missing values and has been found to be a highly efficient and effective technique in addressing missing data. The results of these supplemental analyses were virtually identical to those from the primary analyses.
3. A reviewer made the important point that we are only running bivariate models, thus our results can be confounded by extraneous factors. Given that, by definition, gene–environment correlations (*r*GEs) are correlations, we opted to retain our primary analyses. In this way, our bivariate models should be an appropriate test of *r*GEs. At the same time, however, we do recognize that there could be important advancements gained by looking at our variables in a multivariate fashion. Therefore, we have included replications of our primary analyses in multivariate models that control for age, sex, and race. More specifically, we regressed our main genetic influence variables (i.e., biological father arrested, biological father alcoholic, biological mother arrested, and biological mother alcoholic) on our parental socialization outcome measures (i.e., paternal attachment, maternal attachment, paternal involvement, maternal involvement, paternal permissiveness, maternal permissiveness, and maternal disengagement), while taking into account variance in sex, age, and race among the respondents. We have presented these additional multivariate models as tables in Appendix A and Appendix B. Although our correlational analyses revealed that there was a significant association between paternal alcoholism and parental permissiveness, as well as paternal arrest and paternal attachment, both of these results were null in the multivariate analysis. Future research is needed to explore the effects of sex, age, and race, as well as other key characteristics, on *r*GEs.

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