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Gene–Environment Correlations Affecting Children's Early Rule-Breaking and Aggressive Play Behaviors

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Play among peers is an important developmental context for child socialization. We have earlier shown that children at genetic risk for aggression were more likely to be treated aggressively by unfamiliar peers during peer play, reflecting genotype-evoked behaviors manifested during play. In this study, 118 5-year-old twin pairs were paired randomly with an unfamiliar, same-age, same-sex child, thus controlling for parent- and child-chosen environments (passive and active rGE). Twins played separately from each other with unmatched children. Play behaviors were coded for aggressive and assertive behaviors. Children were also independently rated by parents for rule-breaking problem behaviors at age 5, and 97 children were rated again on these behaviors 2–10 years later. Analyses showed that children at genetic risk for early rule-breaking were more likely to have partners who behaved more aggressively, suggesting that this was evoked behavior during play. Some evidence of an 'early bloomer' phenomenon emerged via early difficult temperament and parent-rated delinquency significantly predicting later delinquency. Children's play, which is one of the most important influences on early development, requires further study from an rGE perspective.

■ **Keywords:** Irving I. Gottesman, gene–environment correlations, children's play

The symposium from which this paper was derived honored the life and contributions of Irving I. Gottesman. Gottesman was an iconoclast. He began his scholarly career in the early 1960s, when psychology leaned almost exclusively toward the 'nurture' side of the nature-nurture divide. Gottesman's work challenged conventional thinking. The impact of his life's scholarly work is demonstrated by the observation that notions that were initially controversial as genetic influence on behavior are now accepted wisdom. But Gottesman also recognized and demonstrated through his work that simple explanations of genetic influence on behavior are not viable. In this article, we illustrate this by focusing on pathways leading to child aggression and delinquency.

In one of the early publications attempting to tie together genes, environment, and delinquent behavior, DiLalla and Gottesman (1989) postulated three groups of delinquents or criminals: transitory delinquents, who engaged in delinquent behaviors as youth but grew out of this behavior in adulthood; continuous antisocials, who engaged in adolescent delinquent behaviors and continued these behaviors into adulthood; and late bloomers, who did not engage in illegal behaviors until adulthood. DiLalla and Gottesman (1989) suggested that an underlying genetic predisposition contributed to a continuous antisocial presentation, whereas transitory delinquency was a normative adolescent phase that was socially constructed and influenced. Moffitt (1993, 2006) subsequently suggested a neuropsychological mechanism underlying what she called life-course-persistent antisocials, similar to continuous antisocials. Supporting this idea, delinquent adolescents who initiated such behavior at the youngest ages showed a higher heritability for delinquency (Taylor et al., 2000), and temperament, which is biologically based, was shown to distinguish early from later onset delinquency in adolescents (Moffitt & Caspi, 2001).

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As an extension of work with Irving Gottesman, we have studied young twins to explore early child behaviors predictive of aggression and rule-breaking behaviors. In a study of peer play (DiLalla & John, 2014) we demonstrated an evocative gene–environment correlation (rGE) for aggression; children with a genetic risk of behaving aggressively were significantly more likely to elicit aggression from unfamiliar, same-age, same-sex (SS) peers. Because peer pairs were randomly assigned and the children did not choose the friend they played with, we could rule out the presence of active rGE. Additionally, because parents did not choose which child their child would play with, as often occurs when parents of preschoolers arrange play dates for their children or introduce them to the children of their friends, we ruled out effects of passive rGE. Similar results were found with prosocial behaviors in the same paradigm (DiLalla et al., 2015).

For the present twin study, we investigated whether similar reactive rGE affects children's rule-breaking behaviors. We included observations of preschoolers' play interactions with peers, as well as parents' ratings of the preschoolers' temperament and externalizing problem behaviors. We hypothesized that preschoolers with a genetic risk for rule-breaking would be more likely to elicit aggressive behaviors by their playmates, but not assertive (more prosocial) behaviors, and also would be more likely to be rated by their parents as engaging in rule-breaking behaviors as preschoolers and again later at school age.

Materials and Methods

Participants

As part of the Southern Illinois Twins/Triplets and Siblings Study (SITSS; DiLalla, 2002; DiLalla et al., 2013), 118 twin pairs (236 children of complete pairs; 41 monozygotic (MZ) pairs, 52 same-sex (SS) dizygotic (DZ) pairs, 25 opposite-sex (OS) DZ pairs) were tested at age 5. Zygosity was assessed either via buccal cell DNA collection or through questionnaires on similarity completed by one parent and one tester at the time of testing (96% similarity to DNA results; see DiLalla et al., 2013). All families were from rural towns within a two-hour drive of a U.S. Midwestern university. Ninety-five percent of families reported themselves as Caucasian, 1% as African American, and 4% as other. Ninety-seven of the twins (41%) participated in a follow-up study when they were between the ages of 6 and 16 years old; these children were included in the longitudinal analyses.

Procedure and Measures

All children visited the university laboratory within two months of their fifth birthdays. Another child was paired with each of the twins. The paired children had never met prior to the test session, were the same sex, and had birthdays within one month of each other. One twin and one unfamiliar peer were brought to the playroom and videorecorded through a one-way mirror while they played freely for 20 minutes; the other twin and an unfamiliar child played next for 20 minutes. Puppets, action figures, kitchen toys, a tool set, and checkers were available in the playroom. Children were rated on several 5-point Likert-type measures by trained undergraduate and graduate research assistants, including aggression (1 = child showed no aggressive behaviors; 5 = child showed many aggressive behaviors such as teasing, hitting, pushing, grabbing) and assertiveness (1 = child never told other child what to do; 5 = child frequently commanded the other child, directed play, told child what to do). Interrater reliability (Cronbach's alpha) was 0.87 for both measures. Coders rated the first 10 minutes of play for each child and then rated the second 10 minutes of play; the average across both scores was used as the final score.

Prior to visiting the lab, parents completed a series of questionnaires, including the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2000) to assess rule-breaking (internal consistency alpha = 0.62; at ages 6–16, internal consistency alpha = .68). The CBCL was again completed by parents during follow-up testing when the children were between the ages of 6 and 16 years old. Test–retest reliability with age 5 was acceptable, $r(97) = 0.34$, $p = .001$.

Results

A genetic risk index for rule-breaking (GRI-RB) was calculated for each twin based on the co-twin's 5-year-old CBCL rule-breaking score and the twins' degree of genetic relatedness. Boys scored significantly higher than girls on rule-breaking, $t(116) = 2.48$, $p = .015$. Thus, we scored GRI-RB as follows, using the method outlined by Jaffee et al. (2005): GRI-RB = 6 (highest genetic risk) if twins were MZ and co-twin's rule-breaking was in the top 25% of the sample; GRI-RB = 5 if twins were SS DZ and co-twin's rule-breaking was in the top 25%; GRI-RB = 4 if twins were OS DZ and co-twin's rule-breaking was in the top 25%; GRI-RB = 3 if twins were OS DZ and co-twin's rule-breaking was in the bottom 75%; GRI-RB = 2 if twins were SS DZ and co-twin's rule-breaking was in the bottom 75%; GRI-RB = 1 (lowest genetic risk) if twins were MZ and co-twin's rule-breaking was in the bottom 75%. (See DiLalla & John, 2014, or Jaffee et al., 2005, for more details about this method.)

To test whether reactive rGE occurred for preschool children's rule-breaking behaviors, we examined Spearman's correlations between GRI-RB and the play environment; specifically, the other child's aggressive and assertive behaviors. As hypothesized, we found that GRI-RB was significantly correlated with peers' aggressive behaviors, $r(218) = 0.19$, $p = .004$, but not with assertive behaviors, $r(217) = -0.08$, $p = .245$, demonstrating reactive rGE for preschool externalizing behaviors. Results were comparable when OS DZ twins were removed from the analyses.

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TABLE 1

Results From Mixed Effects Models of Rule-Breaking and Difficult Temperament Predicting School-Age Rule-Breaking, Controlling for Shared Genetic Effects

	Estimate (*SE*)	*t* (*p* value)
Fixed effects		
Intercept	-3.17 (2.24)	-1.41 (.165)
Age five rule-breaking (RB) deviation	-1.08 (0.65)	-1.67 (.102)
RB deviation * zygosity (DZ effect — MZ effect)	1.24 (0.73)	1.70 (.096)
RB pair average	0.50 (0.16)	3.22 (.002)
Age five difficult temperament (DT) deviation	-4.39 (2.15)	-2.04 (.047)
DT deviation * zygosity	3.07 (2.33)	1.32 (.193)
DT pair average	1.07 (0.66)	1.62 (.112)
Zygosity	0.17 (0.44)	0.40 (.694)
Random effects		
Family (within-family variance)	0.82 (0.44)	Wald $z = 1.87$ (.062)

We then examined predictors of school-age rule-breaking. Using mixed multilevel model analyses, we predicted school-age rule-breaking from the fixed factors of age five parent-rated difficult temperament and rule-breaking at age five, and from the nested factor of family, which accounted for sibship within the family. By employing a twin design, we controlled for shared genetic effects within a family by including a within-family measure of whether higher scores on the predictor variables were related to higher rule-breaking scores within each family. By comparing twins within pairs, we controlled for shared genetic and familial influences that make children within a family more similar to each other (Schwartz, 2016). Using the method described by Turkheimer and Harden (2014), we calculated the mean score within each family for each predictor variable and then the difference between each child and the mean score (calculated as mean score minus child score for each child). Thus, we were able to calculate between-family (beta weight for mean family score) and within-family (beta weight for the difference score) effects.

This analysis (see Table 1) showed that the stability of rule-breaking behaviors was due to environmental rather than genetic factors, $t(47) = 1.70$, $p = .096$, because the interaction with zygosity was not significant. Additionally, the significant between-family effect, $t(47) = 3.22$, $p = .002$, demonstrated that families with overall higher rule-breaking at age five had overall higher rule-breaking later. This is not a causal relationship or the within-family effect (indicating that the child with the higher rule-breaking score at age five continued to have the higher rule-breaking score at follow-up) would also have been significant. For temperament, the within-family effect was significant, $t(47) = -2.04$, $p = .047$, demonstrating that twins with more difficult temperament have higher rule-breaking later, even after controlling for latent sources of genetic and environmental influences (Schwartz, 2016). Additionally, the relationship between early temperament and later rule-breaking was due to environmental rather than genetic effects, $t(47) = 1.32$, $p = .193$.

Discussion

There was a modest association between genetic factors related to rule-breaking behaviors and aggression (but not assertiveness) exhibited by an unfamiliar peer during free play. This finding suggests that the genetically influenced characteristics of rule-breaking, perhaps stylistic or temperamental, evoke aggressive responses in same-aged peers and indicates that genes and environments are not independent influences on development. Rather, in an unfamiliar free play situation that was unstructured by parents, genes are bound up with environment by way of child conduct that evokes and shapes the responses of the environment. One might extrapolate that such effects occur in a variety of social situations in which a child is embedded.

Our longitudinal results indicated that environmental factors were significant underlying causes of the stability of rule-breaking and of the relationship between early temperament and later rule-breaking behaviors. The use of a twin sample for these analyses was essential for uncovering the underlying bases between these behaviors. These results suggest that preschool rule-breaking and early difficult temperament may indeed contribute to later rule-breaking behaviors, but this appears to happen because of environmental influences over time that are shared between these behaviors. Given the findings from the peer play study, perhaps children who engage in these behaviors are more likely to elicit reactions from their peers that reinforce continued engagement in rule-breaking activities.

In closing, we offer some personal reflections about Irv Gottesman. As a teacher and mentor, Irv taught us to question dogma and to approach our field with empirically based skepticism. In terms of science, he recognized no sacred cows, and he did not suffer fools gladly. He also recognized that human behavior is as complex as it is varied, and that simplistic explanations of genetic and environmental influences were unlikely to carry the day. His ethics were admirable. It was as important to him to accurately apply scientific knowledge as it was to acquire it. This made a strong impression on us, which we draw on when we teach sensitive topics related to behavior genetics. We and our

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scholarly 'siblings' knew Irv as a lifelong mentor whose influence did not end on graduation day. He frequently emailed us articles that we should read or articles he was sure we should challenge or refute. The work continued to the last day of his life when Irv, having been contacted by the editor of the American Journal of Medical Genetics to write an article for the journal, played scholarly yenta and referred the editor to me (LFD). Irv's intellect, his wit, and his generosity of spirit will be missed.

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