Research Report

SCHOOL CONTEXT AND GENETIC INFLUENCES ON AGGRESSION IN ADOLESCENCE

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Abstract—*Genetic and environmental contributions to variation in aggression were examined using adolescents' self-reports of aggressive behavior. The National Longitudinal Study of Adolescent Health provided a sample of 1,515 pairs of adolescents in five genetically informative groups (i.e., monozygotic twins, dizygotic twins, full siblings, half siblings, and unrelated siblings reared together). The analysis, a DeFries-Fulker regression within a hierarchical linear model, yielded findings on individual-level heritability (h^2), shared environmental effects (c^2), school-level effects, and school-level moderation of h^2 and c^2. The estimate of h^2 for aggression in the full sample was .32, and c^2 was .05. In the moderating effect, h^2 increased and c^2 decreased with greater school-level family warmth. Two effects on school means were found: Those schools with greater ethnic-racial heterogeneity had higher mean levels of aggression, and schools where students perceived greater family warmth had lower mean levels of aggression.*

There is an increasing body of work from behavioral genetic research that demonstrates a moderate-to-strong genetic influence on aggressive and antisocial behavior (e.g., Edelbrock, Rende, Plomin, & Thompson, 1995; Mednick, Gabrielli, & Hutchings, 1984; Rowe, 1983). This work was carefully evaluated in a meta-analysis of 24 genetically informative studies of aggression (Miles & Carey, 1997). In a genetic model, equations for expected sibling correlations were fit to the observed sibling correlations from eight different kinship groups. From this genetic model of sibling resemblance, Miles and Carey concluded that aggression is heritable, with a heritability (h^2) as high as .58 in adulthood. However, these authors also observed that genetic influences on variation in aggressive behavior were stronger in adulthood than in childhood and adolescence, and that shared environmental factors were more important during childhood and adolescence (e.g., Lyons et al., 1995). In adulthood, shared environmental influences were negligible.

A limitation of the research reviewed by Miles and Carey is that few studies tested whether the heritability of aggressive behavior varies for individuals in different environmental contexts. The idea that different environmental contexts may moderate the expression of genetic influences is a basis for theories about genetic effects (Bronfenbrenner & Ceci, 1994), but has seldom been tested empirically. According to Bronfenbrenner and Ceci, heritabilities of behavioral traits are expected to be greater in more advantaged environments.

The same hypothesis has been proposed by other researchers. Specifically, Scarr (1992) and Rutter (1985) hypothesized that extremely negative environments have a greater influence on behavior than average or above-average environments. Thus, in more adverse environments, environmental influences on variation in behavior should be stronger and, consequently, genetic influences should be weaker. Conversely, more advantaged environments offer a wider range of opportunities, which enhance the potential for active genotype → environment correlations (Scarr, 1992; Scarr & McCartney, 1983). In active genotype → environment correlations, genetically influenced characteristics or behaviors lead the individual to seek out (consciously or unconsciously) environments that reinforce these characteristics. Because genotype → environment correlations are "read" in behavioral genetic models as genetic influences, heritabilities should be higher in more advantaged environments.

Although studies of moderator variables in behavioral genetic research are hampered both by the failure to specify a measured environmental moderator and by the relatively large sample size needed to detect moderator effects (McClelland & Judd, 1993), there is some consistent evidence for the moderation of the heritability of intelligence. Specifically, three separate studies have found that the heritability of IQ, achievement, or both is greater among individuals from more advantaged environments (Fischbein, 1980; Rowe, Jacobson, & Van den Oord, in press; Scarr-Salapatek, 1971). To date, however, there have been no published studies of how environmental context may moderate the heritability of aggressive behavior.

In the current study, we sought to estimate genetic and shared environmental influences on aggression in a large sample of adolescent siblings and to evaluate the effect of moderator variables. In addition to monozygotic (MZ) and dizygotic (DZ) twins, our research design included full siblings, half siblings, and unrelated siblings (e.g., adoptive children and stepchildren). A further contribution of the present study is that the environmental moderator variables examined were school-level variables. We chose to focus on two moderator variables: schools' racial-ethnic heterogeneity and average family warmth. As a main effect, either or both moderators might be associated with schools' mean level of aggression. At the individual level, the moderators might also change the magnitude of the genetic influences (i.e., the heritability) or the magnitude of the environmental influences on the variation of aggression.

ANALYTIC STRATEGY

Our analysis combined a standard behavioral genetic model, the DeFries-Fulker (DF) regression equation (DeFries & Fulker, 1985), within a hierarchical linear model (HLM; Bryk & Raudenbusch, 1992). The DF regression provides estimates of heritability and environmental variance components of aggression from the following equation:

$$AGG_{S1} = ß_1 AGG_{S2} + ß_2 R + ß_3 R^* AGG_{S2} + ß_o + e,$$

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where AGG_{S1} is the aggression score (i.e., phenotype) for Sibling 1, AGG_{S2} is that of Sibling 2, R is the coefficient of genetic relatedness (1.0 for MZ twins, .50 for DZ twins and full siblings, .25 for half siblings, 0 for biologically unrelated siblings), $ß_o$ is the intercept, and e is the error. The unstandardized coefficient, $ß_1$, is the estimate of shared environmental effect. The coefficient on the interaction term, $ß_3$, is the heritability estimate, that is, the proportion of sibling resemblance that depends on the level of genetic relatedness.

We applied the DF model within HLM. This approach differs from the standard DF regression model in that HLM is based on a multilevel data array of students nested within schools in which both within-school and between-school variation of the data are assessed. The method allows for simultaneous estimation of both (a) Level 1 within-school equations in which separate regression slopes and intercepts are estimated for each school and (b) a Level 2 between-schools equation in which within-school slopes and intercepts are treated as dependent variables. We initially computed the following model:

$$\text{Level 1: } AGG_{S1j} = ß_{1j}AGG_{S2j} + ß_{2j}R + ß_{3j}R^*AGG_{S2j} + ß_{0j} + e_{Sj}$$

$$\text{Level 2: } ß_{0j} = \gamma_{00} + \mu_{0j}$$

$$ß_{1j} = \gamma_{10} + \mu_{1j}$$

$$ß_{3j} = \gamma_{30} + \mu_{3j}$$

At Level 1, the DF equation is computed for each school, denoted by the subscript j (plus the within-school random effect, e_{sj}). At Level 2, each school's mean level of aggression ($ß_{0j}$) is modeled as a function of the grand mean (γ_{00}) plus between-school random error (μ_{0j}). Likewise, estimates of heritability ($ß_{3j}$) and shared environment ($ß_{1j}$) within each school are modeled as a function of grand means pooled across schools (γ_{30} and γ_{10}, respectively) plus random error. Because HLM estimates the variance of aggression between and within schools, we were able to calculate the proportion of variance in aggression that could be attributed to being in different schools (i.e., the intraclass correlation for schools).

We next expanded the Level 2 equations to first test whether the ethnic and racial heterogeneity and the average family warmth of schools predicted the schools' mean level of aggression. We then tested whether racial heterogeneity and family warmth were moderators of Level 1 beta coefficients, which in our application are the genetic and environmental variance components. The Level 2 equations were expanded as follows:

$$\text{Level 2: } ß_{0j} = \gamma_{00} + \gamma_{01}(\text{racial heterogeneity}) + \gamma_{02}(\text{family warmth}) + \mu_{0j}$$

$$ß_{1j} = \gamma_{10} + \gamma_{11}(\text{racial heterogeneity}) + \gamma_{12}(\text{family warmth}) + \mu_{1j}$$

$$ß_{3j} = \gamma_{30} + \gamma_{31}(\text{racial heterogeneity}) + \gamma_{32}(\text{family warmth}) + \mu_{3j}$$

where each school's mean level of aggression ($ß_{0j}$) is modeled as a function of the racial heterogeneity (γ_{01}) and average family warmth of the school (γ_{02}). Estimates of heritability ($ß_{3j}$) and shared environment ($ß_{1j}$) within each school are also modeled as a function of the racial heterogeneity (γ_{31} and γ_{11}, respectively) and average family warmth of the school (γ_{32} and γ_{12}, respectively).

METHOD

The National Longitudinal Study of Adolescent Health (Add Health) was designed to assess the health status of a nationally representative sample of adolescents and to explore the causes of their health-related behaviors. Details on the sampling procedure are given in Resnick et al. (1997).

The Add Health pairs sample includes 3,139 sibling pairs, or 6,278 individuals (see Jacobson & Rowe, 1998, for details). Pairs were included only if the two siblings attended the same school. Further, schools that contained fewer than 15 pairs were also deleted, resulting in a final sample size of 1,515 pairs. These sibling pairs were distributed among 52 of the possible 80 high schools and their feeder junior high schools.

All variables came from an in-home, computer-administered survey, with a professional interviewer standing nearby to provide assistance.

- *Aggression.* The dependent variable was a four-item scale of aggression. The scale was computed using self-reports of the frequency with which the respondents had engaged in the following four behaviors during the past year: (a) got into a serious physical fight, (b) hurt someone badly, (c) used (or threatened to use) a weapon, and (d) took part in a gang fight. Responses ranged from 0 (*never*) to 3 (*five or more times*). Items were averaged to create a scale score (alpha = .74). Aggression was associated with age and sex, with males showing greater aggression than females (mean scores of 0.38 and 0.19, respectively, $F[1, 3028] = 251.1$, $p < .001$) and aggression decreasing slightly with age ($r = –.06$). In the analyses that follow, the dependent variable was the residuals from the regression of aggression on age and sex, thus removing age effects and mean-level sex differences.

- *Family warmth.* Family warmth was measured via self-reports to the following five items: How much do you feel that (a) your parents care about you, (b) people in your family understand you, (c) you want to leave home (reverse scored), (d) your family has fun together, and (e) your family pays attention to you? Responses ranged from 1 (*not very much*) to 5 (*very much*). Items were averaged to create a single scale score (alpha = .77).

- *School-level variables.* School-level variables were computed using data from all of the participating students (i.e., sibling pairs + nonpairs subsamples) in the selected 52 schools. The number of participating students ranged from 61 to 1,721, with an average of 234 students per school. Racial-ethnic heterogeneity (H) was computed by first calculating the percentage of white, black, and Hispanic students who participated in each of the 52 schools and then applying these percentages to the following equation:

$$H = 1– [(\%\text{white})^2 + (\%\text{black})^2 + (\%\text{Hispanic})^2].$$

School-level family warmth was calculated by averaging the family-warmth scores from all participating students within each school. The average school-level warmth was 3.92 ($SD = 0.13$).

Table 1. *Results from hierarchical linear model (HLM) analysis of school-level predictors of aggression*

Term	Coefficient	σ_e
Intercept (school means, $ß_{0j}$)		
Intercept (γ_{00})	0.024	.017
Racial heterogeneity (γ_{01})	0.101*	.028
Family warmth (γ_{02})	–0.300*	.145
Shared environment effect ($ß_{1j}$)		
Intercept (γ_{10})	0.021	.061
Family warmth (γ_{12})	–1.010*	.510
Heritability ($ß_{3j}$)		
Intercept (γ_{30})	0.389*	.112
Family warmth (γ_{32})	1.970*	.962

Note. Coefficients are based on the following HLM:

Level 1: $AGG_{S1j} = ß_{1j}AGG_{S2j} + ß_{2j}R + ß_{3j}R*AGG_{S2j} + ß_{0j} + e_{Sj}$

Level 2: $ß_{0j} = \gamma_{00} + \gamma_{01}$(racial heterogeneity) + γ_{02}(family warmth) + μ_{0j}

$ß_{1j} = \gamma_{10} + \gamma_{11}$(racial heterogeneity) + γ_{12}(family warmth) + μ_{1j}

$ß_{3j} = \gamma_{30} + \gamma_{31}$(racial heterogeneity) + γ_{32}(family warmth) + μ_{3j}

*$p < .05$, two-tailed.

RESULTS

The DF regression analysis was conducted twice: once in the standard way,[1] combining pairs from the different schools, and once as a Level 1 analysis within HLM. The two analyses yielded similar estimates of heritability ($h^2 = .35$, $\sigma_e = .10$, $p < .01$, and $h^2 = .32$, $\sigma_e = .12$, $p < .05$, respectively) and shared environment ($c^2 = .06$ and .05, respectively; for both, $\sigma_e = .06$ and $p > .05$).[2] Nonshared environmental influences accounted for the remainder of the variance (in standard DF, 59%; in HLM, 63%). In the HLM analysis, the effect of school was statistically significant, indicating that schools differed with respect to their mean level of aggression. However, the intraclass correlation for schools, .028, showed that tremendous variability in aggression existed within the average school, making the between-schools variability relatively small.

In the HLM analysis that included the two moderator variables (see Table 1), higher mean level of aggression within schools was predicted by greater racial-ethnic heterogeneity ($\gamma_{01} = .101$) and by less family warmth ($\gamma_{02} = –.300$). Inspection of the means within each of the four primary ethnic-racial subgroups (white, black, Hispanic, and other) indicated that aggression increased in more heterogeneous schools for all groups.

The Level 2 HLM analysis also tested for interaction effects with the behavioral genetic variance components. No variance component interacted with ethnic-racial heterogeneity (results not shown), but the shared environment and genetic variance components both interacted with school-level family warmth. The shared environment effect decreased with greater school-level family warmth ($\gamma_{12} = –1.01$, see Table 1). In contrast, heritability increased with greater school-level family warmth ($\gamma_{32} = 1.97$). These results indicate that in schools with greater mean levels of family warmth, individual differences in aggression are due to genetic differences to a greater extent than in schools with lower mean levels of family warmth. Conversely, the effect of shared environmental influences on aggression is larger in schools with lower means levels of family warmth.

Using the intercept and beta weight values in Table 1, we estimated the heritability for three groups categorized by school-level warmth. When the school-level warmth was equal to the mean, estimates of heritability and shared environmental influences were similar to those reported for the DF model in HLM without covariates: $h^2 = .39$, $c^2 = .02$, nonshared environmental influences (e^2) = .59. When school-level warmth was 1 *SD* below the mean, h^2 was lower (.13), c^2 was higher (.15), and e^2 was higher as well (.72). In contrast, when school-level warmth was 1 *SD* above the mean, h^2 was higher (.65), c^2 was essentially zero (–.11), and e^2 was lower (.35).

To verify the results, we divided the sample at the median for school-level warmth and then compared the within-sibling correlations for aggression. These sibling correlations are presented in Table 2. As can be seen in the table, in schools with higher average levels of family warmth, sibling correlations increased markedly with the degree of genetic relatedness, from $r = –.03$ to $r = .56$, indicating strong genetic influence. In contrast, among schools with lower levels of average family warmth, sibling correlations were more similar, indicating weaker genetic effects and stronger shared environmental influences. The correlations for MZ twins were also lower in schools with lower levels of average family warmth, indicating greater nonshared environmental influences in those schools than in schools with higher levels of family warmth. These sibling correlations, however, were computed on an arbitrary split of the sample, whereas the HLM evaluated the moderation effect over the whole range of family warmth.

1. In DF analysis, data are double entered, so that Sibling A's aggression score serves once in the dependent variable *Y* and once in the independent variable P_2, and similarly for Sibling B's score. The number of observations is twice the number of sibling pairs. Standard errors were adjusted to reflect the proper sample size in both the ordinary DF analysis and the within-school HLM analysis. We obtained the same findings using single-entry data (i.e., when only one of the siblings' aggression scores was used as the dependent variable).

2. The estimates of heritability and shared environment are probably affected by the skewness of the aggression data, as 68% of girls and 48% of boys had committed no aggressive act (see Van den Oord & Rowe, 1997). However, the regularity of the within-sibling correlations (discussed later) increases our confidence in the estimates.

Table 2. *Sibling correlations on aggression*

	School-level family warmth	
Group	High	Low
Monozygotic twins	.56*	.42*
Dizygotic twins	.33*	.31*
Full siblings	.18*	.17*
Half siblings	.07	–.10
Unrelated siblings	–.03	.21*

Note. Sibling correlations were computed in Level 1 of the hierarchical linear model.
*$p < .05$, two-tailed.

DISCUSSION

The present study demonstrated a way in which HLM can be used with behavioral genetic designs to determine whether genetic and environmental influences on variation in adolescent aggression vary across context. Overall, our findings confirmed a conclusion from a meta-analysis of behavioral genetic studies (Miles & Carey, 1997): Aggression is heritable, and shared environmental influences are relatively weak. In the HLM, the shared environmental effect was estimated at .05. The HLM-derived estimate of heritability among adolescents (.32), however, was lower than the estimate from Miles and Carey's meta-analysis (.58 in adulthood), which may indicate that the heritability of aggression is lower in adolescence than in adulthood. The remainder of variation was due to reliable nonshared environment and measurement error (.63). Reliable nonshared environmental variation was .37 of total variation.[3]

We did, however, find that environmental context, measured at the school level, moderated both mean level of aggression and the genetic and environmental influences on variation in aggression. Specifically, schools with greater ethnic and racial heterogeneity had higher mean levels of aggression, as did schools with lower average levels of family warmth. Family warmth also moderated genetic and environmental influences on variation in aggression. In line with theoretical postulates of several researchers (e.g., Bronfenbrenner & Ceci, 1994; Rutter, 1985; Scarr, 1992), the heritability of aggression was higher in schools with higher average levels of family warmth. Conversely, both shared and nonshared environmental influences were stronger in schools with lower average levels of family warmth. One explanation for these findings is that a greater genetic effect is required for the expression of aggression in more benign environments. In more adverse environments, processes such as social norms and peer models may promote aggressive behavior even among individuals without a genetic predisposition. In conclusion, this study is consistent with the pattern of results found for intelligence and ability, namely, that environmental context moderates the genetic and environmental components of variation in phenotypes.

3. The logic behind this estimate is as follows. The total variation of aggression (standardized) is 1.0. The reliable variation is .74. Of the reliable variation, .37 (.32 heritability + .05 shared environment) is explained by genes and shared environment. Thus, the remainder is reliable, nonshared variation: .74 – .37 = .37.

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