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Genetic and Environmental Influences on Intimate Partner Aggression: A Preliminary Study

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Social learning theory posits that, because aggression against intimates runs in families, children learn how to behave aggressively through watching their parents and being reinforced for their own aggression. This theory considers only environmental influences on familial resemblance; however, familial resemblance could also be due to genetic factors. The current study uses a twin design (134 monozygotic, 41 dizygotic) to examine the extent to which genetic and environmental factors contribute to individual differences in intimate aggression. Model-fitting analyses consistently showed that shared genes explained the familial resemblance in psychological and physical intimate partner aggression; the remaining variance was explained by unique environments. Multivariate model-fitting analyses showed that most of the genetic influences responsible for the receipt of aggression were also responsible for its use, suggesting that there is a genetic predisposition to get involved in aggressive relationships. These results challenge the prevailing theory to explain familial resemblance in intimate aggression.

There is an abundance of evidence that clearly indicates that intimate partner aggression (IPA) passes through the generations, such that children who are exposed to aggression in their families of origin are more likely to use aggression in their relationships as adults than children who are never exposed to familial aggression (e.g., Kalmuss, 1984; Stith et al., 2000; Widom, 1989). This exposure to aggression can come in one of two forms: witnessing interparental aggression and/or being the direct recipient of parental aggression. The prevailing theory to explain this familial resemblance in IPA is social learning theory, which suggests that people learn these behaviors through observing the behaviors of their parents and through the reinforcement of aggressive behaviors (e.g., Eron, 1997). This social learning theory account assumes that familial patterns of aggression are entirely due to environmental factors. However, the pattern of familial resemblance reported in the literature could also be due to shared genes. Determining whether familial resemblance in IPA is due to shared genes, shared environments, or both is essential for effectively creating prevention and intervention programs.

Several researchers have proposed looking at possible genetic contributors to family aggression; however, to date, possible genetic contributions have not been empirically

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examined. For example, Widom (1989) suggests that physiological predispositions might mediate the effects of the intergenerational transmission of family aggression. DiLalla and Gottesman (1991) state that ignoring possible genetic factors in family aggression would greatly limit our understanding of the intergenerational transmission of aggression. Finally, Herzberger (1996), in her review of social learning theory and aggression in the family, asserts that it is important to test both genetic and social models of the transmission of family aggression. She states that caution is necessary when concluding that family aggression is transmitted through learning in the home environment because the parents who model aggressive behaviors have also passed along their genes to their children. Clearly, there is a need for behavioral genetic studies of IPA to resolve these issues, but thus far, none have been conducted.

If genetic influences are important to a trait or behavior, then behavioral similarity should covary with genetic relatedness; that is, individuals who are more genetically similar should be more behaviorally similar (Hines & Saudino, 2002). From a behavioral genetic perspective, the many studies that show that aggression against intimates tends to transmit through families can be viewed as family studies. Family studies are useful in showing that a behavior *may* have genetic influences. However, because families share both genes and environments, the relative contributions of each cannot be disentangled (Plomin, DeFries, McClearn, & Rutter, 1997). To do this, twin or adoption studies are needed. Although no twin or adoption studies have examined IPA, research in related fields, such as general aggression, can be used as a guide to whether genetic influences on IPA are plausible. Even though the dynamics of aggression toward a family member may be different than the dynamics of aggression toward a stranger, their underlying etiology seems to be similar. Indeed, several studies have shown that extrafamilial and intrafamilial aggression are moderately to highly correlated and that they share many of the same risk factors (e.g., Hotaling, Straus, & Lincoln, 1990; Lackey, 2003; Moffitt, Krueger, Caspi, & Fagan, 2000; Simons, Wu, Johnson, & Conger, 1995; Widom, 1989).

Overall, the literature on extrafamilial aggression consistently shows that monozygotic (MZ) twins are more similar than dizygotic (DZ) twins, and that adoptees are more similar to their biological relatives than their adoptive relatives, for a broad range of antisocial behaviors and traits, including convictions for felonies (e.g., Cloninger & Gottesman, 1987; Raine, 1993), symptom counts for antisocial personality disorder (e.g., Cadoret, O'Gorman, Troughton, & Heywood, 1985; Lyons et al., 1995), self-reported aggression (e.g., Coccaro, Bergeman, Kavoussi, & Seroczynski, 1997; Miles & Carey, 1997), and personality scales for aggression and hostility (e.g., McGue, Bacon, & Lykken, 1993; Tellegen et al., 1988). Furthermore, genetic factors explain more of the variance in antisocial behaviors and traits than do environmental factors (DiLalla & Gottesman, 1991), in that overall nonshared environmental influences account for 40% to 50% of the variance in antisocial behaviors and traits (Carey & Goldman, 1997), whereas heritability is estimated to account for at least 50% of the variance (DiLalla & Gottesman).

The present study examines genetic and environmental contributions to the use and receipt of psychological and physical IPA in adult twins. The finding of genetic influences on individual differences in extrafamilial aggression suggests that the well-documented pattern of familial resemblance for aggression in intimate relationships may be due to shared genes, not shared environments as suggested by social learning explanations. Thus, behavioral genetic research, such as the present study, has the potential to provide a strong empirical test of the prevailing theory by identifying the mechanisms through which aggression against intimates transmits through families. If shared environmental influences

are found, it would be consistent with a social learning theory explanation of familial resemblance. However, a finding that the familial resemblance for IPA is due to genetic, not shared environmental, influences would provide a strong challenge to the current theory.

METHODS

Participants

Participants included 185 pairs of same-sex twins (144 MZ, 41 DZ) with a mean age of 40.3 years (SD = 14.6) who had experience in an adult intimate relationship. Participants were recruited at twins conventions, through advertisements that were placed on twin Internet sites and in twin bulletins distributed at twins conventions, flyers posted in various cities and towns throughout the United States, and word of mouth. To mask the true purpose of the study and thus reduce the likelihood of recruitment bias, potential participants were told that the study was investigating genetic and environmental contributors to conflict resolution techniques in romantic relationships. Of the 266 twin pairs who agreed to participate in the study, 70% (n = 185 twin pairs) returned the completed questionnaires. Ten twin pairs did not correctly complete the aggression measure (i.e., they indicated that they never had a romantic relationship and left it blank, or they indicated that they never had a romantic relationship and filled it out with regard to their twin relationship); therefore, genetic analyses are based on 134 MZ and 41 DZ twins.

Table 1 provides demographic information for the sample. As shown, the majority of participants were White females. Also, the majority of twins were heterosexual (96.6%) and currently in a romantic relationship (71.1%); 55.1% were married, and the average relationship length was 11.8 years (SD = 12.8). According to the Hollingshead index, the average socioeconomic status (SES) was middle class.

Measures

Zygosity Questionnaire. Zygosity was determined through a physical similarity questionnaire completed by both members of the twin pair (Magnus, Berg, & Nance, 1983). The questions addressed degree of physical resemblance and how often the twins were mistaken for each other as children. This method of zygosity classification has been shown to yield accuracy of approximately 95% when compared to tests of single-gene markers in blood (Eaves, Eysenck, & Martin, 1989). It is important to note that any misclassification of twins would mathematically work against the genetic hypothesis.

The Revised Conflict Tactics Scales. To examine the frequency of IPA, the physical and psychological aggression subscales of the Revised Conflict Tactics Scales (CTS2, Straus, Hamby, Boney-McCoy, & Sugarman, 1996) were used. For each participant, the number of acts used and received in the previous year on both subscales was computed. Participants indicated on a scale from 0 to 6 how many times in the previous year they experienced the acts listed. They also indicated whether they had *ever* used any of these acts in their relationship. The items were coded in the following manner to obtain a measure of their current aggression: 0 = 0 times; 1 = not in past year, but has happened; 2 = once in the past year; 3 = twice in the past year; 4 = 3 to 5 times in the past year; 5 = 6 to 10 times in the past year; 6 = 11 to 20 times in the past year; and 7 = > 20 times in the past year. The scores for the individual items pertaining to each subscale were then added together to obtain continuous measures of psychological and physical aggression. This

TABLE 1. Sample Demographics

	Total	MZ Twins[a]	DZ Twins
n pairs	175	134	41
Gender			
Males	23	18	5
Females	152	116	36
Ethnicity			
Native American	1	1	0
Asian American	3	3	0
African American	8	7	1
White	161	121	40
Hispanic	1	1	0
Other	1	1	0
Mean age (*SD*)	40.3 (14.6)	41.8 (15.0)*	35.4 (12.1)
Mean length of relationship (*SD*) (in years)	11.8 (12.8)	13.0 (13.8)*	7.9 (8.0)
Mean level of relationship commitment (*SD*)	5.3 (2.1)	5.4 (2.1)	4.8 (2.2)
Mean Hollingshead SES (*SD*)	47.1 (10.6)	47.0 (10.4)	47.5 (11.2)
% of total in heterosexual relationships	96.6%	96.7%	96.3%
% of total currently involved in a romantic relationship	71.1%	72.0%	68.3%
% of total married	55.1%	58.2%	45.1%
% of total living together	8.9%	6.7%	15.9%
% of total dating	33.7%	32.8%	36.6%

[a]An asterisk in this column indicates a significant difference between MZ and DZ twins; $^*p < .05$.

scoring method did not alter the reliability of the scales, as alphas ranged from .77 for the psychological aggression used and received subscales to .82 for physical aggression received. These continuous data were then log-transformed for model fitting analyses to correct for positive skewness. Because twin correlations can be inflated by variance due to age and sex, scores were residualized for age and sex effects (see McGue & Bouchard, 1984).

Design and Analyses

The twin method involves comparing genetically identical (MZ) twins with fraternal (DZ) twins who share approximately 50% of their segregating genes. Genetic influences are implied when cotwin similarity covaries with the degree of genetic relatedness. Thus, if heredity affects a trait, the twofold greater genetic similarity of MZ twins is expected to make them more similar than DZ twins. Intraclass correlations, which are calculated using a double-entry procedure, typically serve as indices of cotwin similarity. An MZ correlation that is significantly greater than the DZ correlation suggests genetic influence. An estimate of heritability (h^2), the genetic effect size, can be derived by doubling the difference between the MZ and DZ correlations (Plomin et al., 1997).

Heritability is the proportion of observed variance for a trait that can be attributed to genetic influence. The remaining variance is attributed to environmental factors, which

include all nonheritable influences. The environmental variance component can be decomposed into shared and nonshared environmental influences. Shared environmental variance (c^2) is twin resemblance that is not explained by genetic variance. Thus, c^2 includes those environmental influences common to both members of a twin pair that enhance cotwin similarity. Possible shared environmental influences include SES, parental education, and the presence of other siblings in the home, to the extent that these variables serve to enhance twin similarity. DZ correlations that exceed one half the MZ correlation suggest the presence of shared environmental influences. Doubling the "excess" DZ cotwin similarity not accounted for by h^2 provides an estimate of c^2. Nonshared environmental variance (e^2) is a residual variance that includes measurement error and environmental influences that are unique to each individual. These nonshared environmental influences serve to make members of the same family different from each other in a trait, and may include differential peer groups, illnesses and accidents that are unique to each twin, and differential parental treatment. Differences within pairs of MZ twins are due to nonshared environmental influences; thus, e^2 can be estimated by the extent to which the MZ correlation is less than unity (Plomin et al., 1997).

In addition to correlational analyses, model-fitting analyses were used to assess the genetic and environmental influences on each aggression type. Model-fitting procedures provide a more elegant analysis of genetic and environmental influences because they estimate multiple parameters simultaneously, test the model, make assumptions explicit, provide parameter estimates, and permit tests of alternative models (Loehlin, 1987; Neale & Cardon, 1992). For example, reduced models without the genetic and/or shared environmental parameters can be tested against the full model. Because these reduced models are nested within the full model, the change in chi-square from the full model to the reduced model estimates the significance of the parameter(s) not included in the reduced model. Degrees of freedom (*df*) for the change in chi-square is equal to the difference in *df* between the two models.

In the present study, maximum-likelihood model-fitting analyses were performed on twin variance/covariance matrices using *Mx* maximum-likelihood model-fitting procedures (Neale, Boker, Xie, & Maes, 2002). The univariate model is depicted as a path diagram in Figure 1. Observed phenotypic variances of each twin are represented by the two rectangles. The circles represent latent genetic and environmental variables. The curved double-headed arrows indicate correlations between the variables they connect. The single-headed arrows a, c, and e represent paths (i.e., partial regressions of the measured variable on the latent variable). According to the model, phenotypic variation is assumed to be due to three latent variables: additive genetic effects (A), shared environmental effects (C), and nonshared environmental effects (E). Based on the degree of genetic relatedness, the A factors correlate 1.0 and 0.5 for MZ and DZ twins, respectively. All twins in this study were reared in the same family; therefore, both MZ and DZ twins correlate 1.0 for shared environmental effects. Finally, nonshared environmental influences are depicted in the path diagram as residual arrows for each twin and represent the remaining variance not explained by genes or shared environments. Using this model, estimates of heritability and shared and nonshared environmental variances and their 95% confidence intervals were estimated.

The fit of the full ACE model was assessed by the goodness-of-fit χ^2 test, and reduced models were then compared. These reduced models either fixed the additive genetic variance (CE model), the shared environmental variance (AE model), or both (E model) to zero. The CE model assumes that familial resemblance for the behavior is due to shared

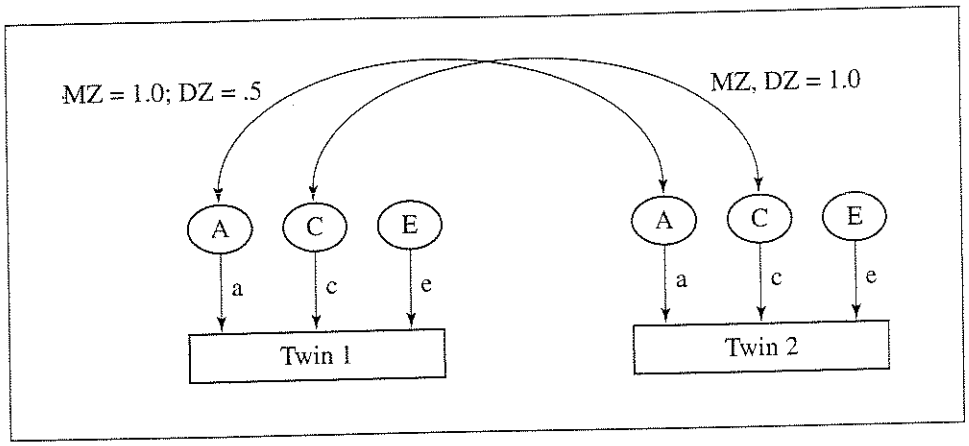

Figure 1. Univariate model. A = latent additive genetic effects; C = latent shared environmental effects; E = latent unique environmental effects; a = genetic effects; c = shared environmental effects; e = unique environmental effects.

environments, the AE model assumes that resemblance is due to shared genes, and the E model assumes no familial resemblance. When submodels did not significantly differ from the full model as assessed through the χ^2 Diff test, two criteria were used to determine the best fitting model:

1. The model with the lowest Akaike Information Criterion (AIC), which equals χ^2 minus twice the *df*; the model with the lowest AIC has the optimal combination of goodness-of-fit and parsimony; and
2. The model with the lowest root mean squared error approximation (RMSEA), which is a measure of fit that is independent of sample size (Neale et al., 2002).

A bivariate model was used to assess the extent to which the same genetic and environmental influences operate on the use and receipt of psychological aggression, and the use and receipt of physical aggression (see Figure 2). This model partitions the phenotypic covariation (i.e., observed covariation) between the two traits (e.g., the use and receipt of psychological aggression) into its genetic, shared, and nonshared environmental components. The latent variables A1, C1, and E1 refer to the additive genetic, shared, and nonshared environmental influences on trait 1 (e.g., the use of psychological aggression); and A2, C2, and E2 refer to the additive genetic, shared, and nonshared environmental influences on trait 2 (e.g., the receipt of psychological aggression). The path coefficients h1, h2, c1, c2, e1, and e2 are standardized partial regressions indicating the relative influence of the latent variables on the phenotype. Of particular interest in this model is the estimated parameter r_g, the genetic correlation between genetic effects on trait 1 and trait 2. The genetic correlation is the extent to which the genetic influences on one trait overlap with the genetic influences on another trait, independent of the heritability of each trait. The genetic correlation between two traits can be unity, such that all of the genes that influence one trait are the same as all of the genes that influence the other, even though the heritability of each trait may be quite low or may substantially differ. Conversely, the genetic correlation between two traits can be zero even though the two traits might be highly heritable. In this case, the genes that influence each of these traits are independent. Similar

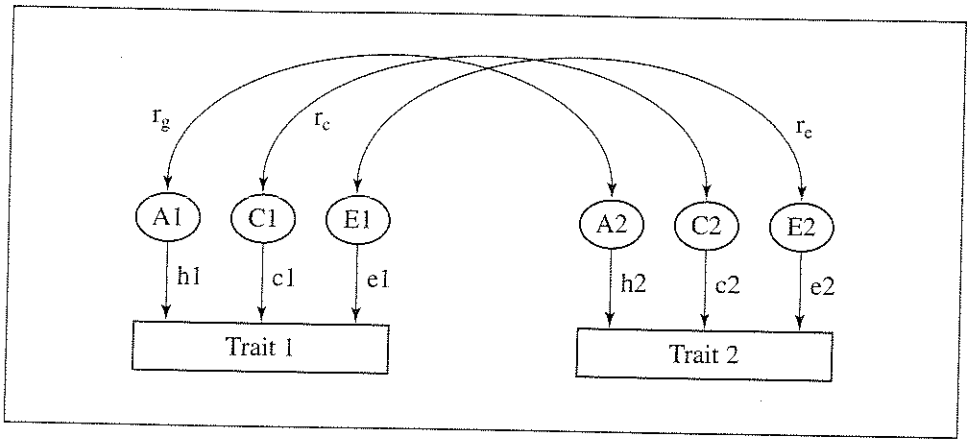

Figure 2. Bivariate model. A1 = latent additive genetic effects on trait 1; C1 = latent shared environmental effects on trait 1; E1 = latent unique environmental effects on trait 1; A2 = latent additive genetic effects on trait 2; C2 = latent shared environmental effects on trait 2; E2 = latent unique environmental effects on trait 2; h1 = additive genetic effects on trait 1; c1 = shared environmental effects on trait 1; e1 = unique environmental effects on trait 1; h2 = additive genetic effects on trait 2; c2 = shared environmental effects on trait 2; e2 = unique environmental effects on trait 2; r_g = genetic correlation; r_c = shared environmental correlation; r_e = unique environmental correlation.

logic applies to r_c and r_e, the estimated shared and nonshared environmental correlations between the two traits.

RESULTS

Descriptive Statistics

T tests were performed on demographic variables to investigate possible differences between MZ and DZ twins (see Table 1). To correct for nonindependence, *t* tests were performed separately for each member of a twin pair (i.e., Twins A and Twins B, determined randomly). The only consistent differences between twin zygosity groups (i.e., significantly different for both A and B twins) were age and length of relationship. MZ twins were significantly older (Twins A: $t = 2.61$, $p < .01$; Twins B: $t = 2.61$, $p < .01$) and in significantly longer relationships (Twins A: $t = 3.48$, $p < .001$; Twins B: $t = 2.32$, $p < .05$) than DZ twins. These differences did not impact the twin analyses, however, because the aggression scores were residualized for age and sex and because length of relationship did not correlate with the use or receipt of aggression in relationships ($r = -.10$ to $.05$, *ns*).

Table 2 shows the prevalence and frequency, overall and stratified by zygosity, of psychological and physical aggression in the participants' current or most recent romantic relationship. These estimates are similar to those obtained in nationally representative samples (e.g., Morse, 1995). Furthermore, there were no differences between MZ and DZ twins in the prevalence or frequency of aggression as measured by each of the subscales. To further explore the representativeness of this twin sample, the physical aggression subscale of the CTS2 was correlated with demographic variables that have been explored in previous research on IPA, namely age and gender. Age and the use of physical aggression were

TABLE 2. Relationship Prevalence and Frequency of Psychological and Physical Aggression

	Total $n = 350$	MZ Twins $n = 268$	DZ Twins $n = 82$
Psychological Aggression			
% used	82.9%	81.7%	86.6%
Mean # of acts (*SD*)	10.7 (7.7)	10.7 (7.8)	10.6 (7.4)
% received	78.6%	78.0%	80.5%
Mean # of acts (*SD*)	10.9 (8.0)	10.6 (7.8)	11.9 (8.7)
Physical Aggression			
% used	22.9%	22.8%	23.2%
Mean # of acts (*SD*)	5.4 (6.8)	5.3 (6.1)	5.5 (8.9)
% received	24.0%	23.1%	26.8%
Mean # of acts (*SD*)	6.6 (6.9)	7.2 (7.4)	5.0 (4.8)

Note. "Mean # of acts" reflects the average number of specific acts experienced by those respondents who reported involvement as either the perpetrator or victim (where appropriate) in either a psychologically or physically aggressive relationship. There were no significant differences between zygosity groups. Numbers shown are collapsed across twins A and B and are not transformed.

consistently significantly correlated, that is, the significance replicated across both Twins A ($r = -.15$, $p < .05$) and B ($r = -.15$, $p < .05$). This result is consistent with previous literature that shows that the older people are, the less likely they are to use physical aggression in their relationships (e.g., O'Leary et al., 1989; Suitor, Pillemer, & Straus, 1990). The only gender difference was for psychological aggression used. Females reported using more psychological aggression for both Twins A (Females: $M = 10.0$, $SD = 8.5$; Males: $M = 5.9$, $SD = 4.8$; $t = 1.81$, $p < .07$) and B (Females: $M = 9.0$, $SD = 8.1$; Males: $M = 4.3$, $SD = 5.9$; $t = 3.31$, $p < .01$). These results are consistent with previous analyses that show that women use more psychological aggression than men (e.g., Hines & Saudino, 2003; Kasian & Painter, 1992) and that there are no gender differences in the use or receipt of physical aggression (e.g., Archer, 2000). Overall, these analyses indicate that our twin sample is comparable to other more representative samples that have assessed IPA.

Genetic and Environmental Influences on Individual Differences in the Use and Receipt of Aggression

Intraclass correlations for the use and receipt of psychological and physical aggression are presented in Table 3. For all four measures, the significant MZ correlations were consistently higher than the nonsignificant DZ correlations, suggesting genetic influences.

To investigate the extent to which genetic and environmental influences were operating for the use and receipt of aggression in relationships, univariate model-fitting analyses were conducted (Table 4). For the use of psychological aggression, the full ACE model fit the data well (i.e., nonsignificant chi-square), and dropping familial resemblance (both A and C) resulted in a significantly worse fit, as indicated by the significant change in chi-square. The best fitting model was the AE model, suggesting that genetic influences account for the familial resemblance in this behavior. For the use of physical aggression, all of the models fit the data well, but the best fitting model was the AE model, suggesting the familial resemblance for this variable was also accounted for by genetic influences.

TABLE 3. Intraclass Correlations

	MZ	DZ
Aggression Use	$n = 133$ pairs	$n = 41$ pairs
Psychological	$.25^{**}{}_{a}$	$-.14_{a}$
Physical	$.17^{*}$	$.05$
Aggression Receipt		
Psychological	$.26^{**}{}_{b}$	$-.08_{b}$
Physical	$.17^{*}{}_{c}$	$-.13_{c}$

Note. Correlations sharing subscripts are significantly different from each other, $p < .05$. $^{*}p < .05$. $^{**}p < .01$.

The model-fitting results for the receipt of psychological aggression in romantic relationships showed a similar picture to the use of psychological aggression. That is, the ACE model fit the data well, and dropping familial resemblance (E only model) resulted in a significantly worse fit. Furthermore, the best fitting model of the remaining models was the AE model. Similarly, there is evidence of genetic influences for familial resemblance in the receipt of *physical* aggression. All of the models fit the data well, and the best fitting model according to AIC was the AE model, suggesting genetic influences. However, the confidence interval for heritability included zero indicating that these genetic influences are not significant. According to RMSEA and the law of parsimony, the best fitting model was the E model, suggesting no familial resemblance in IPA. However, it should be noted that the intraclass correlation for MZ twins was significant and higher than the nonsignificant DZ correlation. Therefore, genetic influences may be operating, but with our small sample, cannot be detected in the univariate model. Indeed, in our bivariate model-fitting analyses (discussed below), genetic influences on this variable were significant, which is not surprising given our mixed univariate results and the fact that the bivariate model has more power to detect significant effects (Schmitz, Cherny, & Fulker, 1998).

Variance Components

Variance components from the best fitting models for the use and receipt of psychological and physical IPA are presented in Figure 3. For both psychological and physical aggression, variance components for *use* of aggression mirror those for its *receipt*. That is, approximately one quarter of the variance in the use of psychological aggression and the receipt of psychological aggression can be attributed to genetic influences, with the remaining variance accounted for by nonshared environmental influences. Similarly, genetic influences accounted for approximately 15% of the variance in use and receipt of physical aggression, with the remaining variance due to nonshared environmental influences.

Do Genetic Influences on the Use of Aggression and on the Receipt of Aggression Overlap?

Examination of the phenotypic correlations (i.e., observed correlations) revealed that there was a high correlation between the use of aggression and its receipt (psychological: $r = .85$, $p < .001$; physical: $r = .59$, $p < .01$). These phenotypic correlations must be due to an overlap in the factors that underlie each of these traits, namely shared genes and/or nonshared environments. As a preliminary step in evaluating genetic and environmental influences on the covariance between the use and receipt of IPA, twin cross correlations were

TABLE 4. Model Fitting Results for the Use and Receipt of Psychological and Physical Aggression

Model	Parameter Estimates (95% CI) h^2	c^2	e^2	χ^2	*df*	*p*	*RMSEA*	*AIC*	χ^2 *Diff*	*p*
Use of psychological aggression										
ACE	.22 (.00–.37)	.00 (.00–.28)	.78 (.63–.93)	3.34	3	.34	.08	-2.66	—	—
AE*	**.22 (.07–.37)**	**—**	**.78 (.63–.93)**	**3.34**	**4**	**.50**	**.06**	**-4.66**	**0.00**	**—**
CE	—	.18 (.03–.32)	.82 (.68–.97)	5.31	4	.26	.09	-2.69	1.97	.16
E	—	—	1.000	10.86	5	.05	.07	0.86	7.53	.02
Use of physical aggression										
ACE	.16 (.00–.31)	.00 (.00–.28)	.84 (.69–1.0)	1.21	3	.75	.00	-4.79	—	—
AE*	**.16 (.01–.31)**	**—**	**.84 (.69–.99)**	**1.21**	**4**	**.88**	**.00**	**-6.79**	**0.00**	**—**
CE	—	.15 (.00–.29)	.85 (.71–1.0)	1.44	4	.84	.00	-6.56	0.23	.64
E	—	—	1.000	5.16	5	.40	.03	-4.48	3.95	.14
Receipt of psychological aggression										
ACE	.25 (.00–.40)	.00 (.00–.28)	.75 (.60–.91)	2.28	3	.52	.05	-3.72	—	—
AE*	**.25 (.09–.40)**	**—**	**.75 (.60–.91)**	**2.28**	**4**	**.68**	**.02**	**-5.72**	**0.00**	**—**
CE	—	.19 (.04–.33)	.81 (.67–.96)	4.71	4	.32	.07	-3.29	2.43	.12
E	—	—	1.000	11.15	5	.05	.08	1.15	8.86	.01
Receipt of physical aggression										
ACE	.15 (.00–.30)	.00 (.00–.24)	.85 (.70–1.0)	3.36	3	.34	.08	-2.64	—	—
AE	.15 (.00–.30)	—	.85 (.70–1.0)	3.36	4	.50	.06	-4.64	0.00	—
CE	—	.11 (.00–.26)	.89 (.74–1.0)	4.36	4	.36	.07	-3.64	1.01	.32
E*	**—**	**—**	**1.000**	**6.58**	**5**	**.25**	**.04**	**-3.42**	**3.23**	**.20**

Note. * and boldface denote best fitting model. h^2 = heritability; c^2 = shared environmental variance; e^2 = nonshared environmental variance.

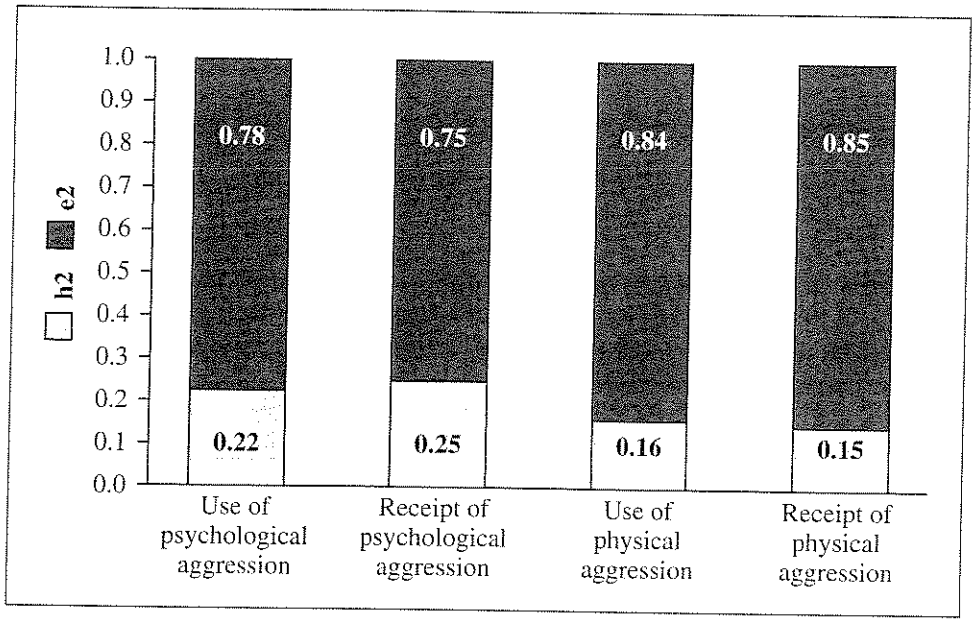

Figure 3. Variance components for the use and receipt of intimate partner aggression.

calculated using the double-entry procedure. The cross correlation is a cross-twin, cross-variable intraclass correlation in which one twin's score on one variable (e.g., Twin A's use of aggression) is correlated with its cotwin's score on the other variable (e.g., Twin B's receipt of aggression). A genetic contribution to the phenotypic correlation is suggested when MZ twin cross correlations exceed DZ twin cross correlations. For psychological aggression, the significant MZ cross correlation ($r = .28$, $p < .01$) exceeded the nonsignificant DZ cross correlation ($r = -.14$, *ns*), suggesting that the phenotypic correlation between the use and receipt of psychological aggression is mediated genetically. For physical aggression, although the MZ correlation ($r = .14$) exceeded the DZ correlation ($r = .06$), neither correlation is significant, suggesting that the phenotypic correlation between the use and receipt of physical aggression is mediated by nonshared environments.

As indicated in the methods section, bivariate model-fitting analyses provide a strong test of genetic and environmental sources of covariance and, more importantly, yield estimates of genetic and environmental correlations. Because the best fitting univariate models for the use and receipt of both psychological aggression and physical aggression were AE models, shared environmental influences cannot contribute to the covariance between use and receipt of aggression, and consequently, c^2 was not included in our bivariate model-fitting analyses. For psychological aggression, the bivariate model provided a good fit to the data (see Table 5). The very high genetic correlation ($r_g = .97$) indicates that almost all of the genetic influences for the use of psychological aggression overlap with the genetic influences for the receipt of psychological aggression. Similarly, there is considerable overlap between nonshared environmental influences on the use and receipt of psychological aggression ($r_e = .82$). Therefore, a substantial majority of both the genes and nonshared environments that influence the use of this type of aggression also influence its receipt. Similar results emerged for physical aggression. The bivariate model fit the data well and showed that most of the genetic influences responsible for the use of physical

E*	—	—	1.000	6.58	5	.25	.04	-3.42	3.23	.20

Note. * and boldface denote best fitting model. h^2 = heritability; c^2 = shared environmental variance; e^2 = nonshared environmental variance.

TABLE 5. Genetic and Environmental Correlations for the Use and Receipt of Aggression

Parameter Estimates (95% CI)													
Psychological Aggression Used			Psychological Aggression Received										
h^2	c^2	e^2	h^2	c^2	e^2	r_g	r_c	r_e	χ^2	*df*	*p*	RMSEA	AIC
.23 (.07–.38)	—	.77 (.62–.93)	.25 (.09–.40)	—	.75 (.60–.91)	.97 (.82–1.0)	—	.82 (.76–.86)	23.51	.14	.06	.11	-4.50
Physical Aggression Used			Physical Aggression Received										
h^2	c^2	e^2	h^2	c^2	e^2	r_g	r_c	r_e	χ^2	*df*	*p*	RMSEA	AIC
.16 (.01–.31)	—	.84 (.69–.99)	.14 (.01–.30)	—	.86 (.70–.99)	.85 (.00–1.0)	—	.55 (.43–.65)	16.89	14	.26	.07	-11.11

Note. h^2 = heritability; c^2 = shared environmental variance; e^2 = nonshared environmental variance; r_g = genetic correlation; r_c = shared environmental correlation; r_e = unique environmental correlation.

aggression overlap with the genetic influences responsible for the receipt of physical aggression (r_g = .85); however, the confidence interval included zero, and therefore, the genetic correlation, although high, was not significant. Furthermore, many of the nonshared environments responsible for the use of physical aggression overlap with those responsible for the receipt of aggression (r_e = .55).

DISCUSSION

This study, the first to examine genetic and environmental influences on individual differences in IPA, provides preliminary evidence that variability in IPA is genetically influenced. The prevailing theory to explain familial resemblance in intimate partner physical aggression has been social learning theory (Eron, 1997). This theory posits that it is the shared familial environment that makes children resemble their parents in their tendency to use aggression in romantic relationships. Consistent with previous research, the current study shows that there is familial resemblance for IPA; but this study also suggests that this resemblance is due to shared genes.

The limitations of this research should be acknowledged. The small sample size, especially for DZ twins, created limitations in several of the twin analyses. For example, because of limited statistical power, we were unable to conclusively reject shared environments as a contributor to the use or receipt of physical or psychological aggression in most of the models. Although the best models consistently were those that included genetic influences, models that included shared environments to explain familial resemblance, albeit not the best models, did not fit significantly worse than the genetic models. In other words, there was not enough power with our small sample to distinguish whether familial influence was definitively due to genetic influences and not shared environments. However, in all instances, in the full models, shared environmental influences were estimated as zero, which was not the case for genetic effects. Similarly, the patterns of twin resemblances in the correlational analyses were consistent with genetic effects, not shared environmental influences. It should also be noted that any recruitment biases associated with our variable of interest would result in shared environmental variance being misattributed to nonshared environmental variance. Therefore, the most prudent interpretation of our data is that the use and receipt of IPA is influenced by both genes and the environment (which might include both shared and nonshared factors). A second limitation was that our sample consisted mostly of White females. Thus, the results may not necessarily be fully generalizable to males or non-Whites. However, our sample was typical in its level of physical and psychological aggression and in its associations between aggression and several major demographic variables (e.g., gender, age). A final limitation was that the majority of our participants who were involved in IPA engaged in what researchers have termed "common couple violence" (Johnson, 1995). Different results might emerge for more severe spousal abuse (battering). Nonetheless, the present results should be viewed as preliminary evidence of genetic influences on IPA, and replication with a larger, more diverse sample is needed to strengthen these preliminary findings.

Although there are limitations to the current study, it is important to note that the results are consistent with behavioral genetic studies of aggression in general. Both twin and adoption studies have confirmed genetic influences on a variety of adult antisocial traits and behaviors, with the remaining variance being accounted for by nonshared environments (Carey & Goldman, 1997; DiLalla & Gottesman, 1991). Furthermore, the current

study suggests that familial resemblance in psychological aggression arises because family members share the genes that influence this behavior, a finding that is consistent with previous behavioral genetic studies that show that the use of verbal aggression in general is genetically influenced (e.g., Seroczynski, Bergeman, & Coccaro, 1999).

This study provides evidence that perhaps the social transmission of aggressive behaviors is horizontal and not vertical (Rowe, 1994). That is, children may inherit genes that predispose them for aggressive behaviors from their parents; however, their eventual use of these behaviors may not depend upon whether their parents behave aggressively (i.e., being exposed to an aggressive familial environment). Instead, the children's genetic predisposition to behave aggressively may influence them to seek out aggressive peer groups (i.e., nonshared environments). Therefore, their eventual use of aggressive behaviors may have little to do with their parents' actual use of aggressive behaviors, but may be due to inheriting a genetic predisposition from their parents and being exposed to aggressive models in their peer groups. This notion is supported by research that shows that exposure to family-of-origin aggression no longer predicts aggressive intimate partner relationships once peer relationships are considered (Silverman & Williamson, 1997).

Victimization from IPA also transmits through families (e.g., Cappell & Heiner, 1990; Kalmuss, 1984). Family violence theorists have traditionally explained this familial resemblance by assuming that children *learn* the victim role either through being victimized by their parents or through watching one of their parents being victimized and identifying with that parent (e.g., Gelles, 1979). That is, the familial resemblance in victimization is due to the shared familial environment. However, as was the case for the use of aggression, the present study suggests that familial resemblance in the receipt of both psychological and physical IPA seems to arise because family members share the genes that influence these behaviors. The fact that there seem to be genetic influences on behaviors performed by individuals other than the respondents seems at first paradoxical. How can behaviors performed by others be influenced by the genotype of the receiver? Are not the participants merely victims of the aggressive behaviors that are used by their partners? Although at first genetic influences on aggressive victimization may seem illogical, there is consistent evidence that people are not merely passive receivers of their environments. That is, to some extent, people's environments reflect their genetically influenced traits (e.g., Kendler, Neale, Kessler, Heath, & Eaves, 1993; Scarr & McCartney, 1983). Therefore, events that may seem external to the individual can have genetic influences. Relevant to the current study are the findings that there are genetic influences on the life events of being robbed and/or assaulted and having marital difficulties (Kendler et al., 1993). Aggression within a marriage not only is symbolic of marital difficulties, it is also analogous with the event of being assaulted. Therefore, the current findings are consistent with previous results that show that the experience of certain negative life events, including being assaulted and having marital problems, are genetically influenced. Genetic influences on the receipt of IPA may be due to *evocative* genotype-environment correlations, where the victimized individuals may receive aggressive responses from their partners because these individuals have genetically influenced traits that could evoke aggressive reactions from others; or to *active* genotype-environment correlations, where the victimized individuals choose aggressive romantic partners because those partners are congruent with certain genetically influenced characteristics of the victims (Scarr & McCartney).

The strong phenotypic correlations between the use and receipt of psychological aggression and between the use and receipt of physical aggression in the present study suggest that aggressive people are choosing aggressive partners. Furthermore, the large

majority of the genetic influences on the receipt of psychological aggression were common to the genetic influences on the use of psychological aggression. Although this last result was not fully replicated with the use and receipt of physical aggression, the substantial genetic correlation suggests that in a larger sample, a genetic link between the use and receipt of physical aggression may be found. Therefore, the general tendency to become involved in aggressive relationships may be genetically influenced.

It is important to delineate what exactly is meant when we say that behaviors are genetically influenced. First and foremost, it means that certain people, due to their genotype, may be more likely to commit aggressive acts in their relationships than people who do not have that same genotype. In other words, genetic influences are *probabilistic,* not deterministic. Genetic influences on aggression in intimate relationships must be seen as a predisposition towards aggression, not as destiny (Gottesman, Goldsmith, & Carey, 1997; Raine, 1993). Thus, the environment and manipulations in the environment can be very successful in reducing aggressive behaviors and preventing the full expression of any genetic predisposition (Hutchings & Mednick, 1977; Raine). Furthermore, although genetic influences explain familial resemblance in IPA in the current study, there was also a substantial influence of the environment, specifically nonshared environments. Environmental influences explained approximately 75% of the variance in these behaviors. The environment *is* important, but it acts to make family members different in IPA. Therefore, the environments that we should be studying are those that are unique to members of the same family and result in *differences* from one another in their aggressive behaviors. Such environments could include differential peer groups, or differential exposure to aggression or traumatic events, such as rape or head injuries. In addition, such environments could include certain family environments that are not shared, such as differential exposure to parental conflicts. That is, although we did not find any shared familial influences, it does not mean that the family environment is unimportant in the development of aggressive behaviors within romantic relationships. Family environments may certainly be important; however, they are important only in the extent to which they make members of the same family different from one another in these behaviors.

By understanding the importance and meaning of genetic influences and by further understanding the impact of the environment, steps can be taken to reduce the problem of IPA. First, genetic predispositions for IPA imply that there are biological processes that are associated with aggression, and medications might be made to alter those processes. Second, environments that are responsible for, or conducive to, IPA can be modified. Finally, behavioral genetic research on aggression in general shows that it is the combination of genetic and environmental risk factors that exponentially increase the chances for aggressive behaviors; because these genotype-environment interactions have been found for aggressive behaviors in general, they may also be operating on IPA. Therefore, we may be able to target those individuals who are at risk genetically for IPA and alter those environments that are responsible for allowing this genetic predisposition to fully express itself (Cadoret, Leve, & Devor, 1997).

The present findings suggest several avenues for future research. For example, possible genetic mediators for IPA should be considered. That is, do genetic influences in IPA arise because the tendency to use or receive aggression is related to other genetically influenced traits of the individual? Two criteria must be fulfilled for a trait to be considered as a possible genetic mediator. First, the trait must be genetically influenced. Second, the trait must covary with IPA. Examples of possible genetic mediators include impulsive sensation seeking, substance use, and extrafamilial aggression.

Possible genetic and environmental differences due to gender and age should also be examined. The current sample was too small to investigate these possible differential effects, but it is possible that males and females may have differential underlying genetic predispositions towards aggressive behaviors. Another exciting avenue of research is age-related changes in the genetic and environmental architecture of IPA. It has been well documented that as people age, they tend to behave less aggressively, both within and outside their families (O'Leary et al., 1989; Rushton, Fulker, Neale, Nias, & Eysenck, 1989; Suitor et al., 1990). However, we do not know whether this decline in aggression is due to genetic or environmental influences or both. Furthermore, as people age, there may be increasing or decreasing genetic and environmental influences. For example, there is evidence for increasing heritability from childhood, to adolescence, to adulthood, for a range of antisocial behaviors in both males and females (Jacobson et al., 2002). Although these results are counter to assumptions that environmental influences become more important throughout the lifespan because we continually accrue life experiences, they are consistent with the notion of an active genotype-environment correlation (i.e., with age people choose environments that are congruent with their genetic predispositions).

This preliminary study of genetic and environmental influences on the use and receipt of psychological and physical aggression within intimate relationships provides evidence that familial resemblance for these behaviors is influenced by shared genes, not shared environments, as a social learning theory explanation for familial resemblance would imply. The environment *is* important, but not in the way that has previously been conceptualized. The present results indicate that environmental influences result in differences, not similarities, between family members. Although replication with larger, more diverse samples is needed, these findings suggest that researchers of IPA may have to rethink their explanations for why aggression tends to run in families.

REFERENCES

Archer, J. (2000). Sex differences in aggression between heterosexual partners: A meta-analytic review. *Psychological Bulletin, 126*, 651–680.

Cadoret, R. J., Leve, L. D., & Devor, E. (1997). Genetics of aggressive and violent behavior. *Psychiatric Clinics of North America, 20*, 301–322.

Cadoret, R. J., O'Gorman, T. W., Troughton, E., & Heywood, E. (1985). Alcoholism and antisocial personality: Interrelationships, genetic, and environmental factors. *Archives of General Psychiatry, 42*, 161–167.

Cappell, C., & Heiner, R. B. (1990). The intergenerational transmission of family aggression. *Journal of Family Violence, 5*, 135–152.

Carey, G., & Goldman, D. (1997). The genetics of antisocial behavior. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), *Handbook of antisocial behavior* (pp. 243–254). New York: John Wiley and Sons.

Cloninger, C. R., & Gottesman, I. I. (1987). Genetic and environmental factors in antisocial behavior disorders. In S. A. Mednick, T. E. Moffitt, & S. A. Stack (Eds.), *The causes of crime: New biological approaches* (pp. 92–109). Cambridge, England: Cambridge University Press.

Coccaro, E. F., Bergeman, C. S., Kavoussi, R. J., & Scroczynski, A. D. (1997). Heritability of aggression and irritability: A twin study of the Buss-Durkee Aggression Scales in adult male subjects. *Biological Psychiatry, 41*, 273–284.

DiLalla, L. F., & Gottesman, I. I. (1991). Biological and genetic contributors to violence—Widom's untold tale. *Psychological Bulletin, 109*, 125–129.

Eaves, L. J., Eysenck, H. J., & Martin, N. G. (1989). *Genes, culture and personality*. San Diego, CA: Academic Press.

Eron, L. D. (1997). The development of antisocial behavior from a learning perspective. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), *Handbook of antisocial behavior* (pp. 140–147). New York: John Wiley and Sons.

Gelles, R. J. (1979). *Family violence*. Beverly Hills, CA: Sage.

Gottesman, I. I., Goldsmith, H. H., & Carey, G. (1997). A developmental and genetic perspective on aggression. In N. L. Segal, G. E. Weisfeld, & C. C. Weisfeld (Eds.), *Uniting psychology and biology: Integrative perspectives on human development* (pp. 107–130). Washington, DC: American Psychological Association.

Herzberger, S. D. (1996). *Violence within the family: Social psychological perspectives*. Boulder, CO: Westview Press.

Hines, D. A., & Saudino, K. J. (2002). Intergenerational transmission of intimate partner violence: A behavioral genetic perspective. *Trauma, Violence, and Abuse, 3*, 210–225.

Hines, D. A., & Saudino, K. J. (2003). Gender differences in psychological, physical, and sexual aggression among college students using the Revised Conflict Tactics Scales. *Violence and Victims, 18*, 197–218.

Hotaling, G. T., Straus, M. A., & Lincoln, A. J. (1990). Intrafamily violence and crime and violence outside the family. In M. A. Straus & R. J. Gelles (Eds.), *Physical violence in American families: Risk factors and adaptation to violence in 8,145 families* (pp. 431–472). New Brunswick, NJ: Transaction.

Hutchings, B., & Mednick, S. A. (1977). Criminality in adoptees and their adoptive and biological parents: A pilot study. In S. A. Mednick & K. O. Christiansen (Eds.), *Biosocial bases of criminal behavior* (pp. 127–141). New York: Gardner Press.

Jacobson, K. C., Prescott, C. A., & Kendler, K. S. (2002). Sex differences in genetic and environmental influences on development of antisocial behavior. *Development and Psychopathology, 14*, 395–416.

Johnson, M. P. (1995). Patriarchal terrorism and common couple violence: Two forms of violence against women. *Journal of Marriage and the Family, 57*, 283–294.

Kalmuss, D. (1984). The intergenerational transmission of marital aggression. *Journal of Marriage and the Family, 46*, 11–19.

Kasian, M., & Painter, S. L. (1992). Frequency and severity of psychological abuse in a dating population. *Journal of Interpersonal Violence, 7*, 350–364.

Kendler, K. S., Neale, M., Kessler, R., Heath, A., & Eaves, L. (1993). A twin study of recent life events and difficulties. *Archives of General Psychiatry, 50*, 789–796.

Lackey, C. (2003). Violent family heritage, the transition to adulthood, and later partner violence. *Journal of Family Issues, 24*, 74–98.

Loehlin, J. C. (1987). *Latent variable models: An introduction to factor, path, and structural analysis*. Hillsdale, NJ: Erlbaum.

Lyons, M. J., True, W. R., Eisen, S. A., Goldberg, J., Meyer, J. M., Faraone, S. V., et al. (1995). Differential heritability of adult and juvenile antisocial traits. *Archives of General Psychiatry, 52*, 906–915.

Magnus, P., Berg, K., & Nance, W. E. (1983). Predicting zygosity in Norwegian twin pairs born 1915–1960. *Clinical Genetics, 24*, 103–112.

McGue, M., Bacon, S., & Lykken, D. T. (1993). Personality stability and change in early childhood: A behavioral genetic analysis. *Developmental Psychology, 29*, 96–109.

McGue, M., & Bouchard, T. J. (1984). Adjustment of twin data for effects of age and sex. *Behavior Genetics, 14*, 325–343.

Miles, D. R., & Carey, G. (1997). Genetic and environmental architecture of human aggression. *Journal of Personality and Social Psychology, 72*, 207–217.

Moffitt, T. E., Krueger, R. F., Caspi, A., & Fagan, J. (2000). Partner abuse and general crime: How are they the same? How are they different? *Criminology, 38*, 199–232.

Morse, B. J. (1995). Beyond the conflict tactics scale: Assessing gender differences in partner violence. *Violence and Victims, 10*, 251–272.

Neale, M., Boker, S. M., Xie, G., & Maes, H. H. (2002). *Mx: Statistical modeling* (6th ed.). Available from Virginia Commonwealth University, Department of Psychiatry, VCU Box 900126, Richmond, VA 23298.

Neale, M., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. Dordrecht, Netherlands: Kluwer.

O'Leary, K. D., Barling, J., Arias, I., Rosenbaum, A., Malone, J., & Tyree, A. (1989). Prevalence and stability of physical aggression between spouses: A longitudinal analysis. *Journal of Consulting and Clinical Psychology, 57*, 263–268.

Plomin, R., DeFries, J. C., McClearn, G. E., & Rutter, M. (1997). *Behavioral genetics* (3rd ed.). New York: W. J. Freeman and Company.

Raine, A. (1993). *The psychopathology of crime: Criminal behavior as a clinical disorder*. San Diego, CA: Academic Press.

Rowe, D. C. (1994). *The limits of family influence: Genes, experience, and behavior*. New York: Guilford.

Rushton, J. P., Fulker, D. W., Neale, M. C., Nias, D. K., & Eysenck, H .J. (1989). Ageing and the relation of aggression, altruism and assertiveness scales to the Eysenck Personality Questionnaire. *Personality and Individual Differences, 10*, 261–263.

Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype-environment effects. *Child Development, 54*, 424–435.

Schmitz, S., Cherny, S. S., & Fulker, D. W. (1998). Increase in power through multivariate analyses. *Behavior Genetics, 28*, 357–363.

Seroczynski, A. D., Bergeman, C. S., & Coccaro, E. F. (1999). Etiology of the impulsivity/aggression relationship: Genes or environment? *Psychiatry Research, 86*, 41–57.

Silverman, J. G., & Williamson, G. M. (1997). Social ecology and entitlements involved in battering by heterosexual college males: Contributions of family and peers. *Violence and Victims, 12*, 147–164.

Simons, R. L., Wu, C. I., Johnson, C., & Conger, R. D. (1995). A test of various perspectives on the intergenerational transmission of domestic violence. *Criminology, 33*, 141–172.

Stith, S. M., Rosen, K. H., Middleton, K. A., Busch, A. L., Lundeberg, K., & Carlton, R. P. (2000). The intergenerational transmission of spouse abuse: A meta-analysis. *Journal of Marriage and the Family, 62*, 640–654.

Straus, M. A., Hamby, S. L., Boney-McCoy, S., & Sugarman, D. B. (1996). The revised conflict tactics scales (CTS2). *Journal of Family Issues, 17*, 283–316.

Suitor, J. J., Pillemer, K., & Straus, M. A. (1990). Marital violence in a life course perspective. In M. A. Straus & R. J. Gelles (Eds.), *Physical violence in American families: Risk factors and adaptation to violence in 8,145 families* (pp. 305–320). New Brunswick, NJ: Transaction.

Tellegen, A., Lykken, D. T., Bouchard, T. J., Wilcox, K. J., Segal, N. L., & Rich, S. (1988). Personality similarity in twins reared apart and together. *Journal of Personality and Social Psychology, 54*, 1031–1039.

Widom, C. S. (1989). Does violence beget violence? A critical examination of the literature. *Psychological Bulletin, 106*, 3–28.

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