J Fam Viol (2007) 22:121–129
DOI 10.1007/s10896-006-9059-6

ORIGINAL ARTICLE

Etiological Similarities Between Psychological and Physical Aggression in Intimate Relationships: A Behavioral Genetic Exploration

Kimberly J. Saudino · Denise A. Hines

Published online: 30 March 2007

Abstract Previous research has consistently shown that there is a strong association between psychological and physical aggression in intimate relationships. Theories as to why this association exists include that they have a single underlying etiology with differing thresholds, or they have separate etiologies and there is a two-step process by which psychological aggression moves to physical. The current study suggests that these two theories are not necessarily competing theories. The genetic and environmental covariance between psychological and physical intimate partner aggression were examined in 134 monozygotic (MZ) and 41 dizygotic (DZ) twin pairs. Results showed that psychological and physical aggression have largely the same genetic etiology, and any differences between the two are a function of differing nonshared environmental influences.

Keywords Physical abuse · Psychological abuse · Intimate partner violence · Etiology · Genetic

Studies that have investigated the link between the use of physical and psychological aggression in intimate relationships consistently show that the two are highly intercorrelated (e.g., Hines & Saudino, 2003; Murphy & O'Leary, 1989; Stets, 1990; Straus, 1974). Although many people who use psychological aggression in intimate relationships do not also use physical aggression, the reverse is not true. That is, there are few relationships in which physical aggression occurs without psychological aggression (e.g., Follingstad *et al*., 1990; Stets, 1990). Moreover, psychological aggression has been shown to predate physical aggression (Murphy & O'Leary, 1989) and psychological and physical aggression share many of the same antecedents, including younger age, fewer children in the family, and increased drunkenness and drug use (Straus & Sweet, 1992). Thus, there is a strong association between these two types of aggression in intimate relationships, and the etiologies of this association have been the subject of much debate.

This strong positive association between psychological and physical aggression has been theorized to be due to one of many processes (Stets, 1990). Some have speculated that psychological aggression may be used as a substitute for physical aggression when conflict occurs between intimate partners; that is, when partners have negative feelings for each other, they may choose to release these feelings through psychological rather than physical aggression, a theory known as the "catharsis theory" of aggression. This particular theory has generally received little support in the literature because venting aggressive feelings usually *increases*, not decreases, the likelihood of aggressive behavior. Furthermore, catharsis theory would predict a negative association between psychological and physical aggression; however, research consistently shows that there is a positive association (Stets, 1990).

An alternative explanation is that psychological and physical aggression are both manifestations of a single underlying etiology (i.e., aggressive behavior), and they have the same threshold for instigation. This explanation has also received little support in the literature because it would suggest that where there is psychological aggression there is also physical aggression, and studies consistently show that there are many couples who engage in psychological aggression but

K. J. Saudino
Department of Psychology, Boston University,
Boston, USA

D. A. Hines (✉)
Department of Criminal Justice,
University of Massachusetts Lowell, 870 Broadway St.,
Lowell, MA 01854, USA
e-mail: Denise_Hines@uml.edu

not physical aggression (e.g., Hines & Saudino, 2003; Stets, 1990). A related explanation suggests that psychological and physical aggression are different manifestations with different thresholds of a construct that has a single underlying etiology. This explanation is more plausible because it suggests that when we observe physical aggression, we will also observe psychological aggression, but not necessarily vice versa, a result that is consistent with most literature (e.g., Follingstad *et al*., 1990; Murphy & O'Leary, 1989; Stets, 1990).

A final theory that has been proposed to explain the association between these two types of aggression is that psychological and physical aggression have separate underlying etiologies and there is a two-step process by which psychological aggression moves into physical aggression (Stets, 1990). The first step represents a process by which, as a result of certain influences, a person moves from being nonaggressive into being psychologically aggressive. In the second step, a person who is psychologically aggressive would then become physically aggressive as a result of factors that are unique to physical aggression.

In the only empirical analysis to date that compares the strength of these two latter explanations, Stets (1990) favored the second explanation over the first. That is, it was concluded that psychological and physical aggression have two distinct underlying etiologies, and what explains the association between the two is the step that individuals take to move from psychological aggression to physical aggression. One flaw in Stets' (1990) analyses was that she assumed that each of these forms of aggression has a single underlying etiology. However, aggression, whether psychological or physical, is a complex phenomenon that is likely influenced by several underlying etiologies. Thus, these two seemingly divergent explanations for the association between physical and psychological aggression may not actually be competing. Physical and psychological aggression may have some etiologies in common, *and* there may also be a two-step process whereby psychological aggression moves into physical aggression, a process which is influenced by etiologies that differ between the two types of aggression.

A behavioral genetic approach

Behavioral genetic methods, such as the twin design, offer a strong test of these two explanations because they allow for the investigation of common genetic and environmental variance between variables. If two variables are correlated, it means that there is some overlap in the factors that influence them. Multivariate behavior genetic methods can be used to investigate whether this covariance is due to the same genetic and/or environmental influences.

The twin method involves comparing genetically identical (monozygotic; MZ) twins with fraternal (dizygotic; DZ) twins who share approximately 50% of their segregating genes. Genetic influences are implied when co-twin similarity covaries with the degree of genetic relatedness. Intraclass correlations typically serve as indices of co-twin 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). Under the simple twin design, h^2 is the proportion of observed variance for a trait that can be attributed to additive genetic influences (i.e., the sum of the average effect of all genes that influence a trait). The remaining variance is attributed to environmental factors which include all non-heritable influences. The environmental variance component can be further 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 co-twin similarity. Possible shared environmental influences include socioeconomic status, 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 co-twin 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 and serve to make twins different from one another on a given trait. Non-shared environmental influences can include different peer groups, teachers, or parental treatment, or accidents and illnesses that occur in only one twin. 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). It should be noted that in quantitative genetic analyses, these genetic and environmental variance components are anonymous. That is, just as twin studies do not specify which particular genes are involved in a behavior, they do not specify the particular environments that operate on the behavior. Instead, they explain how much of the observed variance in behavior can be attributed to genetic and environmental influences generally. Multivariate genetic analyses (described in Methods) examine genetic and environmental contributions to the *covariance* between two measures rather than the variance of each measure considered separately (Plomin & DeFries, 1979).

It is possible that physical and psychological aggression may have the same genetic etiology but different environmental etiologies, or vice versa. For example, a recent study of male twins investigated the genetic and environmental correlations between several different types of extrafamilial

aggression (Coccaro, Bergeman, Kavoussi, & Scroczynski, 1997). Specifically, the researchers found that genetic influences explained 69% of the variance in physical assault, 63% of the variance in indirect aggression (e.g., throwing things, slamming doors), and 53% of the variance in verbal aggression (e.g., swearing at, name calling). For each type of aggression, the remaining variance was due to non-shared environmental influences (i.e., there was no evidence of shared environmental influences). Physical assault and indirect aggression were correlated at .36, and the genetic overlap between these two types of aggression was 42%, whereas the non-shared environmental overlap was 58%. Physical assault and verbal aggression were correlated at .50, and the genetic overlap between these two variables was estimated at 16%, whereas the non-shared environmental overlap was 84%. Thus, there were genetic and non-shared environmental influences that were common between physical and nonphysical aggression, but there were also genetic and non-shared environmental influences that were specific to each form of aggression. In other words, there were multiple etiologies for these types of aggression, some of which were shared between the types and some of which differed.

Previous research on the sample in the current twin study has shown that the use and receipt of both psychological and physical intimate partner aggression are genetically influenced (Hines & Saudino, 2004). Specifically, genetic influences account for 16% of the variance in the use of intimate partner physical aggression and 15% of the variance in its receipt; for psychological aggression, genetic influences account for 22% of the variance in its use and 25% of the variance in its receipt. Similar to Coccaro *et al*. (1997), the remaining variance for the use and receipt of both physical and psychological aggression is accounted for by non-shared environmental influences, with no evidence of shared environmental influences. Thus, the association that has been observed between physical and psychological aggression in the literature could be due to overlapping genetic and/or non-shared environmental influences.

In the present study we first explore the extent to which physical and psychological aggression are associated. Then, using multivariate behavior genetic methods we investigate the extent to which this association is due to common genetic and/or environmental influences. Therefore, the purpose of the current study was to investigate through the use of multivariate behavioral genetic methods whether there is a common underlying etiology between psychological and physical aggression, whether their etiologies are entirely distinct, or perhaps whether there are some common *and* unique aspects underlying the etiologies of both. Based on the only previous behavioral genetic study to investigate these issues in extrafamilial aggression (Coccaro *et al*., 1997), it was hypothesized that there would be some common genetic and environmental overlap between the two types of aggression, but also some genetic and environmental effects specific to each.

Methods

Participants included 185 pairs of same-sex twins (144 MZ, 41 DZ) with a mean age of 40.7 years ($SD = 15.0$), who had experience in an adult intimate relationship. Participants were recruited at twin conventions, through advertisements that were placed on twin Internet sites, flyers posted in various cities and towns throughout the United States, and through word of mouth. To mask the true purpose of the study and thus reduce the likelihood of recruitment bias, potential twins were told that the study was a questionnaire study 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 (e.g., they completed it with regard to their twin relationship; they stated that they had never been involved in a romantic relationship and left it blank); therefore, genetic analyses are based on 134 MZ and 41 DZ twins. The majority of these participants were White (91.9%) and female (85.9%). Furthermore, the majority of twins were heterosexual (96.6%) and currently involved in a romantic relationship (71.1%); 55.1% were married and the average length of relationship was 11.8 years ($SD = 12.8$). The average socioeconomic status (SES), according to the Hollingshead index, was predominantly middle class, although the participants ranged from working class to upper class. Further demographic details can be found in our previous description of this sample (Hines & Saudino, 2004).

Measures

Zygosity questionnaire

Zygosity was determined through a physical similarity questionnaire completed by both members of the twin pair (Magnus *et al*., 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 *et al*., 1989). It is important to note that any misclassification of either MZ or DZ twins would mathematically work *against* the genetic hypothesis (i.e., result in underestimates of heritability).

The Revised Conflict Tactics Scales

The Revised Conflict Tactics Scales (*CTS2*, Straus *et al*., 1996) contain 78 items assessing the amount of negotiation, psychological aggression, physical aggression, sexual coercion, and injuries that occur between couples, as reported by the participants. For the current study, only the psychological and physical aggression subscales were analyzed. The *CTS2* has demonstrated good construct and discriminant validity and good reliability with internal consistencies ranging from .79 to .95 (Straus *et al*., 1996).

To examine the severity and frequency of aggressive acts, the *chronicity* of each of the subscales was computed. For each participant, the number of acts used and received in the previous year on each of the subscales was computed. Participants indicated on a scale from 0 to 6 how many times in the previous year they experienced the acts listed (0 = 0 times; 1 = 1 time; 2 = 2 times; 3 = 3–5 times; 4 = 6–10 times; 5 = 11–20 times; 6 = more than 20 times). Furthermore, they indicated whether or not they had *ever* used any of these acts in the current relationship. Many participants indicated that they had used several types of aggressive acts, but not in the previous year, and in order to increase the power of our analyses, the data were transformed 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 past year; 3 = twice in the past year; 4 = 3–5 times in the past year; 5 = 6–10 times in the past year; 6 = 11–20 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 new 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).

Analyses

Multivariate behavior genetic analyses allow for the examination of genetic and environmental contributions to the *covariance* between two measures rather than the variance of each measure considered separately (Plomin & DeFries, 1979). A preliminary step in the multivariate analysis of covariance is the cross-twin correlation. The cross-twin correlation involves correlating Twin 1's score on measure A (e.g., use of psychological aggression) with Twin 2's score on measure B (e.g., use of physical aggression) and vice versa. Genetic contributions to the covariance between two measures are implied when the MZ cross-twin correlation is greater than the DZ cross-twin correlation.

In the present study, maximum-likelihood model-fitting analyses were performed on twin variance/covariance matrices using *Mx* maximum-likelihood model-fitting procedures (Neale *et al*., 2002). We examined genetic and environmental sources of covariance between the *use* of psychological and physical aggression. We also explored sources of covariance for the *receipt* of psychological and physical aggression. As illustrated in Fig. 1, bivariate genetic models were used to assess the genetic and environmental contributions to the phenotypic correlation (i.e., observed correlation) between the use of psychological and physical aggression, and the receipt of psychological and physical aggression. This model partitions the phenotypic covariation between the two traits 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 psychological aggression, and A2, C2, and E2 refer to the additive genetic, shared, and nonshared environmental influences on physical 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 psychological and physical aggression. The genetic correlation is the extent to which the genetic influences

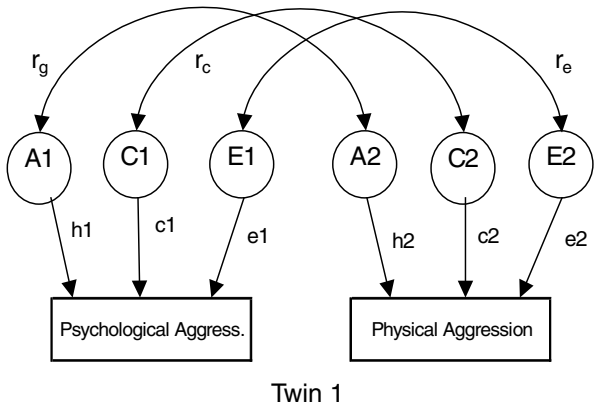

Fig. 1 Bivariate Model: *A1* = Latent Additive Genetic Effects on Psychological Aggression; *C1* = Latent Shared Environmental Effects on Psychological Aggression; *E1* = Latent Nonshared Environmental Effects on Psychological Aggression; *A2* = Latent Additive Genetic Effects on Physical Aggression; *C2* = Latent Shared Environmental Effects on Physical Aggression; *E2* = Latent Nonshared Environmental Effects on Physical Aggression; r_g = genetic correlation; r_c = shared environmental correlation; r_e = nonshared environmental correlation; *h1* = additive genetic effects on psychological aggression; *c1* = shared environmental effects on psychological aggression; *e1* = nonshared environmental effects on psychological aggression; *h2* = additive genetic effects on physical aggression; *c2* = shared environmental effects on physical aggression; *e2* = nonshared environmental effects on physical aggression

Table 1 Prevalence and frequency of psychological and physical aggression

	Total ($n = 370$)	MZ twins ($n = 268$)	DZ twins ($n = 82$)
Psychological aggression			
% Used	83.1%	82.0%	86.6%
Mean # of acts (*SD*)	10.7 (7.8)	10.7 (7.8)	10.6 (7.4)
% Received	78.8%	78.3%	80.5%
Mean # of acts (*SD*)	11.0 (8.1)	10.7 (7.9)	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%	23.2%	26.8%
Mean # of acts	6.5 (6.8)	7.1 (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.

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

In addition to estimating the degree of genetic and environmental covariance between two measures, the multivariate model also permits the estimation of genetic and environmental contributions to the phenotypic correlation. That is, to what extent do common genetic and/or environmental factors contribute to the observed (i.e., phenotypic) correlation? In Fig. 1, the genetic contribution is estimated as the product of the genetic paths linking the two variables (i.e., $h1 \times r_g \times h2$). Environmental contributions to the phenotypic correlation are derived similarly (i.e., $e1 \times r_e \times e2$). When the model fits the data well, summing across the genetic and environmental contributions yields a reasonable estimate of the phenotypic correlation.

Three criteria were used to determine whether the bivariate model represented a good fit of the data: 1) a non-significant χ^2; 2) a low Akaike Information Criterion (*AIC*), which equals the χ^2 minus twice the *df*; a model with a low *AIC* value has the optimal combination of goodness-of-fit and parsimony, and 3) a low Root Mean Squared Error Approximation (*RMSEA*), which provides a measure of fit that is independent of sample size; models with an *RMSEA* lower than .05 represent good fits; those with an *RMSEA* lower than .10 represent adequate fits, and those with an *RMSEA* greater than .10 represent poor fits (Neale *et al*., 2002).

Results

Table 1 presents the prevalence and frequency of psychological and physical aggression, overall and stratified by zygosity, 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 in the prevalence or frequency of each of the subscales between MZ and DZ twins.

As has been found in other studies, there was a significant phenotypic correlation between psychological and physical aggression (Use: $r = .38$, $p < .001$; Receipt: $r = .46$, $p < .001$). As a preliminary step in evaluating genetic and environmental influences on the covariance between physical and psychological intimate partner aggression, twin cross correlations were calculated using the double-entry procedure. For the use of psychological and physical aggression, the significant MZ cross correlation ($r = .15$, $p < .05$) exceeded the non-significant DZ cross correlation ($r = -.03$, *ns*). Similarly, for the receipt of psychological and physical aggression, the significant MZ cross correlation ($r = .22$, $p < .01$) exceeded the non-significant DZ cross correlation ($r = -.01$, *ns*). This pattern suggests that the phenotypic correlations between both the use of psychological and physical aggression and the receipt of psychological and physical aggression are mediated genetically.

Model-fitting procedures provide a more elegant analysis of genetic and environmental covariance than cross correlations because they estimate multiple parameters simultaneously, test the model, make assumptions explicit, provide parameter estimates, and permit tests of alternative models if necessary (Loehlin, 1987; Neale & Cardon, 1992). Because prior univariate analyses indicated that the use and receipt of psychological and physical aggression were influenced by only genetic and nonshared environmental influences (see

Table 2 Bivariate model-fitting results

Parameter estimates (95% C.I.)												
Psych. aggression used			**Physical aggression used**					**Goodness-of-fit measures**				
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	–	.77	.17	–	.83	.74	–	.30	9.30	14	.81	.04	−18.70
(.07, .38)		(.63, .93)	(.01, .32)		(.68, .99)	(.18, 1.0)		(.15, .43)					
Physical aggression received			**Phys. aggression received**										
.26	–	.74	.17	–	.83	1.00	–	.32	8.11	14	.88	.02	−19.89
(.10, .40)		(.60, .90)	(.04, .31)		(.69, .96)	(.65, 1.0)		(.19, .44)					

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.

Hines & Saudino, 2004), shared environmental influences cannot contribute to the covariance between psychological and physical aggression; thus c^2 was not included in our bivariate model-fitting analyses.

For the use of aggression, the bivariate model provided a good fit to the data (Table 2). Moreover, the high genetic correlation ($r_g = .74$) indicates that most of the genetic influences for the use of psychological aggression overlap with the genetic influences for the use of physical aggression. There is also some overlap between nonshared environmental influences on the use of psychological and physical aggression ($r_e = .30$). Similar results emerged for the receipt of aggression. The model fit the data well and showed that all of the genetic influences responsible for the receipt of psychological aggression overlap with those for the receipt of physical aggression ($r_g = 1.00$). Furthermore, some of the nonshared environments responsible for the receipt of psychological aggression overlap with those responsible for the receipt of physical aggression ($r_e = .32$).

Figure 2 presents the genetic and nonshared environmental contributions to the phenotypic correlation between the use and receipt of psychological and physical aggression. As can be seen, genetic influences accounted for over one-third (.14/.38) of the phenotypic correlation between the use of psychological and physical aggression. In other words, although the same genetic effects operate on the use of psychological and physical aggression, the phenotypic correlation between the use of psychological and physical aggression is primarily due to overlapping nonshared environmental influences (.24/.38). For the receipt of psychological and physical aggression, genetic influences accounted for almost one-half of the phenotypic correlation (.21/.46). Thus, the phenotypic correlation between the receipt of psychological and physical intimate partner aggression is due approximately equally to overlapping genetic and nonshared environmental influences.

Discussion

Consistent with previous studies (e.g., Stets, 1990), psychological and physical aggression were significantly correlated in the current study. Moreover, there was considerable overlap between the factors that influence both. For both the use and receipt of aggression, the high genetic correlations between psychological and physical aggression suggests that they are influenced by essentially the same genetic factors. However, the nonshared environmental factors that influence each seem to have some independence as indicated by the low nonshared environmental correlations between psychological and physical aggression. Thus, although there was some overlap in the nonshared environmental influences that

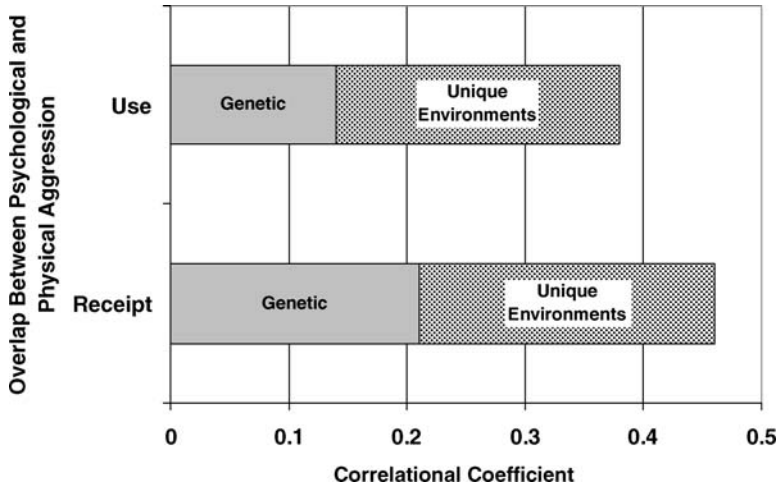

Fig. 2 Genetic and environmental contributions to the phenotypic correlations between psychological and physical aggression

operate on each type of aggression, the etiological differences between the two are largely a function of nonshared environmental influences.

Therefore, it seems that there are both etiologic similarities and independence in the factors that influence psychological and physical aggression. These findings are consistent with both of the seemingly competing explanations for the associations found between physical and psychological intimate partner aggression in prior research. That is, consistent with the explanation that Stets (1990) rejected, we found that physical and psychological aggression have the same underlying *genetic* etiology and that they share some nonshared environmental etiologies. Furthermore, in agreement with Stets' (1990) explanation, we found that physical and psychological intimate partner aggression are influenced by largely separate nonshared environmental etiologies.

Our results are also consistent with those from the only behavioral genetic study to address the overlap between physical and psychological forms of extrafamilial aggression. In that study, for both types of nonphysical forms of aggression (i.e., indirect and verbal aggression) there was some significant genetic and environmental overlap with physical aggression, but also some independence for both the genetic and environmental influences (Coccaro *et al*., 1997). Contrary to their study, however, our results showed that there was little to no independence for the genetic influences on physical and psychological intimate partner aggression. These differences may arise for several reasons, including 1) measuring intrafamilial versus extrafamilial aggression may lead to different results, or 2) analyzing different types of psychological aggression (e.g., verbal versus indirect) versus a measure designed to assess intimate partner psychological aggression in general may lead to different results. Future studies should address this issue by analyzing the genetic and environmental covariance of different types of intimate partner psychological aggression (e.g., intimidation, withdrawal) with physical aggression.

An interpretation of our results could be that people who have a genetic predisposition towards engaging in aggressive behavior will engage in different *types* of aggressive behaviors depending upon their differences in nonshared environmental influences. That is, there may be a modest genetic predisposition towards aggressive behavior, but what determines whether someone is physically versus psychologically aggressive is primarily due to environmental experiences. Specifically, it is those environmental experiences that are unique to each member of a family that are essential in explaining individual differences in aggressive responses, and differences between types of aggressive behaviors. Most likely, these nonshared environmental influences will be negative, such as stress or high exposure to violence. The current study cannot test this particular hypothesis because the genetic and environmental influences we tested were latent; therefore, future studies investigating specific nonshared environmental influences that may be responsible for differentiating those people who use psychological aggression from those who use physical aggression are needed. In addition, future research should also explore specific genes that are responsible for physical and psychological aggression.

Although the nonshared environmental factors that influence psychological aggression were largely independent from those that influence physical aggression and genetic effects were almost completely overlapping, the phenotypic association between the two types of aggression was due primarily to common nonshared environments, not common genes. This occurs because even though the genetic covariance between types of aggression is high, genes explain only a modest proportion of variance for each; whereas although the nonshared environmental covariance is modest, nonshared environmental factors explain a substantial proportion of variance for each. Consequently, relative to genetic effects, nonshared environments can account for a larger proportion of covariance between the two types of aggression. What this means is that the modest nonshared environments

that are common to both psychological and physical aggression are primarily responsible for the correlation between the two.

At this point, we must clearly define what is meant when we say that behaviors are genetically influenced and correlated. First and foremost, it means that 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 *et al*., 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, 1993). Therefore, even though we found genetic influences for psychological and physical intimate partner aggression and evidence that most, if not all, of the genes operating on one type of aggression are operating on the other, it does not mean that people who are genetically predisposed to engage in aggressive relationship behaviors will necessarily do so. It is important not to ignore the substantial influence of the environment found in this study – the environments of people who are genetically predisposed to behave aggressively can be altered to reduce or eliminate their aggressive behaviors (see Hines & Saudino, 2004 for further explanation).

It is important to also address the genetic and nonshared environmental overlap for the *receipt* of aggressive 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. Therefore, events that may seem external to the individual can have genetic influences. Genetic influences on the receipt of intimate partner aggression 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 correlation, where the victimized individuals choose aggressive romantic partners because those partners are congruent with certain genetically-influenced characteristics of the victims (Scarr & McCartney, 1983). Our previous analyses of the data from this twin sample (Hines & Saudino, 2004) showed that there were strong phenotypic correlations between the use and receipt of psychological aggression and between the use and receipt of physical aggression, which suggests that aggressive people may be choosing aggressive partners (i.e., active genotype-environment correlation). Therefore, the general tendency to become involved in aggressive relationships may be genetically influenced, and the genetic and nonshared environmental overlap between the receipt of psychological and physical aggression may merely reflect the fact that we are probably investigating genetic and environmental overlap on people's tendencies to get involved in aggressive relationships as *both* perpetrators and victims. The fact that the parameter estimates for both models are nearly identical is congruent with this line of thought.

The current study has limitations that should be considered in future research. For example, the sample is small and, because the large majority of participants were White and female, may not be representative of the population overall. However, comparisons between this sample and other samples of intimate partner aggression show remarkably consistent estimates of the prevalence and frequency of aggression (see Hines & Saudino, 2004, for more details). Furthermore, the results from the bivariate model-fitting were strong even with the small sample; therefore, there was adequate power to detect significant genetic and environmental influences and correlations. Nonetheless, future research should concentrate on replicating and expanding these results on a larger, more representative twin sample and on male twins.

Another important point to consider is that the majority of the twins who were physically aggressive engaged in mostly minor forms of physical aggression. Therefore, the results should not be generalized to those who use severe physical aggression (i.e., battering). These battering types of behavior occur infrequently, and because of our small sample size, we were unable to model the genetic and environmental covariance between psychological and severe physical aggression or between minor physical and severe physical aggression, which are both important avenues for future research.

In sum, the current study provided evidence to support Stet's (1990) findings that psychological and physical intimate partner aggression have separate underlying etiologies. Consistent with her study, we found that physical and psychological aggression have some independent environment etiologies. However, we also provided evidence in support of the theory that Stets (1990) rejected; that is, we found that psychological and physical intimate partner aggression have a similar *genetic* etiology. Furthermore, we also found that some of the environmental influences that operate on psychological aggression also operate on physical aggression. Thus, physical and psychological aggression

share a genetic etiology and some of their environmental etiologies, and any differences in etiology is due to differences in the nonshared environmental influences on these behaviors.

Acknowledgements This research was supported by Grant MH64252-01 from the National Institute of Mental Health and the Twins Days Research Grant. Special thanks go to the Family Research Lab and Crimes Against Children Research Center seminar participants for their feedback on a previous version of this manuscript. This paper is based on the doctoral dissertation research of the first author.

References

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.

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

Follingstad, D. R., Rutledge, L. L., Berg, B. J., Hause, E. S., & Polek, D. S. (1990). The role of emotional abuse in physically abusive relationships. *Journal of Family Violence*, *5,* 107–120.

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.

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.

Hines, D. A., & Saudino, K. J. (2004). Genetic and environmental influences on intimate partner aggression: A preliminary study. *Violence and Victims, 19,* 701–718.

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.

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

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

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

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

Murphy, C. M., & O'Leary, K. D. (1989). Psychological aggression predicts physical aggression in early marriage. *Journal of Consulting and Clinical Psychology*, *57,* 579–582.

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

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

Plomin, R., & DeFries, J. C. (1979). Multivariate genetic analysis of twin data on scholastic abilities. *Behavior Genetics*, *9,* 505–517.

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.

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

Stets, J. E. (1990). Verbal and physical aggression in marriage. *Journal of Marriage and the Family*, *52,* 501–514.

Straus, M. A. (1974). Leveling, civility, and violence in the family. *Journal of Marriage and the Family*, *36,* 13–29.

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

Straus, M. A., & Sweet, S. (1992). Verbal/symbolic aggression in couples: Incidence rates and relationships to personal characteristics. *Journal of Marriage and the Family*, *54,* 346–357.