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**Indicators of Domestic/Intimate Partner Violence are Structured by
Genetic and Nonshared Environmental Influences**

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Abstract

One of the most consistent findings to emerge from domestic/intimate partner violence (IPV) research is that IPV tends to “run in the family.” Social learning theories appear to be consistent with empirical data, but almost no attention has been given to alternative explanations, including that genetic factors explain intergenerational transmission of IPV. Data for this study were drawn from wave 4 of the National Longitudinal Study of Adolescent Health (Add Health). Three indicators of IPV were measured and genetic factors accounted for 24% of the variance in hitting one’s partner, 54% of the variance in injuring one’s partner, and 51% of the variance in forcing sexual activity on one’s partner. The shared environment explained none of the variance across all three indicators and the nonshared environment explained the remainder of the variance. These findings point to the importance of genetic factors in the etiology of IPV.

Domestic and intimate partner violence (IPV) remains a serious public health concern due to the eminent and ubiquitous threat of harm that is imposed on victims. Recent evidence from the National Intimate Partner and Sexual Violence Survey suggests an estimated 11 million women will be raped by an intimate partner in their lifetime and 39 million women will experience physical violence at the hands of an intimate partner (Black et al., 2011). Prevalence rates for males are substantially lower for the experience of rape (indeed, stable estimates were unable to be calculated) but are similar for physical violence with nearly 32 million men reporting a victimization experience over their lifetime (Black et al., 2011). Other self-report surveys paint a similarly bleak picture. National Crime Victimization Survey data from 2010 revealed that nearly 1 out of every 1,000 males and 3 out of every 1,000 females experienced IPV in that year (Truman, 2011). Silverman et al. (2001), drawing on survey data taken from females in grades 9 through 12, reported that approximately 1 in 5 female students had already experienced physical and/or sexual violence from an intimate partner.

Given the high prevalence of IPV in the U.S., research into the consequences of victimization has taken on an important role in the scientific community. Indeed, scholars have begun to reveal the myriad negative consequences associated with being the victim of IPV. Female victims of dating violence, for example, have been found to be at a greater risk for a variety of negative outcomes such as substance use, unhealthy weight control, pregnancy, sexually-transmitted diseases, depression, and suicide (Campbell, 2002; Coker et al., 2002; Silverman et al., 2001). Scholars have also shown IPV to be associated with negative outcomes for male victims (Coker et al., 2002) and for those who are not directly victimized but have witnessed an IPV event (Chio et al., 2012; Straus, 1992). In a review of more than 30 research

articles, Edleson (1999) concluded that behavioral, emotional, and cognitive-functioning problems were correlated with a child's exposure to domestic violence.

Due to the threat it poses to public health and safety, some research has begun to focus on the causes and correlates of IPV. Research suggests that certain traits, some that can be traced to early childhood, are predictive of one's likelihood of committing intimate partner violence. One of the most consistent findings to emerge from domestic violence research is that IPV tends to "run in the family" (Hines & Saudino, 2002; Pinto et al., 2010). Indeed, research has revealed that individuals who are exposed to domestic violence as children are much more likely to be abusers when they reach adulthood (Egeland, 1993; Roberts et al., 2010; Stith et al., 2000) but the theoretical explanation for this association has largely centered on social learning mechanisms while simultaneously dismissing (via omission) the potential role of genetic factors. When considered in terms of IPV, social learning theory (Bandura, 1977) would predict that an individual (e.g., child) who witnesses his/her parent(s) committing IPV are more likely to model that behavior in their own lives, especially when dealing with altercations with loved ones. This occurs because the child observes the behavior being rewarded and reinforced (i.e., the altercation is ended quickly) and thus has a propensity to believe IPV to be a reasonable solution to personal problems. In short, social learning theory attributes much of the causes of IPV to the early rearing environment.

Although much previous literature has leaned on social learning theory to explain the etiology of IPV, some research suggests genetic factors may play a role (Hines & Saudino, 2002, 2004). To be sure, there are at least three reasons to suspect that genetic influences may underlie the etiology of IPV. First, genetic influences are often the primary reason family members resemble one another on any trait (Harris, 1998; Turkheimer, 2000). Second, traits that are

known to be at least partially sculpted by genetic factors, verbal IQ for example (Bouchard et al. 1990), have also been linked with IPV (Lussier et al., 2009). Thus, it may be that certain genetically influenced traits serve as an endophenotype (i.e., a mediator variable) for a genetic influence on IPV. Third, and perhaps most importantly, behavior genetic research has revealed that all human traits are likely to be under at least partial genetic control (Turkheimer, 2000). As of the writing of this paper, however, only one study has addressed this possibility (Hines & Saudino, 2004).¹ Hines and Saudino (2004) reported that intimate partner violence—as measured via the Revised Conflict Tactics Scales (Straus et al., 1996) was around 20% heritable, meaning that genetic factors accounted for roughly 20% of the variance observed in the scales.

Hines and Saudino (2004) provided an important extension of the research into the etiology of IPV by being the first to analyze the genetic influences on this type of behavior. Two points, however, must be considered by future research. First, the sample analyzed by Hines and Saudino was relatively small ($n = 185$ twin pairs) and was not drawn using probability sampling methods. Second, IPV was operationalized as two scales of psychological and physical aggression. These scales included many different behaviors (see Straus et al., 1996) ranging from minor acts (e.g., pushing or shoving partner) to serious acts of violence (e.g., using a knife or gun on partner). Collapsing these diverse forms of IPV together into scales may mask important etiological differences (i.e., introducing heterogeneity). The current study, therefore, sought to extend the Hines and Saudino findings by analyzing a large sample of twins drawn from a probability sample of American adolescents and by separately analyzing indicators of IPV.

METHODS

¹ Hines and Saudino (2007) analyzed the same data and same IPV scales to determine whether psychological and physical forms of IPV shared a genetic etiology.

Data

Data were drawn from the National Longitudinal Study of Adolescent Health (Add Health; Harris, 2009). The Add Health data have been described previously (Harris et al., 2009). Briefly, the Add Health is a nationally representative prospective longitudinal sample of American adolescents. Information has been collected in four waves, beginning with wave 1 in 1994. Wave 1 data collection began as a school-level study, where 132 schools were selected using probability selection methods. All students enrolled in these schools were then asked to complete a self-report questionnaire during class. More than 90,000 students responded and of these participants, a pool of 20,745 was asked to complete a more in-depth follow-up interview (i.e., wave 1 in-home survey). Three more waves of data were collected over the next 13 years. Wave 2 data collection began approximately one year after wave 1 in-home interviews were completed. Wave 3 data collection commenced roughly 6 years after wave 2 interviews were completed. Finally, in 2008, a fourth wave of interviews was conducted. Respondents had reached adulthood during wave 4 interviews (ages ranged between 24 and 34). All data analyzed in the current study were drawn from wave 4 interviews.

One feature of the Add Health data deserves mentioning. To be specific, during wave 1 interviews, twin pairs were identified and added to the sample. During wave 1 data collection, any respondent who identified himself or herself as a twin had his or her co-twin added to the sample with certainty. All total, 578 monozygotic (MZ) twins and 900 dizygotic (DZ) twins were included in the sample. After removing cases with missing data on the measures described below, 462 MZ twins and 721 DZ twins remained in the sample.

Indicators of Intimate Partner Violence (IPV)

Hitting partner. During wave 4 interviews, respondents were asked the following question: “How often (have/did) you (slapped/slap), hit, or (kicked/kick) {initials}?” where {initials} was their current or most recent intimate partner’s initials.² Responses were originally coded on a scale from 0 (*never*) to 7 (*more than 20 times in the last year of the relationship*). More than 90% of the responses were either 0 (*never*) or 1 (*this has not happened in the past year, but it did happen before then*), meaning that there was little justification for retaining the original coding scheme. For the analysis, the variable was dichotomized such that 0 = *never* and 1 = *happened at least once*. Prevalence rates are reported in Table 1. Prevalence rates did not significantly differ across zygosity ($\chi^2 = 2.08, p = .15$).

Injuring partner. Respondents were asked the following question during wave 4 interviews: “How often (has/did) {initials} (had/have) an injury, such as a sprain, bruise, or cut because of a fight with you?” Only respondents who indicated they had hit their partner previously (i.e., the previous question [“Have you hit {initials}?”] was answered with a response other than *never*) were asked this question. Originally, the response options ranged from 0 (*never*) to 7 (*more than 20 times in the last year of the relationship*). Because of limited variation, there was little justification for retaining the original coding scheme. For the analysis, the variable was dichotomized such that 0 = *never* and 1 = *happened at least once*. Respondents who had not hit their partner previously (i.e., the Hitting Partner question [“Have you hit

² The Add Health research team included the following note in the data codebook section where each of the three measures analyzed here appeared: This section is administered for ONE current partner. If there are multiple current partners, priority is: marriage partner, cohabitation partner, pregnancy partner, dating partner. If two or more partners fall in the same type of relationship, the longer/longest relationship is selected. If two or more partners fall in the same type of relationship, and they are of the same duration, then the respondent is asked to pick the partner they care about the most. If there are no current partners then the most recent partner is selected. If there is no current partner and no most recent partner, end dates for each marriage, cohabitation, and relationship with a pregnancy are reviewed to select the one partner with the most recent end date. If two or more partners have the same end date, select the longer/longest relationship.

{initials}”] was answered with a response of *never*) were coded as 0 (*never*) for the current question. Prevalence rates did not significantly differ across zygosity ($\chi^2 = .04, p = .85$).

Forcing sexual activity. Wave 4 respondents were asked one final question regarding intimate partner violence. Specifically, each participant was asked “How often (have/did) you (insisted/insist) on or (made/make) {initials} have sexual relations with you when {he/she} didn’t want to?” The response options originally ranged from 0 (*never*) to 7 (*more than 20 times in the last year of the relationship*), but roughly 95% of all respondents indicated that this had *never* happened (i.e., coded as 0). Due to the limited variation, there was little justification for retaining the original coding scheme. For the analysis, the variable was dichotomized such that 0 = *never* and 1 = *happened at least once*. Prevalence rates did not significantly differ across zygosity ($\chi^2 = .11, p = .74$).

ANALYSIS PLAN

The analysis unfolded in a series of three steps. The first step was to analyze cross-twin correlations for each of the three IPV indicators. Due to the dichotomous coding of the variables, cross-twin correlations were estimated as tetrachoric correlations. These cross-twin correlations provided an initial glimpse into whether genetic factors impact the likelihood of involvement in IPV. To the extent that MZ twins are more similar on any of the three measures as compared to DZ twins, this will preliminarily suggest that genetic factors play a role in the etiology of that behavior. In this way, the analysis utilizes behavior genetic theory (Plomin et al., 2008) to infer genetic and environmental influences. MZ twins should resemble one another more closely than DZ twins on any specific trait because MZ twins share twice as much genetic material (MZ twins share 100% of their DNA) as compared to DZ twins (DZ twins share, on

average, 50% of their distinguishing DNA). Thus, to the extent that the MZ twin correlation (r_{MZ}) is larger than the DZ twin correlation (r_{DZ}), genetic factors may influence the trait.

The second step to the analysis was to estimate three logistic regression models: one for each of the IPV indicators. These analyses allowed us to plot predicted probabilities of IPV as a function of one's co-twin's involvement in IPV across zygosity. Specifically, a logistic regression model was estimated utilizing twin 1's value on the IPV indicator as the dependent variable and twin 2's value as the independent variable (twins were randomly assigned to appear as twin 1 or twin 2). This process was carried out for all twin pairs, for MZ twin pairs only, and then for DZ twin pairs only. As before, to the extent that MZ twins' probability of involvement in IPV is increased *at a greater rate* as compared to DZ twins' probability, genetic influences can be inferred. Standard errors were corrected for the clustering of twins within the same family during this stage of the analysis. Also, respondent gender was controlled when estimating the logistic regression model for all twins and for DZ twins. Substantively identical results were gleaned when the gender control was omitted from the analysis.

The third and final step to the analysis was to estimate the ACE model. The ACE model is a structural equation model that decomposes the variance in a variable into three components: 1) a genetic component (i.e., A); 2) a shared environmental component (i.e., C—environmental influences that cause twins to be more similar to one another); and, 3) a nonshared environmental component (i.e., E—environmental influences that cause twins to differ from one another). The ACE model has been discussed previously (Neale & Maes, 2004; Plomin et al., 2008). All ACE models were estimated using threshold techniques with the structural equation modeling program, *Mx*, a program specifically designed to handle twin data and to estimate behavior genetic models such as the ACE model. Importantly, all ACE models were compared to

“trimmed” models (i.e., where the A component, the C component, or both were fixed to zero and the model re-estimated) to find the most parsimonious solution (i.e., the best-fitting model).

FINDINGS

Along with the prevalence rates, cross-twin correlations (tetrachoric correlations) are presented in Table 1 for all twins, MZ twins only, and DZ twins only. For the first IPV indicator, Hitting Partner, the cross-twin correlations revealed that MZ twins ($r_{MZ} = .21, p = .14$) were more similar as compared to DZ twins ($r_{DZ} = .09, p = .42$) but neither of the correlations reached statistical significance. As can be seen, the MZ cross-twin correlation was higher ($r_{MZ} = .44, p = .04$) than the DZ cross-twin correlation ($r_{DZ} = .34, p = .02$) for the second IPV indicator, Injuring Partner and both were statistically significant. The same pattern of findings emerged for the last IPV indicator, Forcing Sexual Activity. Specifically, the MZ cross-twin correlation was $r_{MZ} = .62 (p = .0001)$ and the DZ cross-twin correlation was $r_{DZ} = -.02 (p = 1.00)$. Taken together, these results suggest that genetic factors may underlie the etiology of all three behaviors because, in all three instances, the MZ cross-twin correlation exceeded the DZ cross-twin correlation.

Insert Table 1 about Here

The second step of the analysis was to estimate logistic regression models for each of the three IPV indicators. Presented in Figure 1 are the results for the Hitting Partner variable. As shown, the predicted probability that twin 1 reported hitting his/her partner increased as a function of their co-twin's status on that same variable. The *rate* of increase, however, appeared to differ across zygosity level; MZ twins' predicted probability of hitting their partner as a function of their co-twin's status increased more than did DZ twins' predicted probability. The odds ratio was 2.25 ($b = .81, \text{standard error} = .82, 95\% \text{ confidence interval for } b = -.79-2.42, z =$

.99, $p = .32$) for MZ twins as compared to an odds ratio of 1.17 ($b = .16$, standard error = .51, 95% confidence interval for $b = -.84-1.16$, $z = .31$, $p = .75$) for DZ twins. In short, these findings offer tentative support for the results gleaned from the tetrachoric correlations: genetic factors may play a role in the etiology of hitting an intimate partner. It is important to note, however, that neither of the logistic regression estimates reached statistical significance.

Insert Figure 1 about Here

The results of the logistic regression equations for the Injuring Partner variable are presented in Figure 2. The substantive results are similar to those garnered from the tetrachoric correlation analysis. Specifically, MZ twins appear to be more similar (odds ratio = 7.68, $b = 2.04$, standard error = 1.19, 95% confidence interval for $b = -.29-4.37$, $z = 1.72$, $p = .09$) than DZ twins (odds ratio = 4.20, $b = 1.43$, standard error = .80, 95% confidence interval for $b = -.14-3.01$, $z = 1.79$, $p = .07$), suggesting that genetic factors influence this behavior. Neither odds ratio reached statistical significance, though.

Insert Figure 2 about Here

The final set of results from this step of the analysis is presented in Figure 3. As before, the findings show that the likelihood of forcing sexual activity upon an intimate partner is closely linked with whether the co-twin had forced sexual activity on his/her own intimate partner in the past. Furthermore, MZ twins were more similar in their behavior (odds ratio = 13.21, $b = 2.58$, standard error = .81, 95% confidence interval for $b = 1.00-4.16$, $z = 3.20$, $p = .001$) as compared to DZ twins (odds ratio = .92, $b = -.08$, standard error = .107, 95% confidence interval for $b = -2.17-2.01$, $z = -.07$, $p = .94$). Indeed, the DZ twin odds ratio was not statistically significant.

Insert Figure 3 about Here

The third step to the analysis was to estimate threshold versions of the ACE model for each of the three IPV indicators. The results of these model-fitting analyses are presented in Table 2, with the best-fitting model presented in bold. As displayed in Table 2, the AE model was the best-fitting model across all three indicators of IPV. To be specific, the AE model revealed that approximately 24% of the variance in the Hitting Partner variable was attributable to genetic factors, but the 95% confidence interval for this path included zero. The remaining variance was attributable to nonshared environmental influences ($E = .76$). It is worth noting that a model including only the nonshared environment (i.e., $E = 1.00$, $\Delta\chi^2 = 1.56$, $p = .46$, $\Delta AIC = -2.44$) also provided a suitable fit but the AE model is presented here because it exactly replicated the findings from the full ACE model with one fewer parameter (i.e., one more degree of freedom). The E model had two fewer parameters (as compared to the ACE model) but the AIC decrease was only 2.44 (out of a possible 4 unit difference), making the AE model the slightly better fit.

Genetic factors explained a larger portion of the variance when the Injuring Partner variable was analyzed ($A = .54$). The remaining variance was attributable to the nonshared environment ($E = .46$). As noted in the table, the AE model produced a non-significant $\Delta\chi^2$ when compared with the full ACE model and the ΔAIC was -1.86, both of which indicate the AE model was the better fit. The CE model also produced a non-significant $\Delta\chi^2$ ($= .18$, $p = .67$) and a reduced AIC ($\Delta AIC = -1.82$) as compared to the full ACE model but the AE model was the slightly better fit according to both statistics. For reference, however, the CE parameter values were $C = .37$ (95% CI = .03-.66) and $E = .62$ (95% CI = .34-.97).

A similar pattern of results was gleaned from the analysis of the Forcing Sexual Activity variable ($A = .51$ and $E = .49$).³ It is important to note that the best-fitting model indicated the shared environment (i.e., C) had no impact on variance in the three IPV indicators, though the AE model was only a marginally better fit as compared to the CE model for the hitting partner variable and $C = .18$ for the injuring partner variable in the full ACE model.^{4,5}

Insert Table 2 about Here

DISCUSSION

Though rates of IPV have declined in recent years (Pinker, 2011), these forms of interpersonal violence continue to pose serious health and safety risks. Research has begun to uncover many correlates of IPV, with most studies identifying within-the-home influences such as being raised in a family where abuse is prevalent (Kalmuss, 1984; Stith et al., 2000) or having been abused as a child. These findings have led many scholars to argue that IPV “runs in the family” due to socialization or learning mechanisms (Bandura, 1977; see Hines & Saudino, 2002 for similar arguments about the current state of the evidence). Typically, intergenerational transmission of IPV is explained in terms of learned behaviors such that a child exposed to abusive parents will learn to cope with his/her own anger by lashing out at others. In short, violence in the home is thought to send the message that displaying physical forms of violence is an acceptable strategy for handling altercations with loved ones.

³ Because the MZ cross-twin correlation was more than twice the size of the DZ cross-twin correlation, an ADE model was also estimated. The results indicated that the AE model was the best-fitting model when compared against the ADE model (see Note in Table 2).

⁴ A series of sex limitation ACE models were analyzed for each of the outcome variables in order to determine whether the etiology of IPV differed across sex and whether this impacted the parameter estimates. For all three variables, the AE models presented here were the best-fitting model as compared to a series of solutions that allowed for several parameters to vary across males and females.

⁵ A multivariate Cholesky decomposition model was explored to determine whether the three IPV indicators shared a portion of their genetic and environmental influences. Unfortunately, the model failed to converge on a proper solution and, as a result, we do not report the results from this analysis here.

While these theoretical explanations are intuitive and have seemingly been supported by prior research, some have argued that the causal pathway may be more complex involving both genetic and environmental influences (Hines & Saudino, 2002; Pinto et al., 2010). Indeed, Widom (1989) revealed early on that exposure to violence in childhood was not a perfect predictor of violent behavior later in life, suggesting that a purely “sociological” explanation of IPV cannot completely explain the phenomenon. These types of findings suggest that genetic influences may play a role and may begin to explain why violent behavior, especially IPV, tends to run in families (Hines & Saudino, 2004, 2007; Pinto et al., 2010).

The goal of the current study was to test the hypothesis that genetic factors explain a portion of the variance in three indicators of IPV: hitting one’s partner, injuring one’s partner, and forcing sexual activity on one’s partner. The analyses revealed three important findings. First, genetic factors explained a portion (around 50%) of the variance in each of the three IPV indicators, a finding that conforms to recent evidence and theoretical statements (Hines & Saudino, 2004; Pinto et al., 2010). Recall that intergenerational transmission of IPV is a common and robust finding (Stith et al., 2000). The current results—that genetic factors account for a portion of the variance in IPV—offers a viable explanation of *how* and *why* IPV is more prevalent in some families as opposed to others.

The second finding of note was that the shared environment accounted for no portion of the variance in each of the three IPV indicators. Recall that the shared environment taps environmental influences that make twins more similar to one another. Thus, the shared environment is likely to tap into within-the-home (the rearing home) influences such as parental rearing strategies and exposure to violence and IPV as a child. In other words, the standard explanation of intergenerational transmission effects (i.e., that IPV is learned by observing that

same behavior in one's parents) does not appear to map with the findings presented here, suggesting that much of the previous research into IPV may have been misspecified (Cleveland et al., 2011; Hines & Saudino, 2004).

The third finding deserving attention is that the nonshared environment explained the remainder of the variance in each of the three IPV indicators. This finding falls in line with a large literature which suggests that the nonshared environment, as opposed to the shared environment, explains a sizable portion of the variance in most human phenotypes (Harris, 1998; Turkheimer, 2000). Researchers should prioritize studies that are able to identify the nonshared environmental factors tied to IPV. Recall that nonshared environments make twins differ from one another. This suggests that experiences specific to each twin's relational setting may be the most powerful environmental influences. Economic hardship experienced by intimate partners has long been shown to correlate with IPV (Benson et al., 2003) and may represent a portion of the nonshared environmental effect identified here.

The current findings were not without limitation and it is important that they be considered. First, this analysis was unable to identify *which* genes influence IPV. Researchers have revealed a link between certain polymorphisms (e.g., *MAOA*, *DRD2*, and *DRD4*) and antisocial behaviors (Charney & English, 2012). These genetic markers may play a role in IPV, but future research will be necessary to uncover those relationships. In order to illustrate how such an effect might unfold, consider the functioning of genes in the body (Raine, 2008). One of the primary functions of a gene is to assemble proteins that will form tissues which will eventually comprise complex systems like the central nervous system (i.e., the brain and its constituent parts). This becomes important when one considers that variation in certain regions of the brain can lead to impulsive, imprudent, aggressive and even violent behavior (Raine,

2008). To the extent that certain polymorphisms correspond to differences in either functioning or structure (or both) in various brain regions, one might begin to see how genetic factors could increase the likelihood of behaviors such as IPV, as well as other deleterious outcomes.

Second, the current study did not tap lifetime involvement in IPV. To be specific, respondents were asked to report on their involvement in IPV with their most recent partner. This raises the possibility that certain respondents engaged in IPV with prior partners but were not captured as batterers in the current study. Future work should replicate the current findings while taking into account lifetime involvement in IPV. Third, some of the data cells had limited responses and, as a result, may have impacted the stability of the estimates. It was for this reason that we reported exact *p*-values and 95% confidence intervals in the tables/text whenever possible. Future research would benefit from including an even larger sample than that used here in order to determine whether such limited variance impacted the current estimates. Given that these estimates fall within the error range of those reported by Hines and Saudino (2004) it may be safe to conclude, albeit tentatively, that the limited variance did not negatively impact the findings in any systematic way, though it would contribute to larger standard errors and wider 95% confidence intervals.

Taken together, the current findings indicate that much of the intergenerational transmission of IPV may be due to the transmission of genetic risk factors as opposed to the transmission of certain values or because of the learning of these behaviors. This is not to say that IPV is completely under genetic control and that the environment holds *no* sway over these behaviors. Indeed, the findings revealed that the nonshared environment explained a large portion of the variance in each of the three indicators and it has long been known that gene-environment interactions (GxE) as well as epigenetic influences allow for the environment to

affect the genome, which in turn affects behavior but may manifest as a genetic influence in ACE models (Purcell, 2002). Recent research published by The ENCODE Project Consortium (e.g., Encode, 2012) revealed the impact of “junk DNA” on epigenetic processes. Epigenetic research has underscored the intricate entanglement of environmental stimuli with the genome (Berger et al., 2009) showing how environmental factors affect the genetic “switches” which turn genes “on or off.” To the extent that exposure to IPV in the home environment flips these relevant switches through processes such as stress response (Franklin et al., 2010), the identification of genetic versus environmental causes of behavioral variation becomes more difficult to disentangle. In light of these complicated issues, we feel it is too hasty to conclude that our findings disprove research which explains IPV in terms of early exposure in the home environment, though the current study does suggest that these theories may be augmented by the integration of findings from genetic research. We look forward to the coming wave of IPV research that seeks to address these important issues.

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Table 1. Prevalence of Domestic Violence and Cross-twin Correlations by Zygosity Levels

	All Twins	MZ Twins	DZ Twins
<i>Hitting Partner</i>			
Prevalence	10.20%	8.62%	11.22%
Tetrachoric Correlation	.13	.21	.09
<i>Injuring Partner</i>			
Prevalence	3.79%	3.66%	3.88%
Tetrachoric Correlation	.38*	.44*	.34*
<i>Forcing Sexual Activity</i>			
Prevalence	5.24%	4.97%	5.41%
Tetrachoric Correlation	.28*	.62*	-.02

* $p < .05$

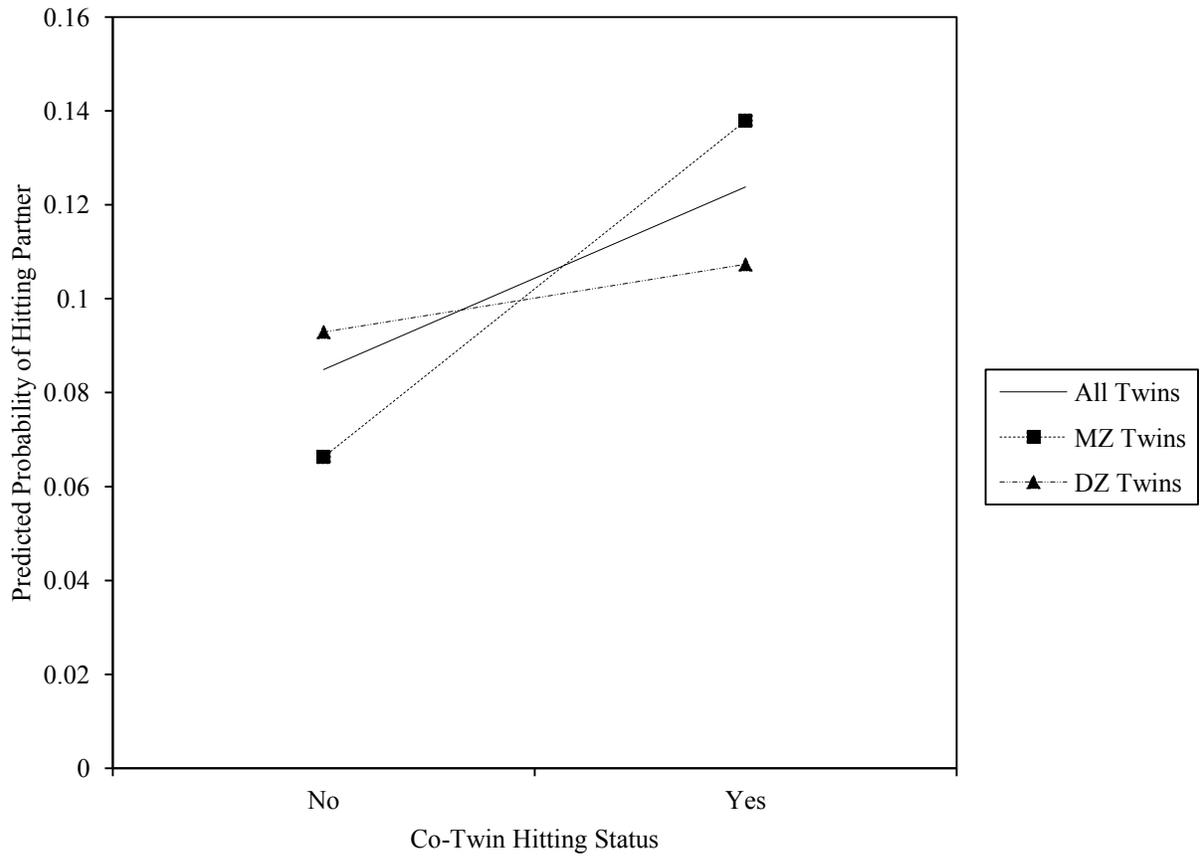
Table 2. ACE Model Parameter Estimates and Fit Statistics

	<u>Parameter Estimates</u>			χ^2	<u>Model Fit Statistics</u>		
	A	C	E		$\Delta\chi^2$	AIC	Δ AIC
<i>Hitting Partner</i>							
ACE	.24	.00	.76	780.03	--	-1585.97	--
95% CI	(.00-.57)	(.00-.36)	(.43-1.00)				
AE	.24	.00	.76	780.03	.00	-1587.97	-2.00
95% CI	(.00-.57)	(.00-.00)	(.43-1.00)				
<i>Injuring Partner</i>							
ACE	.30	.18	.52	378.08	--	-1987.92	--
95% CI	(.00-.85)	(.00-.65)	(.15-.96)				
AE	.54	.00	.46	378.22	.14	-1989.78	-1.86
95% CI	(.04-.85)	(.00-.00)	(.15-.96)				
<i>Forcing Sexual Activity</i>							
ACE	.51	.00	.49	480.47	--	-1881.53	--
95% CI	(.00-.81)	(.00-.43)	(.19-.90)				
AE^a	.51	.00	.49	480.47	.00	-1883.53	-2.00
95% CI	(.10-.81)	(.00-.00)	(.19-.90)				

* $p < .05$

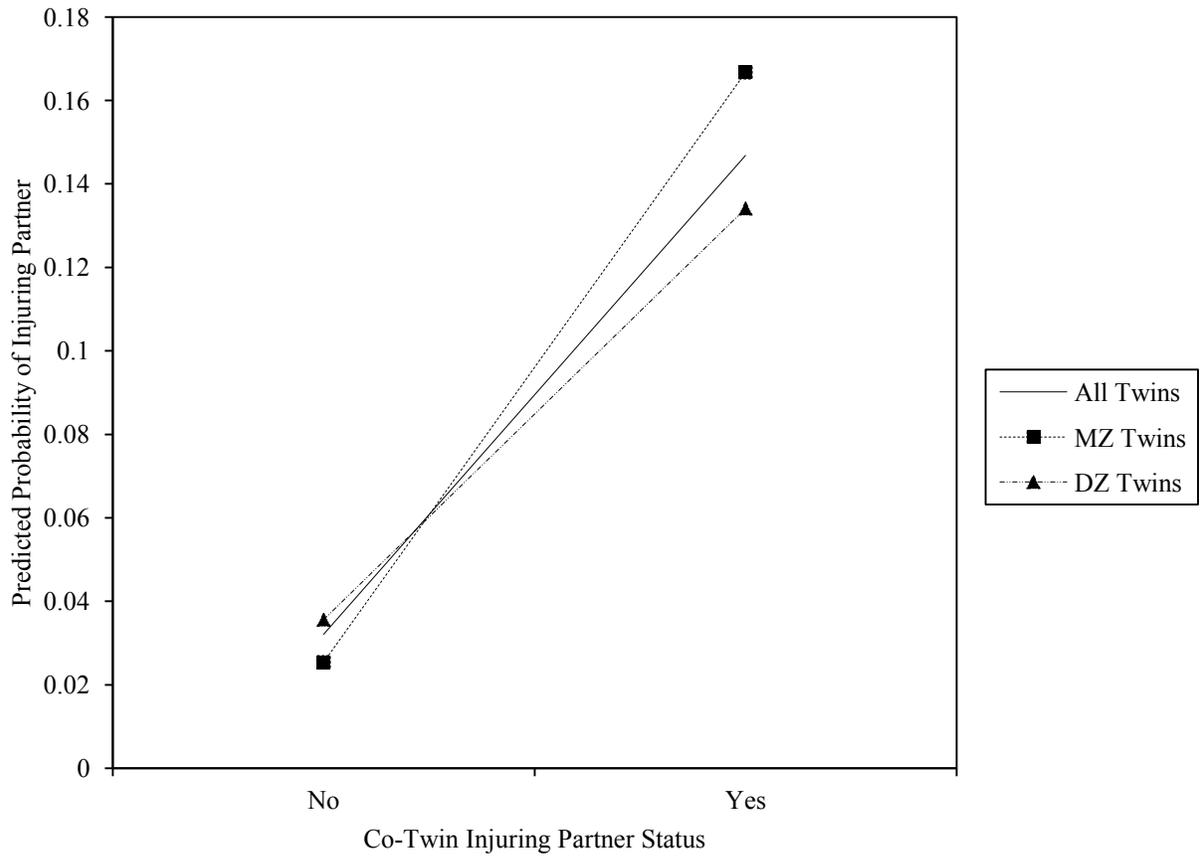
Note: Best-fitting model in bold; CI = confidence interval; a. An ADE model was analyzed and the parameter estimates were A = .00, D = .60, E = .40, $\Delta\chi^2$ between ADE and AE model was 1.42, $p > .05$ suggesting that the AE model was the best fit to the data.

Figure 1. Predicted Probability of Hitting Partner as a Function of Co-Twin’s Hitting



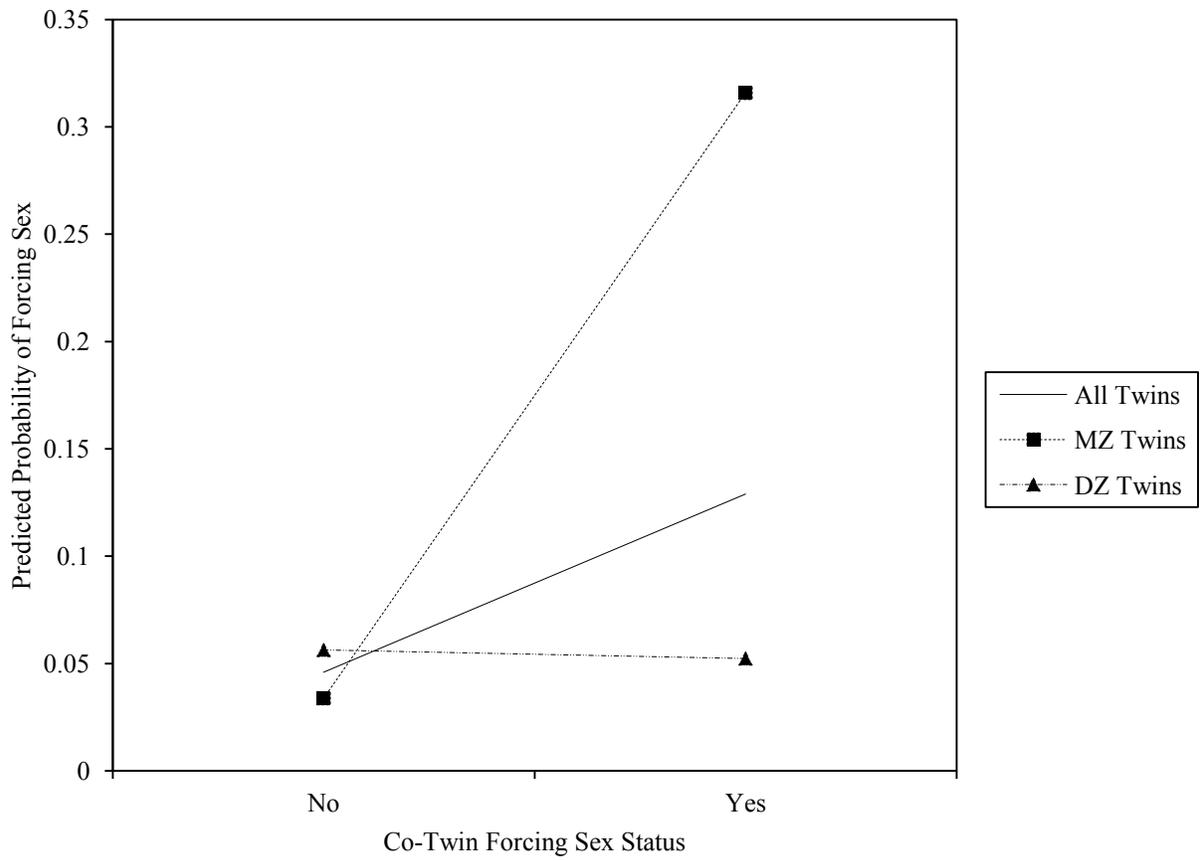
Note: Equations for All Twins and for DZ Twins included a control for gender.

Figure 2. Predicted Probability of Injuring Partner as a Function of Co-Twin’s Injuring



Note: Equations for All Twins and for DZ Twins included a control for gender.

Figure 3. Predicted Probability of Forcing Sexual Activity on Partner as a Function of Co-Twin's Forcing Sexual Activity



Note: Equations for All Twins and for DZ Twins included a control for gender.