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Associations between attachment anxiety and intimate partner violence perpetration and victimization: Consideration of genetic covariation

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ABSTRACT

Adult attachment anxiety shows consistent and robust associations with intimate partner violence (IPV) perpetration and IPV victimization. Prior research, however, has not investigated whether this association would be robust to genetic covariation between the traits of interest. We expand the limited research regarding genetic influences on IPV by examining the extent to which accounting for potential genetic covariation between IPV and attachment anxiety can inform our understanding of both perpetration and victimization. Study 1 analyzed self-report data (n = 277), which included measures of romantic attachment and IPV perpetration and victimization. Study 2 employed simulation-based modeling procedures to estimate the extent to which genetic covariation can explain observed phenotypic associations between attachment anxiety and IPV. Study 1 demonstrated significant positive associations (βs = 0.20–0.30) between attachment anxiety (but not avoidance) and IPV perpetration and victimization. Models from Study 2 showed that genetic covariation has the potential to explain approximately 25% of the phenotypic association between attachment and IPV. Findings suggest that attachment anxiety is a robust predictor of both IPV perpetration and victimization. Future IPV research should consider both genetic and environmental mediation of the association between attachment anxiety and IPV outcomes.

1. Introduction

Psychological and social science research on intimate partner violence (IPV; psychological, physical, or sexual aggression perpetrated toward a current intimate partner) is diverse, spanning numerous theoretical perspectives. Two broad camps of research have emerged in the literature: sociological (Ali & Naylor, 2013a) and psychological (Ali & Naylor, 2013b). Sociological perspectives on IPV focus on socialization experiences over the lifespan as risk factors for involvement in violent relationships as an adult (Ali & Naylor, 2013a; Renner & Whitney, 2012; Walby & Allen, 2004), such as a history of domestic abuse (either as a recipient or witness). Psychological perspectives regarding IPV focus on individual difference risk factors (e.g., personality traits) for involvement in violent relationships either as a perpetrator or victim (Ali & Naylor, 2013b). Within the domain of IPV research, adult attachment theory (Fraley & Shaver, 2000; Hazan & Shaver, 1987) is a widely applied *psychological* theory used to investigate whether individual differences in attachment patterns predict IPV outcomes.

Adult attachment theory addresses how individual variation in attachment orientations influence (and are influenced by) affective, cognitive, and behavioral responses in romantic relationships (Fraley & Shaver, 2000; Hazan & Shaver, 1987). Attachment orientations toward romantic partners are conceptualized along the two dimensions of *anxiety* and *avoidance* (Brennan, Clark, & Shaver, 1998; Fraley, Waller, & Brennan, 2000). Attachment anxiety reflects hyperactivation of the romantic attachment system, characterized by attempts to maintain proximity to a partner, and an overdependence on a partner for stability and security (Cassidy, 2000). More anxiously attached individuals are hypervigilant to cues of rejection by a partner (Rholes & Simpson, 2004), and have difficulty disengaging from cues indicative of relationship distress (Mikulincer, Gillath, & Shaver, 2002). More anxiously attached individuals may deploy controlling or coercive behaviors in response to cues of rejection to elicit support and investment from a partner (Mikulincer & Shaver, 2007). Attachment avoidance, in

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contrast, reflects hypoactivation of the romantic attachment system, characterized by attempts to evade emotional intimacy with and physical proximity to a partner (Cassidy, 2000). More avoidantly attached individuals emphasize independence and self-reliance in relationships to facilitate decreased partner dependence and proximity-seeking behaviors (Edelstein & Shaver, 2004). More avoidantly attached individuals are also more likely to discount information and cues to relationship threats (Dykas & Cassidy, 2011; Kruger et al., 2013).

A corpus of empirical work has addressed the phenotypic associations between individual differences in attachment orientations and IPV outcomes. Adult attachment orientations have significant associations with IPV perpetration (Babcock, Jacobson, Gottman, & Yerington, 2000; Barbaro & Shackelford, 2019; Bookwala & Zdaniuk, 1998; Dutton, Saunders, Starzomski, & Bartholomew, 1994a, 1994b; Fournier, Brassard, & Shaver, 2011; Gormley & Lopez, 2010; Henderson, Bartholomew, Trinke, & Kwong, 2005; Mauricio, Tein, & Lopez, 2007; Miga, Hare, Allen, & Manning, 2010; Orcutt, Garcia, & Pickett, 2005; Sommer, Babcock, & Sharp, 2017) and IPV victimization (Doumas, Pearson, Elgin, & McKinley, 2008; Henderson et al., 2005; Kuijpers, van der Knaap, & Winkel, 2012; Sandberg, Valdez, Engle, & Menghrajani, 2019; Sommer et al., 2017) for both men and women. A review of this literature indicates that attachment anxiety, but not attachment avoidance, is a consistent and robust predictor of IPV perpetration and victimization: Whereas all but one of the above-cited studies (Kuijpers et al., 2012) on the phenotypic association between attachment anxiety and IPV report significant associations, only a handful document associations between attachment avoidance and IPV (Barbaro & Shackelford, 2019; Gormley & Lopez, 2010; Mauricio et al., 2007; Miga et al., 2010; Sommer et al., 2017).

Several characteristics of anxious attachment, in particular, are associated with IPV outcomes. Mayseless (1991) argued, for example, that IPV is motivated by fear of abandonment, and partner-directed violence is one strategy an anxiously attached individual may deploy to preserve the relationship (Gormley, 2005). For men in particular, IPV can also function to guard sexual access to a romantic partner (Barbaro & Shackelford, 2016; Thornhill & Palmer, 2000). Attachment anxiety is also associated with affect escalation, impulsivity, anger and resentment, displaced aggression, and low self-control (Alexander & Anderson, 1994; Gormley, 2005; Mikulincer, 1998), and such individuals have greater difficulty regulating emotional and behavioral responses produced by fear of rejection and abandonment (Dutton, 2007). These characteristics may account, in part, for the partner-directed controlling, coercive, and violent behavior displayed by more anxiously attached individuals (Barbaro, Pham, Shackelford, & Zeigler-Hill, 2016; Barbaro, Sela, Atari, Shackelford, & Zeigler-Hill, 2019; Mikulincer & Shaver, 2007). More avoidantly attached individuals, in contrast, report less chronic jealousy (Sharpsteen & Kirkpatrick, 1997) and are more attentive to potential alternative romantic partners for themselves (DeWall et al., 2011).

Several mechanisms have been proposed to explain why attachment anxiety predicts IPV outcomes. Three areas of psychological research have focused on the theoretical mechanisms underlying the association between attachment anxiety and IPV outcomes: Developmental, social-personality, and evolutionary. Developmental and social-personality perspectives have theoretical commonalities with regard to how adult attachment develops across the life course, and propose that insecure attachments to primary caregivers in infancy inform the development of insecure internal working models that influence later adult attachment relationships (Ali & Naylor, 2013b; Fraley & Shaver, 2000). A characteristic of insecure attachment is argued to be poor conflict resolution strategies, which may include manipulative and violent behavior (Gormley, 2005). Evolutionary perspectives propose that romantic attachment bonds, especially attachment anxiety, regulate and monitor threats (actual or imagined) to a romantic relationship (Barbaro et al., 2019; Barbaro, Boutwell, Barnes, & Shackelford, 2017a), such as cues to partner infidelity or decreased commitment. A response to such threats may be greater jealousy (Buss, 2000), which cross-culturally is a leading predictor of IPV (Daly & Wilson, 1988; Dutton, 2007). Despite differing perspectives with regard to the theoretical underpinnings of this association, this literature supports a general consensus that insecure attachment – especially attachment anxiety – is a psychological risk factor for IPV outcomes.

The theoretical explanations underpinning why attachment anxiety, specifically, is associated with IPV outcomes, however, is irrelevant to the question of whether the association is robust and reliable. There are two strategies researchers can employ to discern the robustness of an association. One strategy is to employ experimental designs capable of discerning whether one variable causes a specified outcome. In this case, whether attachment anxiety causes IPV perpetration and/or victimization. Experimental designs are not always permissible due to ethical concerns, as is true for the current effect under investigation. To circumvent such ethical obstacles, an alternative strategy is to infer an effect as true to the extent that the effect can withstand the inclusion of relevant confounding variables. Even so, statistical models can only account for so many (potentially confounding) variables simultaneously before encountering collinearity issues or variance constraint problems, assuming a single research study can secure data on all relevant confounders. This is not meant to discourage research assessing the robustness of the association between attachment anxiety and IPV outcomes, but instead to encourage careful consideration of what confounders need to be included.

The present research aims to reassess the robustness of the association between attachment anxiety and IPV outcomes against a novel potential confounder that has thus far been neglected in this literature: genetic influences known to operate on the traits of interest. There is limited work that has attempted to quantify the possibility that genetic variation might impact IPV outcomes (Barnes, TenEyck, Boutwell, & Beaver, 2013; Hines & Saudino, 2004; Schwab-Reese, Parker, & Peek-Asa, 2017; Stuart et al., 2014) or the documented psychological predictors of IPV perpetration and victimization (Barnes & Beaver, 2012). The aim of the current research, therefore, is to expand this limited corpus of behavioral genetic work (Barnes et al., 2013; Hines & Saudino, 2004) by first examining the association of key psychological predictors – specifically, attachment dimensions – and IPV perpetration and victimization using the traditional methods. We then demonstrate the extent to which our results may be impacted by previously unmeasured genetic factors underlying the traits of interest.

Understanding how genetic influences affect our understanding of observed phenotypic associations is important for several reasons. Nearly all complex psychological and behavioral traits demonstrate non-zero heritability, which is to say that nearly every trait investigated by psychologists and other social scientists is influenced, to some extent, by genetic variation (Plomin, DeFries, Knopik, & Neiderhiser, 2016; Polderman et al., 2015; Turkheimer, 2000). Significant heritability estimates have been documented for physical and psychological IPV perpetration (Barnes et al., 2013) and victimization (Hines & Saudino, 2004). Hines and Saudino (2004), for example, investigated genetic influences on the victim-offender overlap – the observation that perpetrators of aggressive behavior are disproportionately likely to also be victims of aggressive behavior – for IPV (also referred to as "bidirectional" IPV; Langhinrichsen-Rohling, Misra, Selwyn, & Rohling, 2012). Using a sample of 134 monozygotic twins and 41 dizygotic twins, Hines and Saudino produced heritability estimates (h^2) for IPV perpetration (h^2 = 0.16–0.22) and victimization (h^2 = 0.15–0.25). Barnes et al. (2013) provided similar heritability estimates for IPV perpetration (h^2 = 0.24–0.54) using a larger sample of 462 monozygotic twins and 721 dizygotic twins.

Quantitative genetic analyses of adult (i.e., 18+ years of age) attachment patterns also have been conducted. Crawford et al. (2007; 239 twin pairs) and Donnellan et al. (2008; 273 twin pairs) produced heritability estimates for continuous measures of attachment anxiety of h^2 = 0.40 and 0.45, respectively. Brussoni, Jang, Livesley, and Macbeth

(2000), however, used a categorical model of attachment, producing $h^2 = 0.25$ and 0.43 for the preoccupied and fearful categories (both of which are characterized by high anxiety), respectively. Although categorical models have been criticized in favor of continuous models for research use (Fraley, Hudson, Heffernan, & Segal, 2015), the categorical heritability estimates are included in the current research because of the dearth of available behavioral genetic studies on attachment. In contrast to the significant heritability estimates produced for attachment anxiety, genetic influences on attachment avoidance are less robust, with significant estimates reported only by Donnellan et al.

The current research focuses on the traits of attachment anxiety and IPV, specifically, for two reasons. First, attachment anxiety, but not attachment avoidance, is a consistent and robust predictor of IPV perpetration and victimization (see above). Second, attachment anxiety, but not avoidance, show consistent heritability estimates in the available research, and investigation of genetic covariation impacting phenotypic associations requires that both traits of interest (e.g., *X* and *Y*) are heritable. Given that genetic variation plays a non-negligible role for perpetration and victimization of IPV and attachment anxiety, ignoring potential genetic covariation of such associations could hinder a comprehensive understanding of IPV, such that common genetic influences underpinning both traits could explain a portion of the associations between attachment anxiety and IPV outcomes. The current research includes two studies aimed at assessing the robustness of the association between attachment anxiety and IPV outcomes in light of potential genetic covariation underpinning both traits.

2. Study 1

Study 1 uses survey methodology to test the hypothesis that attachment anxiety (but not attachment avoidance) will be associated with IPV perpetration and victimization across the domains of psychological, physical, and sexual violence—replicating previous work. This study was approved by the Institutional Review Board at the university at which the data were collected.

2.1. Method

2.1.1. Participants

We secured data from 277 participants (59% men) in a committed, heterosexual, romantic relationship via MTurk. Participants' mean age was 32.7 years ($SD = 15.7$), and the mean relationship length was 56.8 months ($SD = 70.9$). The racial/ethnic makeup of the same was 44% White, 38% Asian, 10% American Indian or Alaska Native, 7% Black or African American, 1% Native Hawaiian or Other Pacific Islander, and 27% Hispanic or Latino.

MTurk is a crowdsourcing website used by psychological researchers. Researchers can collect survey data in exchange for monetary compensation. Participants recruited via MTurk are more socioeconomically and ethnically diverse than traditional Internet or college samples (Casler, Bickel, & Hackett, 2013). Data collected via MTurk have been shown to be of equal quality to data collected by other Internet methods and by college undergraduates in person (Buhrmester, Kwang, & Gosling, 2011; Casler et al., 2013).

2.1.2. Procedure

Prospective participants viewed an advertisement for the study on MTurk's job listings. Those interested in and eligible to participate (i.e., at least 18 years of age, currently in a committed, heterosexual relationship) were provided a link to an informed consent statement about the study. Those who agreed to participate could access and complete the survey, and those who did not agree to participate were exited from the study. We implemented recommended MTurk filters (Peer, Vosgerau, & Acquisti, 2013) such that individuals could only participate if they had successfully completed 95% of at least 500 accessed MTurk jobs. Participants were compensated $0.50 for completing the study. All study procedures were university-approved prior to survey initiation.

2.1.3. Materials

To assess romantic attachment, participants completed the Experiences in Close Relationships Scale-Revised (ECR-R; Fraley et al., 2000), a 36-item measure assessing attachment bonds along the dimensions of anxiety and avoidance. Participants were instructed to respond to statements as they relate to their current romantic partner on a 7-point scale ranging from 1 (*strongly disagree*) to 7 (*strongly agree*). Statements in the ECR-R were modified to be partner-specific, rather than partner-general. For example, the statement, “When I show my feelings for romantic partners, I'm afraid they will not feel the same about me” was modified to, “When I show my feelings for my romantic partner, I'm afraid my partner will not feel the same about me”. Composite scores were calculated for each participant by averaging their responses to the 18 anxiety items ($\alpha = 0.95$) and the 18 avoidance items ($\alpha = 0.92$).

Frequency of IPV perpetration was measured with the Revised Conflict Tactics Scale (CTS2; Straus, Hamby, Boney-McCoy, & Sugarman, 1996). The CTS2 includes 38 statements regarding perpetration of IPV. Participants were instructed to read each statement, which describes an aggressive act, and then indicate the number of times they perpetrated this act against their partner in the past year (frequency of perpetration) and the number of times their partner perpetrated this act against them in the past year (frequency of victimization) on the following 8-point scale: 1 (*this has never happened*), 2 (*once in the 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*), 7 (*20+ times in the past year*), and 8 (*not in the past year, but it has happened before.*)

The CTS2 contains five subscales: *psychological aggression*, *physical assault*, *sexual coercion*, *negotiation*, and *sustained injury*. The focus of the current research is on perpetration and victimization of IPV and, therefore, subsequent analyses focus on the three perpetration and victimization domains—psychological (e.g., “Shouted or yelled at my partner”), physical (e.g., “Slammed my partner against a wall”), and sexual (e.g., “Used [physical] force to make my partner have sex”). Following Straus et al. (1996), responses to each statement were recoded as the midpoint of the response category the participant reported. For example, if the participant reported an act occurring “3–5 times in the past year,” the response was recoded as occurring 4 times in the past year. The response category of “20+ times in the past year” was recoded as the act occurring 20 times the past year to avoid inflation of perpetration and victimization rates. If participants reported for an act that “this has never happened” (1) or that the act “has not occurred in the past year, but has happened before,” the response was recoded as “0.”

Sum scores for perpetration and victimization over the previous year across each violence domain—psychological aggression, physical assault, and sexual coercion—were calculated by adding the recoded response category midpoints. Higher composite scores in each domain indicate greater frequency of IPV perpetration and victimization over the previous year. We then calculated monthly rates of IPV perpetration and victimization for use as the outcome variable in correlational and regression analyses. Summed scores of IPV perpetration (psychological, physical, and sexual) and victimization (psychological, physical, and sexual) were divided by 12 (months in a year) to obtain average monthly rate estimates for each domain of IPV. For participants reporting relationship length of fewer than 12 months, their summed scores for IPV perpetration and victimization domains were divided by the participants' reported relationship length (measured in months). This was done because not all participants were in a relationship for at least one year. Frequency scores are therefore confounded by relationship length, given that individuals that have been in relationships for longer periods of time have more opportunity for IPV exposure.

Table 1
Descriptive statistics and correlations between attachment dimensions and intimate partner violence perpetration and victimization.*

	Bivariate correlations							
	1	2	3	4	5	6	7	8
1. Anxiety	–							
2. Avoidance	0.68***	–						
3. Psychological perpetration	0.27***	0.27***	–					
4. Physical perpetration	0.32***	0.32***	0.92***	–				
5. Sexual perpetration	0.26***	0.26***	0.94***	0.96***	–			
6. Psychological victimization	0.35***	0.27***	0.89***	0.95***	0.93***	–		
7. Physical victimization	0.31***	0.26***	0.92***	0.97***	0.95***	0.90***	–	
8. Sexual victimization	0.27***	0.21***	0.91***	0.96***	0.95***	0.96***	0.90***	–
Mean	3.52	2.99	3.54	4.78	3.19	3.38	4.82	2.92
SD	1.43	1.05	8.29	11.25	7.93	6.83	11.31	7.51

	Partial correlations						
	1	2	3	4	5	6	7
Controlling for avoidance							
1. Anxiety	–						
2. Psychological perpetration	0.16**	–					
3. Physical perpetration	0.21**	0.91***	–				
4. Sexual perpetration	0.16**	0.94***	0.95***	–			
5. Psychological victimization	0.23***	0.88***	0.94***	0.93***	–		
6. Physical victimization	0.19**	0.91***	0.97***	0.95***	0.89***	–	
7. Sexual victimization	0.17**	0.90***	0.96***	0.95***	0.96***	0.90***	–
Controlling for anxiety	1	2	3	4	5	6	7
1. Avoidance	–						
2. Psychological perpetration	0.07	–					
3. Physical perpetration	0.05	0.91***	–				
4. Sexual perpetration	0.05	0.94***	0.95***	–			
5. Psychological victimization	0.05	0.88***	0.94***	0.93***	–		
6. Physical victimization	0.07	0.91***	0.97***	0.95***	0.90***	–	
7. Sexual victimization	0.05	0.90***	0.95***	0.95***	0.96***	0.90***	–

Notes. Psychological, Physical, and Sexual Perpetration and Victimization reflect average monthly rates.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

2.2. Results

Descriptive statistics for the study variables are displayed in Table 1. Bivariate correlations revealed positive associations between all study variables, such that attachment anxiety and attachment avoidance were positively associated with all domains of IPV perpetration and victimization (Table 1). Partial correlations revealed significant associations of only attachment anxiety with IPV perpetration and victimization, such that all associations between IPV and attachment avoidance become non-significant once the variance of attachment anxiety was partitioned out. Domains of IPV perpetration and victimization were strongly associated (all bivariate and partial *r*s ≥ 0.88), consistent with the victim-offender overlap hypothesis.

Hierarchical multiple regression analyses were conducted to examine the unique predictive utility of each attachment dimension and to investigate whether participant sex influenced monthly rates of IPV perpetration and victimization (see Table 2). We also explored interaction effects between attachment dimensions and participant sex. No significant interactions emerged for IPV perpetration or victimization across any domain (interaction results are available in Electronic Supplementary Material [ESM] Table 1). Attachment anxiety was a positive predictor of all IPV outcomes, such that greater attachment anxiety predicted higher average monthly rates of IPV perpetration and victimization. There were no significant effects of attachment avoidance on any IPV outcome. Participant sex was a significant predictor for only physical victimization, such that being male predicted higher average rates of physical victimization.

We also conducted hierarchical regression (ESM Table 2) and negative binomial regression (ESM Table 3) analyses with IPV count frequencies as the outcome variable. The results are substantively similar to the IPV monthly rate outcomes, with the exception that sexual IPV perpetration was negatively predicted by attachment avoidance in the negative binomial regression model (ESM Table 3). Overall, the results of the alternative regression models similar results between attachment anxiety and IPV outcomes.

3. Study 2

The results of Study 1 support the hypothesis that attachment anxiety is positively associated with both IPV perpetration and victimization, consistent with previous research. The results also accord with the victim-offender overlap hypothesis (Barnes & Beaver, 2012; Hines & Saudino, 2004) in that IPV perpetration and victimization were strongly and positively correlated. Study 2 addresses how the inclusion of a thus far unaccounted for potential confounder – shared genetic variation – could affect the association between attachment anxiety and IPV perpetration and victimization.

The primary aim of Study 2 was to investigate the implications of shared genetic covariation for understanding phenotypic associations between attachment anxiety and IP. Stated differently, Study 2 sought to examine the degree to which the observed phenotypic correlations between attachment anxiety and IPV perpetration and victimization may be explained by the degree to which attachment anxiety correlates genetically with both IPV perpetration and victimization. We employ simulation-based modeling (Barbaro, Boutwell, Barnes, & Shackelford, 2017b; Barnes, Barbaro, Boutwell, & Shackeford, 2017; Barnes,

Table 2
Regression analyses predicting monthly rates of intimate partner violence perpetration and victimization.

	Perpetration								
	Psychological (F (3,273) = 8.90***, R^2 = 0.09)			Physical (F (3,273) = 11.71***, R^2 = 0.11)			Sexual (F (3,273) = 7.93***, R^2 = 0.08)		
	B (SE)	β	t	B (SE)	β	t	B (SE)	β	t
Sex	1.75 (1.50)	0.10	1.75	2.43 (1.33)	0.11	1.81	1.76 (0.96)	0.11	1.83
Anxiety	1.21 (0.46)	0.21	2.65**	2.09 (0.61)	0.27	3.42**	1.13 (0.44)	0.20	2.57*
Avoidance	0.52 (0.63)	0.07	0.83	0.50 (0.84)	0.05	0.60	0.37 (0.60)	0.05	0.61
	Victimization								
	Psychological (F (3,273) = 13.50***, R^2 = 0.13)			Physical (F (3,273) = 11.82***, R^2 = 0.12)			Sexual (F (3,273) = 8.25***, R^2 = 0.07)		
	B (SE)	β	t	B (SE)	β	t	B (SE)	β	t
Sex	1.32 (0.80)	0.10	1.64	2.66 (1.34)	0.12	1.98*	1.70 (0.91)	0.11	1.87
Anxiety	1.42 (0.37)	0.30	3.87***	1.93 (0.61)	0.24	3.14**	1.12 (0.42)	0.21	2.67**
Avoidance	0.27 (0.50)	0.04	0.53	0.74 (0.84)	0.07	0.88	0.30 (0.57)	0.04	0.53

Notes. Only main effects shown. No significant results of any two-way or three-way interactions (interaction results available in ESM Table 1). Sex (0 = female; 1 = male). B = unstandardized coefficient; β = standardized coefficient. VIF < 2 for attachment anxiety and avoidance. N = 277.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

Boutwell, Beaver, Gibson, & Wright, 2014) using estimates obtained from the published literature (see Table 3) as input parameters to examine the impact of potential genetic covariation between attachment anxiety and IPV on understanding of the phenotypic associations.

3.1. Analysis plan

The simulation tool has been utilized previously in Barbaro et al. (2017b, for formal mathematical details of the simulation tool, see Barnes et al., 2017), and is underpinned by the logic of Bayesian analysis. The goal of the simulation approach is to estimate the degree to which shared genetic variation, r_g (i.e., genetic correlation), between two traits of interest, X and Y (note that the simulation is agnostic regarding the causal relation between X and Y), accounts for the observed phenotypic correlation, r_p, between the two traits, represented as h^2_{cov}. The simulation tool is intended to be used when genetically-sensitive data (e.g., twin or sibling data) are not available, allowing researchers to estimate the degree to which genetic factors might impact phenotypic observations. In the current study, we estimated h^2_{cov} for the association between IPV perpetration and attachment anxiety (simulation A) and IPV victimization and attachment anxiety (simulation B).

Four input parameters must be specified to estimate h^2_{cov} for each variable pair of interest: (1) the heritability estimate of X, represented as h^2_X; (2) the heritability estimate of Y, represented as h^2_Y; (3) the phenotypic correlation, r_p, of the variable pair; and (4) the genetic correlation, r_g, of the variable pair. The four input parameters are then entered into the equation below to produce an estimate of h^2_{cov}.

$$h^2_{cov} = \frac{\sqrt{h^2_X} * r_g * \sqrt{h^2_Y}}{r_p}$$

Heritability estimates for almost any trait of interest can be obtained from consulting the behavioral genetic literature (Plomin, DeFries, Knopik, & Neiderhiser, 2013; Polderman et al., 2015). A range of values for the phenotypic correlation between the target traits also can be obtained from the literature. Values for the genetic correlation between the target traits may be more difficult to identify, given that estimation of a genetic correlation between two traits requires large-sample genetic analyses. In line with recommendations of Barnes et al. (2017), a range of plausible but conservative values can be used to estimate h^2_{cov}.

Given that there is not one "true" heritability estimate for any specific trait, nor are observed correlations fixed at one "true" value (estimates can fluctuate across populations and ecological conditions, for example), it is recommended that researchers draw on a range of values for each input parameter based on the literature. Given the estimates for each parameter in the literature, one can then produce independent beta distributions for each parameter (see Fig. 1). The simulation model samples from the specified beta distribution for each parameter to solve the h^2_{cov} equation, and for this process is repeated k times for each simulation, where k is set to the recommended 10,000.

The development of the beta distributions for the current study was informed by prior research on the heritability of IPV perpetration and victimization, and attachment anxiety. To our knowledge, only two studies report heritability estimates for IPV perpetration, and the beta distribution for IPV perpetration was centered around 0.33 for the analyses (see Table 3; Barnes et al., 2013; Hines & Saudino, 2004). Only Hines and Saudino (2004) report heritability estimates for IPV victimization, and the beta distribution was centered around 0.20 (see Table 3). Three studies reported heritability estimates for attachment anxiety in adult samples, and the beta distribution for attachment anxiety was centered around 0.38 (see Table 3; Brussoni et al., 2000; Crawford et al., 2007; Donnellan et al., 2008). Distributions for the phenotypic correlations between each variable pair were based on results from Study 1 and studies in the literature reporting correlations between the variables of interest (see Table 3). For IPV perpetration and attachment anxiety r_p was centered around 0.21, and for IPV victimization and attachment anxiety r_p was centered around 0.24 (see Fig. 1).

Given that genetic correlations between variables are rarely reported in the literature, we follow the recommendations of Barnes et al. (2017) to examine a reasonable range of values for r_g for each h^2_{cov} simulation. To our knowledge, no estimates of r_g have been published for the variable pairs of IPV and attachment anxiety (simulation A) and IPV victimization and attachment anxiety (simulation B). We therefore chose a range of values for r_g that were plausible, given that genetic structure often accords closely with phenotypic structure (i.e., $r_g \approx r_p$) (Plomin et al., 2016), but conservative (by most standards, a small to moderate correlation). For simulations A and B, we begin with r_g values indicating no genetic correlation between the two variables (i.e., r_g = 0.0) and increase in increments of 0.01 up to 0.25.

Table 3
Input parameters for simulation models.

Intimate partner violence perpetration		
h^2	Measure	Source
0.16	CTS2—Physical	Hines and Saudino (2004)
0.24	Add Health—Physical (hitting)	Barnes et al. (2013)
0.54	Add Health—Physical (injuring)	Barnes et al. (2013)
0.22	CTS2—Psychological	Hines and Saudino (2004)
0.51	Add Health—Sexual	Barnes et al. (2013)

Intimate partner violence victimization		
h^2	Measure	Source
0.15	CTS2—Physical	Hines and Saudino (2004)
0.25	CTS2—Psychological	Hines and Saudino (2004)

Attachment anxiety		
h^2	Measure	Source
0.45	Adult Attachment Scale (AAS)	Donnellan et al. (2008)
0.40	Relationship Scales Questionnaire	Crawford et al. (2007)
0.25	Relationship Scales Questionnaire—Preoccupied	Brussoni et al. (2000)
0.43	Relationship Scales Questionnaire—Fearful	Brussoni et al. (2000)

Intimate partner violence perpetration and attachment anxiety		
r_p	Measure	Source
0.21	CTS2—Physical; ECR-R	Study 1 (partial *r*)
0.16	CTS2—Psychological; ECR-R	Study 1 (partial *r*)
0.16	CTS2—Sexual; ECR-R	Study 1 (partial *r*)
0.23	CTS2—Physical; HAI—Preoccupied	Henderson et al. (2005)
0.38	PMWI—Psychological; HAI—Preoccupied	Henderson et al. (2005)
0.13	CIR—Physical; ECR	Miga et al. (2010)
0.15	PMES-Verbal; ECR	Miga et al. (2010)
0.26	CIR—Physical; AAI Q-set	Miga et al. (2010)
0.25	PMES-Verbal; AAI Q-set	Miga et al. (2010)
0.17	CTS2—Physical; AAS	Sommer et al. (2017)
0.17	CTS2—Sexual; AAS	Sommer et al. (2017)
0.17	CTS2—Physical; AAS	Sommer et al. (2017)
0.21	CTS2—Psychological; AAS	Sommer et al. (2017)
0.24	CTS2—Sexual; AAS	Sommer et al. (2017)
0.33	CTS—Violence; RQ	Bookwala and Zdaniuk (1998)
0.31	CTS2—Psychological; ECR-R (men)	Fournier et al. (2011)
0.28	CTS2—Physical; ECR-R (men)	Fournier et al. (2011)
0.33	MMEA; ECR-R (men)	Gormley and Lopez (2010)
0.24	CTS—Physical; ECR (men)	Mauricio et al. (2007)
0.50	CTS–Psychological; ECR (men)	Mauricio et al. (2007)

Intimate partner violence victimization and attachment anxiety		
r_p	Measure	Source
0.19	CTS2—Physical; ECR-R	Study 1 (partial *r*)
0.23	CTS2—Psychological; ECR-R	Study 1 (partial *r*)
0.17	CTS2—Sexual; ECR-R	Study 1 (partial *r*)
0.23	CTS2—Physical; HAI–Preoccupied	Henderson et al. (2005)
0.38	PMWI—Psychological; HAI–Preoccupied	Henderson et al. (2005)
0.23	CTS2—Physical; ECR	Sandberg et al. (2016)
0.36	CTS—Physical; RQ	Doumas et al. (2008)
0.26	PMWI—Psychological; RSQ—Preoccupied (women)	Dutton et al. (1994a, 1994b)

3.2. Results

Each simulation was set to run $k = 10{,}000$ calculations, each time drawing a random value from the distributions of h^2_X, h^2_Y, and r_p for each simulation model (A and B, distributions displayed in Fig. 1), and each time using a specified value of r_g. Each simulation model will be discussed in turn. Simulation code is available in the ESM.

3.2.1. Simulation A

Simulation A examined the extent to which the shared genetic variation between IPV perpetration and attachment anxiety could explain the phenotypic correlation between the two traits. For this variable pair, values of r_g were set to range between 0.00 and 0.25, in increments of 0.01. For each value of r_g, 10,000 calculations were conducted, each time selecting a random value from the distributions of

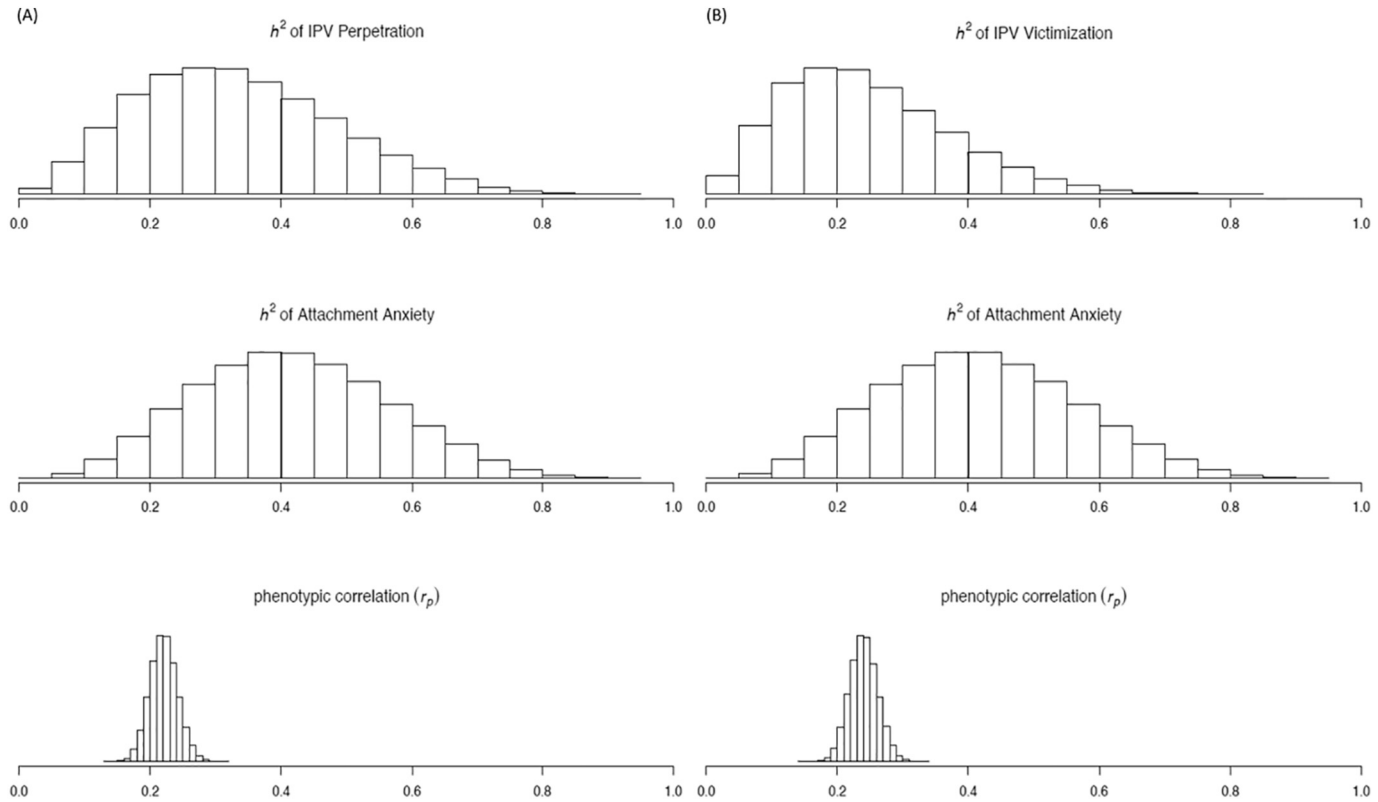

Fig. 1. Probability (beta) distributions for variable pairs used in simulation analyses. (A) = Simulation for h^2_{cov} of Intimate Partner Violence Perpetration and Attachment Anxiety; (B) = Simulation for h^2_{cov} of Intimate Partner Violence Victimization and Attachment Anxiety.

h^2_X, h^2_Y, and r_p (see Fig. 1) to solve h^2_{cov}. These calculations result in a distribution of h^2_{cov} at each value of r_g. The results for simulation A are presented in a series of 24 histogram plots displayed in Fig. 2 (exact values are presented in Table 4). Beginning with the upper-most left panel of Fig. 2, where $r_g = 0.01$, shared genetic variance explains 0% of the phenotypic association. As r_g increases to more plausible, but conservative values, however ($r_g > 0.10$), shared genetic variance can explain approximately 20% of the association, and potentially, > 40% of the association when r_g is estimated at 0.25. At the maximum estimated value of $r_g = 0.25$, shared genetic variation has the *potential* to explain nearly 70% of the phenotypic association between IPV perpetration and attachment anxiety, as indicated by the 95% credibility interval. What these results also suggest is that the remaining portion of the phenotypic association (1- h^2_{cov}) is explained by environmental factors.

3.2.2. Simulation B

Simulation B examined the extent to which the shared genetic variation between IPV victimization and attachment anxiety could explain the phenotypic correlation between the two traits. For this variable pair values of r_g were set to range between 0.00 and 0.25, in increments of 0.01. Calculations for simulation B followed the same procedure as outlined above for simulation A. The results for simulation B are presented in a series of 24 histogram plots displayed in Fig. 3 (exact values are presented in Table 4). The results of simulation B largely mirror those for simulation A. Beginning with the upper-most left panel of Fig. 3, where $r_g = 0.01$, shared genetic variance explains 0% of the phenotypic association. As r_g increases to more plausible, but conservative values, however ($r_g > 0.10$), shared genetic variance can explain approximately 15% of the association, and > 32% of the association when r_g is estimated at 0.25. At the maximum estimated value of $r_g = 0.25$, shared genetic variation has the *potential* to explain up to 56% of the phenotypic association between IPV perpetration and attachment anxiety, as indicated by the 95% credibility interval. What these results also suggest is that the remaining portion of the phenotypic association (1- h^2_{cov}) is explained by environmental factors.

4. General discussion

The current research examined the robustness of the association between attachment anxiety and intimate partner violence (IPV). In Study 1, we replicated the robust associations of attachment anxiety with both IPV perpetration and victimization. Study 1 also identified strong positive associations between domains of IPV perpetration and victimization—consistent with the victim-offender overlap hypothesis (Barnes & Beaver, 2012; Hines & Saudino, 2004). Study 2 examined the extent to which the associations between attachment anxiety and IPV outcomes would be robust to known genetic influences operating on each trait.

The current research has two primary findings of interest. Our results suggest that a portion of the phenotypic association between attachment anxiety and IPV outcomes can be explained to the extent to which genetic variation operating on attachment anxiety covaries with genetic variation operating on IPV outcomes. Second, although our model estimates suggest that shared genetic variation can explain a non-negligible proportion of the phenotypic association between attachment anxiety and IPV outcomes, a significant proportion of the phenotypic association remains to be explained by environmental variation. In other words, the observed associations between attachment anxiety and IPV reported in the literature appear to be robust to plausible genetic covariation. These findings of the current research provide an important extension of the few available studies on the genetic nature of IPV (Barnes et al., 2013; Hines & Saudino, 2004) and adult attachment (Brussoni et al., 2000; Crawford et al., 2007; Donnellan et al., 2008). The extent to which genetic influences on attachment anxiety and IPV covary can provide a novel explanation for

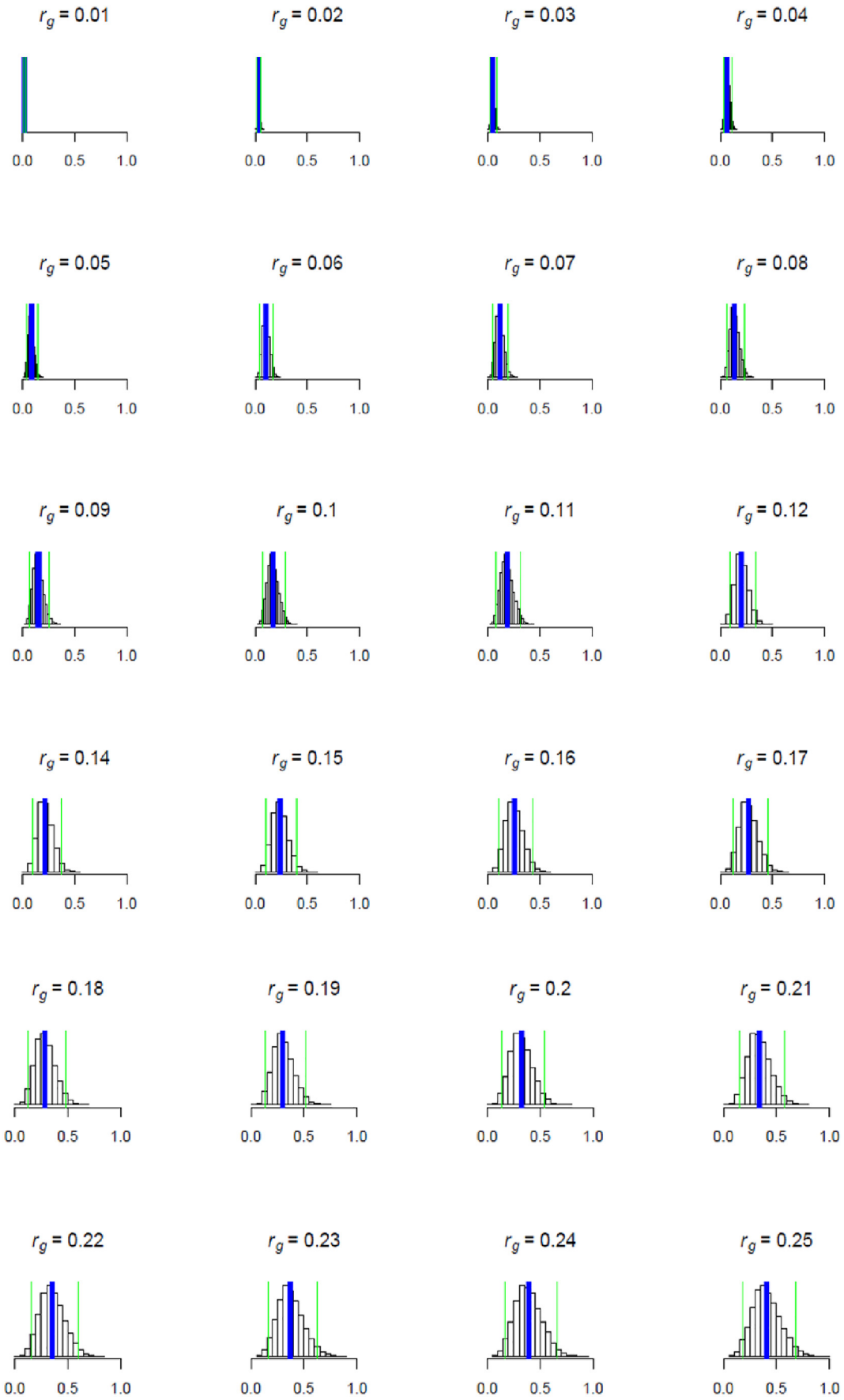

Fig. 2. Distribution of h^2_{cov} of Intimate Partner Violence Perpetration and Attachment Anxiety (simulation A) at different values of r_g. Blue (middle) bars represent mean values. Green (outer) bars represent 95% credibility intervals. See Table 4 for exact values. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 4
Mean and mode values, and 95% credibility intervals for h^2_{cov} estimates.

IPV Perpetration & attachment anxiety				IPV Victimization & attachment anxiety			
r_g	h^2_{cov} mean	h^2_{cov} mode	95% CI	r_g	h^2_{cov} mean	h^2_{cov} mode	95% CI
0.00	0.00	0.00	0.00–0.00	0.00	0.00	0.00	0.00–0.00
0.01	0.02	0.02	0.01–0.03	0.01	0.01	0.01	0.01–0.02
0.02	0.03	0.03	0.01–0.06	0.02	0.03	0.02	0.01–0.05
0.03	0.05	0.05	0.02–0.09	0.03	0.04	0.04	0.02–0.07
0.04	0.07	0.06	0.03–0.11	0.04	0.05	0.05	0.02–0.09
0.05	0.08	0.07	0.04–0.14	0.05	0.07	0.06	0.03–0.12
0.06	0.10	0.09	0.04–0.17	0.06	0.08	0.07	0.03–0.14
0.07	0.12	0.10	0.05–0.20	0.07	0.09	0.08	0.04–0.16
0.08	0.13	0.12	0.06–0.23	0.08	0.10	0.09	0.05–0.19
0.09	0.15	0.13	0.07–0.26	0.09	0.12	0.11	0.05–0.21
0.10	0.17	0.15	0.07–0.29	0.10	0.13	0.12	0.06–0.23
0.11	0.19	0.16	0.08–0.31	0.11	0.15	0.13	0.06–0.25
0.12	0.20	0.18	0.09–0.34	0.12	0.16	0.14	0.07–0.28
0.14	0.22	0.19	0.10–0.37	0.14	0.17	0.15	0.07–0.30
0.15	0.24	0.21	0.10–0.40	0.15	0.19	0.16	0.08–0.32
0.16	0.25	0.22	0.11–0.43	0.16	0.20	0.18	0.08–0.35
0.17	0.27	0.24	0.12–0.46	0.17	0.21	0.19	0.09–0.37
0.18	0.29	0.26	0.13–0.49	0.18	0.23	0.20	0.10–0.39
0.19	0.30	0.27	0.13–0.51	0.19	0.24	0.21	0.10–0.42
0.20	0.32	0.28	0.14–0.54	0.20	0.26	0.22	0.11–0.44
0.21	0.34	0.30	0.15–0.57	0.21	0.27	0.24	0.11–0.46
0.22	0.36	0.32	0.15–0.60	0.22	0.28	0.25	0.12–0.49
0.23	0.37	0.33	0.16–0.63	0.23	0.29	0.26	0.12–0.51
0.24	0.39	0.34	0.17–0.66	0.24	0.31	0.27	0.13–0.53
0.25	0.41	0.36	0.18–0.69	0.25	0.32	0.28	0.14–0.56

why attachment anxiety is associated with both perpetration and victimization.

4.1. Research implications

Several implications for our understanding of the association between attachment anxiety and IPV perpetration and victimization follow from the results of the current research. First, because the genetic influences on IPV perpetration and victimization almost completely overlap (Hines & Saudino, 2004), perpetration and victimization are two-sides of the same coin *from a behavioral genetic perspective* (Plomin et al., 2016)—although perpetration and victimization can have different *phenotypic* expressions and consequences. The primary implication of this is that, from a behavioral genetic perspective, IPV perpetration and victimization can be largely considered the same trait when examining genetic relationships with associated traits, such as we do here with attachment patterns. The understanding of the genetic nature of IPV must account for this substantial overlap.

Second, given that the genetic structure of domains is similar to the phenotypic structure (Plomin et al., 2016), the simulation models from Study 2 provide the first reported estimates of the proportion of the phenotypic association between attachment anxiety and IPV experience accounted for by genetic covariation between the traits. Our models suggest that approximately 25% of the phenotypic correlation between attachment anxiety and IPV experience may plausibly be a result of common genetic influences operating on both traits. An implication of this finding—in combination with the findings of Barnes et al. (2013) and Hines and Saudino (2004)—is that continued research on IPV and its associated psychological risk factors must consider the impact of genetic influences to achieve an accurate understanding of these relationships. Failure to appropriately control for such influences may result in unacknowledged genetic confounding of phenotypic associations or biased estimates—which are especially problematic for small phenotypic effects (i.e., $r_p < 0.20$; Barnes et al., 2014)

Third, the results of the current research suggest that more than one-half of the association between attachment anxiety and IPV experience can plausibly be explained by *environmental variation* underpinning the traits. That environmental variation might explain a substantial portion of the phenotypic association accords with environmental-based perspectives on IPV and associated individual differences (see Ali & Naylor, 2013a, 2013b). Such environmental-based perspectives include ecological systems theories that propose that differential exposure to violence-conducive environments – such as with peers or with family (e.g., Valgardson & Schwartz, 2019) – increase the likelihood that individuals will be in situations in which perpetration and victimization are more likely to occur (Berg & Mulford, 2017). With regard to adult attachment anxiety, twin models show that more than half of the observed variance is explained by non-shared environmental factors (Brussoni et al., 2000; Crawford et al., 2007; Donnellan et al., 2008). The implication of this finding is that unique experiences influencing attachment anxiety may covary with unique experiences influencing IPV experiences. A primary take-away is that the well-documented association between attachment anxiety and IPV outcomes appears relatively robust to potential genetic confounds, but genetically-sensitive data are needed to confirm the model estimates reported here.

In addition to the empirical implications, the current research suggests future avenues for practice. Attachment dynamics are foundational to romantic relationship functioning. Attachment patterns predict several classes of aggressive behavior toward partners, including controlling behaviors (Barbaro et al., 2016, 2019), affect escalation (Shaver & Mikulincer, 2008), and physical violence (see Table 3). Our results, moreover, suggest that the impact of attachment on IPV is likely to persist, even when genetic variation is taken into account. Assessment of attachment patterns, therefore, may be advisable for practitioners as a target of cognitive therapies (e.g., cognitive reframing of anxiety; Hofmann & Smits, 2008) focused on reducing both (re)perpetration and (re)victimization. Relatedly, practitioners treating individuals seeking assistance for perpetration or victimization need to take into account the victim-offender overlap (Anderson, 2002; Langhinrichsen-Rohling et al., 2012; Renner & Whitney, 2012), and other related individual difference factors, such as self-control, that are also related to aggression, more generally (Vazsonyi, Mikuška, & Kelley, 2017). Efforts to reduce instances of re-victimization, for example, must

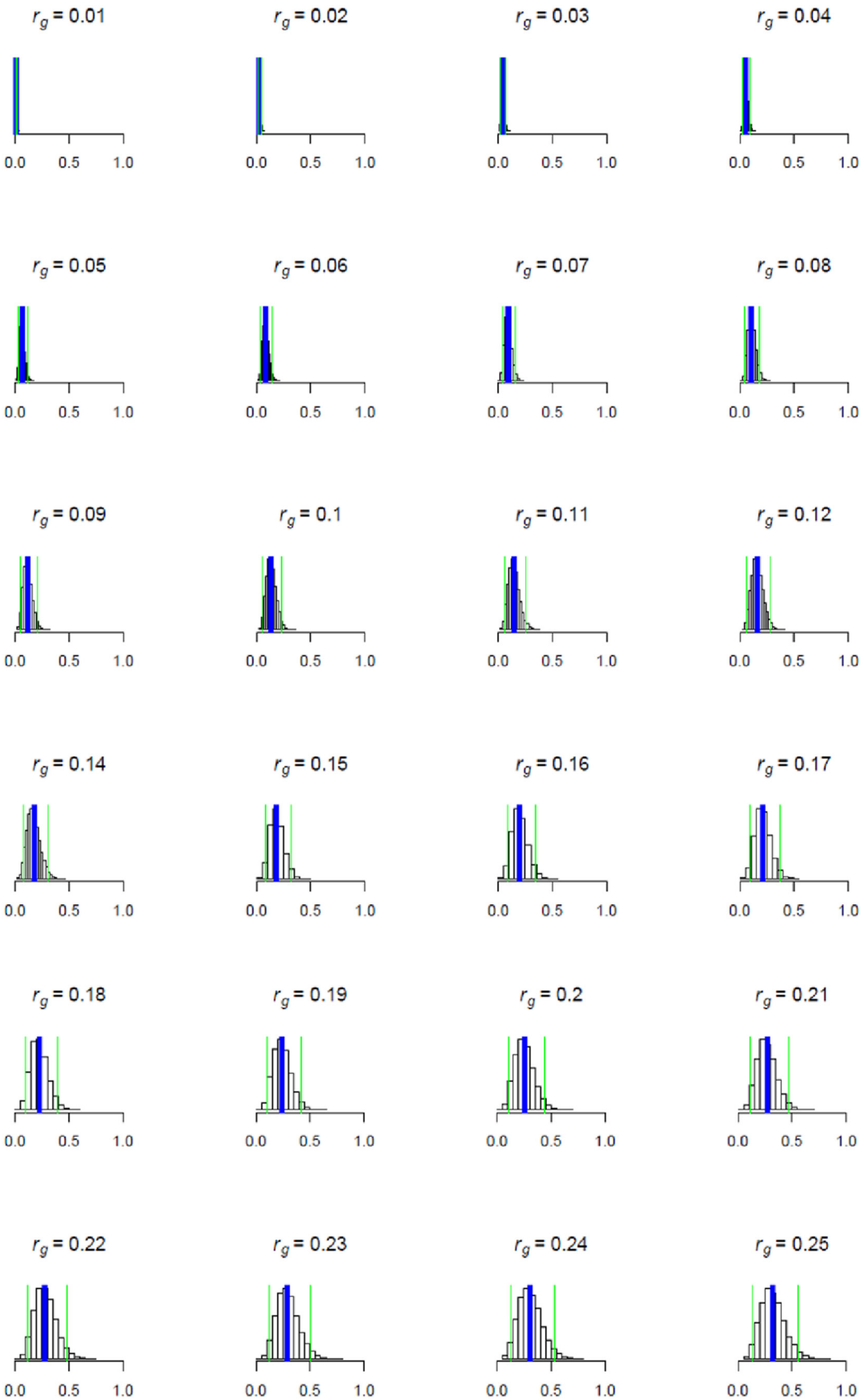

Fig. 3. Distribution of h^2_{cov} of Intimate Partner Violence Victimization and Attachment Anxiety (simulation B) at different values of r_g. Blue (middle) bars represent mean values. Green (outer) bars represent 95% credibility intervals. See Table 4 for exact values. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

focus on not only preventative efforts of re-victimization, but also preventative efforts for future perpetration, given that individuals experiencing one side of relationship violence are at increased risk for experiencing the other side of relationship violence.

4.2. Limitations

Limitations of the current research suggest directions for future investigation. First, rates of self-reported perpetration and self-reported victimization may be biased (i.e., under- or over-reported; Ackerman, 2018; Hamby, 2005). Collection of dyadic data (i.e., perpetration and victimization reports from both members of a romantic couple) can, to an extent, mitigate issues of biased self-reports (Sommer et al., 2017). Continued research should include multiple or varied measures of IPV given differences in reporting across measures (see Cascardi & Muzyczyn, 2016). It should also be noted that our sample differs from the general IPV population in the United States (Caetano, Vaeth, & Ramisetty-Mikler, 2008), and generalizability of our findings to diverse populations may not be appropriate.

The sample size of Study 1 was underpowered for accurate detection of interaction effects, and thus we chose not to interpret significant interactions found in our analyses (results available in the ESM). Across models and outcomes, attachment anxiety was a reliable and robust predictor of perpetration and victimization outcomes; in contrast, two-way interactions explored were inconsistent across models, with the majority of *p*-values ranging between 0.01 and 0.05, indicating higher likelihood of false positives.

We were unable to include all potential covariates in our models. This research focused on the potential role of unmeasured genetic factors on the phenotypic associations between attachment patterns and IPV outcomes. Previous research has shown that other individual difference factors covary with attachment anxiety, such as neuroticism (Noftle & Shaver, 2006); and covary with perpetration and victimization, such as intelligence (Boutwell et al., 2017). The omission of alternative covariates should be considered when interpreting the results.

Lastly, the simulation strategy employed in Study 2 relies on data available in the literature to build the beta distributions, which are used as input parameters for the simulations. The accuracy of the model estimates presented in Table 4 therefore rest on the validity of the input parameters obtained from previous research. Published phenotypic correlations between attachment anxiety and IPV (see Table 3) may be affected by publication bias, and therefore be overestimates. If this is the case and the true phenotypic correlations are smaller than what are used in the current research, the resulting h^2_{cov} estimates would likely be *greater* because smaller phenotypic effects are more susceptible to genetic confounding (Barnes et al., 2014, 2017; Plomin et al., 2016). The estimates reported here are therefore likely to be conservative. That said, confirmation of the reported estimates with genetically-sensitive datasets (e.g., twin or sibling data) that include both attachment and IPV measures is warranted. Thus far, genetic analyses have only been published on each trait independently. From a practical perspective, what these simulations can provide is mathematical justification for the need to conduct genetically-sensitive empirical research on these traits.

4.3. Conclusion

The current research examined the associations between attachment anxiety and IPV perpetration and victimization. The results of Study 1 supported the hypothesis that attachment anxiety, but not attachment avoidance, is positively associated with IPV experiences, according with the majority of the published literature. Using simulation-modeling, Study 2 demonstrated the probable robustness of the well-documented association between attachment anxiety and IPV outcomes. The modeling results also offer a novel explanation for why attachment anxiety may be a strong predictor of both perpetration and victimization: A moderate proportion of the genetic underpinnings of the traits likely covary, and can account for, in part, the observed association. The results of the current research highlight the need to assess both perpetration *and* victimization in IPV research, given the victim-offender overlap of IPV (Langhinrichsen-Rohling et al., 2012), and highlight the importance of examining (or at least controlling for) genetic variation (Hines & Saudino, 2004) in future research to provide a more comprehensive understanding of IPV.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.paid.2019.05.008.

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