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Gender Moderates the Association Between Exposure to Interpersonal Violence and Intermittent Explosive Disorder Diagnosis

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Abstract

Exposure to interpersonal violence (EIV) is a prevalent risk-factor for aggressive behavior; however, it is unclear whether the effect of EIV on clinically significant aggressive behavior is similar across gender. We examined whether gender moderates the association between experiencing and witnessing interpersonal violence and the diagnosis of intermittent explosive disorder (IED). We also examined potential pathways that might differentially account for the association between EIV and IED in men and women, including emotion regulation and social information processing (SIP). Adult men and women (N = 582), who completed a semistructured clinical interview for syndromal and personality disorders, were classified as healthy controls (HC; n = 118), psychiatric controls (PC; n = 146) or participants with an IED diagnosis (n = 318). Participants also completed the

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life history of experienced aggression (LHEA) and life history of witnessed aggression (Lhwa) structured interview and self-report measures of emotion regulation and SIP. Men reported more EIV over the lifetime. In multiple logistic regression analysis, experiencing and witnessing aggression within the family and experiencing aggression outside the family were associated with lifetime IED diagnosis. We found that the relationship between EIV and IED was stronger in women than in men. Affective dysregulation mediated certain forms of EIV, and this relation was observed in both men and women. SIP biases did not mediate the relation between EIV and IED. EIV across the lifespan is a robust risk factor for recurrent, clinically significant aggressive behavior (i.e., IED). However, the relationship between EIV and IED appears to be stronger in women. Further, this relation appears partially mediated by affective dysregulation.

Keywords
trauma, aggression, intermittent explosive disorder, gender, childhood maltreatment

Introduction

Exposure to interpersonal violence (EIV) is a widespread public health issue that can exert lasting negative effects on health and well-being (Finkelhor et al., 2009). EIV includes experiencing or witnessing violence in the family, community, or among peers, and includes exposure to abuse, family violence, and community violence. Studies show that lifetime exposure to traumatic events, including interpersonal violence spanning from childhood to adulthood, is associated with recurrent, clinically significant impulsive aggressive behavior in the form of intermittent explosive disorder (IED) (Fanning et al., 2014; Fincham et al., 2009; Nickerson et al., 2012). Less is known, however, about how gender impacts the association between lifetime EIV and clinically significant aggressive behavior.

Early Aggression Exposure

Children and adolescents in the United States experience and witness aggression at a high rate, with up to 60.6% of people under age 18 having experienced or witnessed abuse in the past year (Finkelhor et al., 2009). Further, exposure to violence is often chronic and recurrent (Finkelhor et al., 2009). Childhood EIV is associated with concurrent aggression and expressions of anger (Maxwell & Maxwell, 2003; Wongtongkam et al., 2017) as well as

increased risk for psychopathology (McLaughlin et al., 2012) and later aggression (Fanning et al., 2014; Farrell et al., 2014). These relationships have been observed in children, adolescents, and adults who experienced and witnessed violence in the home and in the community. The potential effects of childhood EIV (including witnessing community and familial violence) may persist into adulthood, as suggested by studies showing links between EIV and subsequent physical aggression and psychological distress (Farrell et al., 2020; Henning et al., 1997).

There is evidence that boys and girls differ in exposure to forms of violence and may show different exposure outcomes. Maxwell and Maxwell (2003) found that, although boys and girls reported similar rates of witnessing aggression, boys both experienced more aggression and reported committing more aggressive acts. However, findings are mixed as to whether gender moderates the effect of EIV on aggression (Farrell et al., 2020; Henning et al., 1997; Tisak et al., 2019). Relatedly, it is unclear how gender moderates pathways implicated in aggressive behavior following trauma.

Heritability of Aggressive Behavior

Evidence from twin and family studies suggests that both heritable and environmental factors contribute to aggressive behavior. Genetic factors exert a large influence on aggression in childhood and over the lifespan (Porsch et al., 2016), and estimates of heritability of aggression are consistently around 50% or higher (Coccaro et al., 1997a; Miles & Carey, 1997; Porsch et al., 2016). However, nonshared environmental influences also contribute significantly to aggressive behavior (Burt, 2009b; Burt & Klump, 2012; Coccaro et al., 1997a). By comparison, studies find relatively less impact of shared environmental factors on aggression (Burt, 2009a, 2009b; Coccaro et al., 1997a; Rowe et al., 1999). Evidence is mixed as to whether heritable and environmental influences on aggression differ by gender. Some studies found greater heritability among boys, others among girls, and some studies found no difference (e.g., Burt, 2009a; Mason & Frick, 1994; Miles & Carey, 1997).

Mediation Pathways

Emotion regulation.

A range of biological, psychological, and social mechanisms are implicated in the relationship between early exposure to violence and difficulties with aggressive behavior (Kerig & Becker, 2010). Evidence suggests that emotion regulation deficits play a role in EIV and aggressive behavior. As defined by Linehan et al. (2007), emotion dysregulation is the inability to control

emotional cues, experiences, actions, and verbal or behavioral responses in normative situations. Research suggests that EIV negatively impacts emotion regulation throughout the lifespan. Indeed, a large body of literature supports the notion that witnessing and experiencing aggression leads to emotion dysregulation in children (Calkins, 1994; Cooley & Fite, 2016; Shackman & Pollak, 2014; Thompson & Calkins, 1996). A longitudinal study of juvenile offenders (N = 892) found that increasing emotional regulation skills can cause a decline in aggressive behaviors (Kim, 2018). Maladaptive emotion regulation skills are also a significant predictor of aggression in adult offenders (Roberton et al., 2014), of physical assault perpetration (Grigorian et al., 2020) and of IED diagnosis (Fettich et al., 2015). Most of these studies did not explore gender differences in emotion dysregulation in their samples; those that do find small or no differences (Kim, 2018; Roberton et al., 2014). In the current study, we examine the effect of gender on emotion regulation, a potential mediator of the association between EIV and aggression behavior.

Social information processing

EIV may also contribute to biases in social information processing (SIP). SIP theory proposes that biases in cognitive and emotional processes involved in evaluating and reacting to social cues contribute to maladaptive behavior such as aggression (Dodge et al., 1990). SIP has been shown to be impacted by adverse childhood experiences and emotional processes, and deficits in SIP have been robustly linked to aggressive behavior (Dodge, 2011) including IED (Coccaro et al., 2009, 2016). Children and adolescents who witness or experience violence show deficits in SIP, including hostile attribution bias, hypervigilance to potential threat, and the inability to access appropriate behavioral responses (Berlin et al., 2011; Calvete & Orue, 2011; Dodge et al., 1990). Adolescents who experienced severe violence demonstrated increased hostile attribution bias, greater approval of violence, and more hostile social goals, whereas those who witnessed severe violence reported greater confidence that violence would result in a positive outcome (Shahinfar et al., 2001). SIP deficits associated with adverse childhood experiences can last into early adulthood, and these deficits can mediate violence exposure and aggressive behavior (Berlin et al., 2011; Chen et al., 2012; Pettit et al., 2010). There is evidence that boys generate more aggressive responses than girls (Feldman & Dodge, 1987) and that women perceive more goal-directed intent and report greater negative affect in response to aggressive vignettes than men (Coccaro et al., 2009). Accordingly, there is evidence that SIP biases may contribute to the link between EIV and aggressive behavior, and that these contributions may differ between men and women. As biases in SIP may contribute to aggressive behavior, we examine how SIP may mediate the pathways from EIV to aggressive behavior.

Gender differences

Overall, there is evidence suggesting potential gender differences in the victimization-aggression link, but little direct evidence of moderation of this association by gender. We therefore seek to examine how gender moderates the relationship between EIV and recurrent impulsive aggressive behavior, or IED diagnosis. We also examine how gender may moderate the pathways from EIV to aggressive behavior.

Trauma Exposure

EIV is potentially traumatic depending on the nature of the exposure. Gender differences are well documented among other disorders related to trauma exposure. A well-replicated finding is that women have higher rates of post-traumatic stress disorder (PTSD) compared to men (e.g., Breslau et al., 1997). PTSD symptoms are a robust risk factor for aggressive behavior (Orth & Wieland, 2006), suggesting women may show a stronger relationship between victimization and aggression. On the other hand, men have been shown to manifest more externalizing psychopathology and women more internalizing psychopathology in general (Kramer et al., 2008). This pattern of comorbidity has also been observed in borderline personality disorder, which is associated with traumatic exposure, affective dysregulation, and aggressive behavior (Johnson et al., 2003). However, results are mixed (Hourani et al., 2015). A meta-analysis on the association between trauma exposure on aggression did not find a significant association between the proportion of females studied and effect size (Orth & Wieland, 2006).

In the current study, we examine how gender moderates the relationship between EIV and recurrent impulsive aggressive behavior, or IED diagnosis. We also investigate the impact of gender on psychological variables, emotion regulation and SIP, that are implicated in aggressive behavior following exposure to trauma. We examined these questions in a large sample of participants with IED, psychiatric controls (PC) participants, and healthy volunteers. We predict that exposure to aggression will be associated with IED diagnosis in both men and women. We also predict that the indirect pathway from exposure to IED through impaired emotion regulation will be stronger in women than in men. Finally, we predict that gender will moderate the role of SIP biases in the EIV-aggression association. Specifically, we predict that biases in SIP response evaluation processes will represent a stronger indirect effect in men compared to women. We examine lifetime IED diagnosis, rather than a lifetime count of aggression, to reflect clinically significant recurrent aggressive behavior.

Methods

Participants

The sample included 582 men (n = 258) and women (n = 324) participants aged 18 to 58 (M = 34.1; SD = 9.6) recruited from the Chicago community. Based on clinical interviews (see below), participants were assigned to the following three groups: healthy control (HC) participants (n = 146); PC participants (n = 118), and IED participants (n = 318). Self-identified race and ethnicity for the sample are as follows: African American 31%, White 54%, Hispanic or Latino 9%, Asian or Asian American 5%, and another category 2%. Participants comprised the full range of socioeconomic status (SES) (Hollingshead categories: Category 1, 11%, Category 2: 50%, Category 3, 15%, Category 4, 8%, Category 5, 14%). Volunteers were recruited through media advertisements seeking individuals who: (a) reported psychosocial difficulties related to DSM-5 diagnoses (PC or IED); or (b) had little evidence of psychopathology (HC). PC participants were included in analyses to control for potential confounding between aggression and general psychopathology that can occur in case-control study designs. All participants provided written informed consent in accordance with procedures approved by the Institutional Review Board. Participants were compensated $50 for completing the research interview and $25 for completing the self-report questionnaire battery.

Semistructured Clinical Interviews

Participants were systematically evaluated for syndromal and personality disorder diagnoses using in-person semistructured clinical interviews followed by a team best-estimate consensus procedure as previously described (Coccaro et al., 2016). Interviews were conducted by master's or doctoral-level clinicians who completed a diagnostic interviewing training program and reliability testing. Lifetime history of aggression by the research participant was assessed using the Life History of Aggression interview, Aggression subscale (LHA-A; Coccaro et al., 1997b). The LHA-A interview assesses the life history of actual aggression including temper outbursts, verbal aggression, and physical aggression. The LHA-A shows good internal consistency (α = .87) and test-retest reliability (r = .91; Coccaro et al., 1997b).

Lifetime exposure to aggression as a target or witness was assessed using the life history of experienced aggression (LHEA) and the life history of witnessed aggression (Lhwa; Coccaro et al., 2021) interviews. The Lhea assesses the number of instances of experiencing aggression, while the Lhwa assesses the number of instances of witnessing aggression. The Lhea and the

Lhwa both include multiple forms of aggression: temper tantrums, verbal fighting, physical assaults on property, physical assaults that did not end in injury, and physical assaults that did end in injury. Both the Lhea and Lhwa assess aggression exposure within and outside of the family and across stages of life (childhood, adolescence, and adulthood). In a validation sample of 77 adult participants (38 men, 39 women), with and without DSM psychopathology, the Lhea and Lhwa demonstrated excellent internal consistency reliability (α = .93 to .95). The Lhea and Lhwa within-family scales correlate strongly with the Childhood Trauma Questionnaire (CTQ-2; Bernstein et al., 1994), physical and emotional abuse subscales (LHEA: r = .66, p < .001; Lhwa: r = .51, p < .001) and less strongly with the CTQ neglect (LHEA: r = .46, p <.001; Lhwa: r = .40, p < .001) and sexual abuse (LHEA: r = .26, p < .001; Lhwa: r = .24 = p < .001) subscales. The CTQ assesses experiences of abuse and neglect during childhood and adolescence but does not have separate subscales for exposure within versus outside the family and does not separately assess different stages of life. In the current sample, exposure to aggression in childhood correlated strongly with exposure in adolescence among both men (r = .87, p < .001) and women (r = .87, p < .001) participants. Exposure to aggression in adolescence and adulthood also correlated strongly, albeit somewhat less so, in men (r = .65, p < .001) and women (r = .71, p < .001) participants. To avoid issues of collinearity in regression analyses, exposure to and witnessing aggression are considered across the lifespan, respectively.

Self-report Measures

Participants completed a series of self-report questionnaires in the laboratory in either paper-and-pencil or computerized format. The Affective Lability Scale (ALS; Harvey et al., 1989) is a 54-item, self-report measure of changeability in various moods. The measure includes subscales assessing anxiety, depression, mania, and anger. The ALS total score was used as a measure of global affective dysregulation in the current study.

The Affect Intensity Measure (AIM; Larsen & Diener, 1987) is a 40-item, self-report questionnaire designed to assess the degree or intensity of one's emotional experiences. The AIM assesses positive and negative affectivity, reactivity, and affective intensity along with other aspects of emotional experience (Larsen & Diener, 1987). The AIM has good test-retest reliability at 1- to 3-month intervals (r = .80-.81) and excellent internal consistency (α = .90 to .94; Larsen & Diener, 1987). The AIM negative reactivity and negative intensity subscales were used in this study as indexes of negative affective instability and negative affective intensity.

The Social-Emotional Information Processing Questionnaire (SEIP-Q; Coccaro et al., 2017) is a self-report, vignette-based assessment of social and emotional information processing constructs. The measure includes eight hypothetical vignettes describing ambiguous social situations with a negative outcome. For each vignette, the participant is asked about their attributions of the situation and their emotional response. For each scenario, three hypothetical response scenarios are presented including a prosocial, overtly aggressive, and relationally aggressive response. Participants are asked to evaluate each possible response, yielding scores for response valuation, response efficacy, outcome expectancy, and (likelihood of) response enactment. The SEIP-Q shows good internal consistency and distinguishes healthy from aggressive research participants. For the current study we examined hostile attribution response bias (HAB), negative emotional response (NER), and the aggregated SIP response evaluation variables (SIP-RED) for overt and relational aggression responses as measures of SIP processes.

Statistical Analyses

Assumptions of statistical tests were evaluated, and steps were taken to address violations of assumptions. Bonferroni correction for multiple comparisons was applied where appropriate. We compared HC, PC, and IED participants on demographic variables, diagnostic comorbidity, and study variables using chi-square analysis, analysis of variance (ANOVA), and *t* tests. Brown-Forsythe F statistic and Games-Howell *post hoc* test were used where homogeneity of variance was violated. We compared men and women on diagnostic comorbidity and study variables using chi-square analysis and independent samples *t* tests. Next, we examined gender differences in reported EIV across the lifespan using repeated measures ANOVA. Hierarchical binary logistic regression was used to evaluate the unique associations between experiencing and witnessing aggression within and outside the family and lifetime IED diagnosis, controlling for covariates. Significant predictors were evaluated as independent variables (IVs) in separate subsequent path models in PROCESS implemented in SPSS (Hayes, 2013). Potential mediator variables were selected based on significant ($p < .05$) bivariate correlations with Lhea and Lhwa and the LHA. Bivariate correlations were conducted in the full sample and separately for men and women, given our interest in gender moderation effects. Direct and indirect mediation effects on IED diagnosis were examined in separate PROCESS models for each IV of interest. All analyses were conducted in SPSS 24.

Results

Group Differences in Demographic Variables

There were no significant differences in gender composition across the three diagnostic groups ($p = .842$). The IED participants were modestly older than the HC and PC participants (HC: $M = 31.34$, $SD = 9.18$; PC: $M = 33.25$, $SD = 9.47$; IED: $M = 35.74$, $SD = 9.60$, $p < .001$; $d = 0.38$). The HC and PC participants were more likely to be White compared to the IED participants (HC: 62.3%; PC: 59.3%; IED: 48.1%; $p = .007$). The PC and IED participants had lower SES than the HC participants (HC: $M = 44.16$, $SD = 12.54$ PC: $M = 39.05$, $SD = 14.68$; IED: $M = 38.10$, $SD = 13.76$; $p < .001$; $d = 0.44$). As such, we added the above demographic variables as covariates in analyses involving diagnostic group, including the PROCESS models.

Group Differences in Trauma and Stressor-Related Variables

The diagnostic groups were compared on trauma- and stressor-related variables. Alpha was set to .007 to account for multiple statistical tests. The three groups differed in overall childhood maltreatment (CTQ-2 total score; $n = 376$; $F[2,371] = 57.7$, $p < .001$), controlling for sex and race. Individuals with IED reported the most maltreatment and healthy participants reported the least. The three groups did not differ in history of military service or parents never being married ($ps > .05$). Individuals with IED were more likely to have divorced parents ($\chi^2 = 23.3$, $df = 4$, $p < .001$; $n = 547$) and were less likely to be raised by both parents ($\chi^2 = 18.3$, $df = 2$, $p < .001$, $n = 544$). Individuals with IED were more likely to have a lifetime diagnosis of trauma- or stressor-related psychiatric disorder ($\chi^2 = 44.0$, $df = 2$, $p < .001$, $n = 582$). In summary, individuals with IED experienced childhood maltreatment, certain adverse childhood experiences, and lifetime trauma-related psychopathology at higher rates compared to other participants.

Life History of Experienced and Witnessed Aggression (Lhea and Lhwa)

Experienced aggression

Men were exposed to significantly higher levels of aggression than women, with modest effect sizes ($d = 0.37$-0.40; see Table 1). Effects of gender, stage of life (childhood, adolescence, and adulthood), and context (in vs out of the family) on EIV were examined. Main effects of gender, stage, and context were observed (Table 2). Men experienced more aggression overall compared to women. Participants reported more experienced aggression within

the family than outside the family. Finally, participants experienced the most aggression in adolescence, followed by childhood, and the least in adulthood. These main effects were qualified by significant interactions: gender × context, gender × stage, and stage × context (Table 2). Men and women experienced similar amounts of aggression within the family, but men experienced more aggression outside of the family than women. Women experienced less aggression in childhood compared to adolescence and adulthood. Across both men and women, participants experienced more aggression within the family in childhood and adolescence and more aggression outside of the family in adulthood.

Table 1. Demographic, Functional, and Psychometric Characteristics of Study Participants.

Psychometric Variables	Men	Women	*p*	Contrast
Experienced agg in family[a]	23.1 ± 16.2	23.6 ± 16.4	=.747	M = F
Experienced agg outside family[a]	21.2 ± 14.1	16.2 ± 13.0	<.001	M > F
Witnessed agg in family[b]	23.7 ± 17.0	24.2 ±17.6	=.700	M = F
Witnessed agg outside family[b]	32.9 ± 17.4	24.9 ± 16.2	<.001	M > F
Affective lability score[c]	96.9 ± 32.7	100.8 ± 36.7	=.220	M = F
AIM negative intensity	18.0 ± 5.7	19.0 ± 6.6	=.079	M = F
AIM negative reactivity	21.2 ± 5.0	23.2 ± 5.1	<.001	M < F
SEIP-Q hostile attribution	0.8 ± 0.5	0.8 ± 0.5	=.605	M = F
SEIP-Q negative emotional response	1.6 ± 0.5	1.6 ± 0.5	=.556	M = F
SEIP-Q response evaluation	2.8 ± 1.7	2.8 ± 1.7	=.749	M = F
SEIP-Q outcome expectation	2.9 ± 1.3	2.8 ± 1.3	=.504	M = F
SEIP-Q aggression efficacy	3.7 ± 2.7	3.7 ± 2.5	=.975	M = F
SEIP-Q aggression enactment	3.5 ± 2.3	3.4 ± 2.1	=.898	M = F
Life history of aggression[d]	13.5 ± 7.3	12.1 ± 7.4	=.017	M = F

Note. Means ± *SD* based on raw data. Bonferroni-corrected.
Statistics based on independent samples *t* test.
[a]Life history of experience aggression, *n* = 580.
[b]Life history of witnessed aggression, *n* = 582.
AIM = affective intensity measure, *n* = 447.
SEIP-Q = social emotional information processing questionnaire, n = 353.
[c]*n* = 475. [d]n = 558.

Table 2. Main Effects and Interactions on LHEA.

Effect	*F*	*df*	*p*	Contrast	Main Effect	Interaction	*M* (*SE*s)
Gender	4.1	1,578	=.045	M > W	M		7.40 (0.30)
					W		6.64 (0.23)
Stage	20.3	2,1156	<.008	Ado > Chi > Adu	Chi		7.03 (0.21)
					Ado		7.50 (0.21)
					Adu		6.52 (0.19)
Context	68.8	11,156	<.001	In > Out	In		7.79 (0.23)
					Out		6.25 (0.19)
Sex × stage	24.2	21,156	<.001	M > W	Chi	M	7.83 (0.32)
						W	6.23 (0.29
				M > W	Ado	M	8.04 (0.31)
						W	6.96 (0.28)
				M = W	Adu	M	6.32 (0.29)
						W	6.75 (0.26)
Sex × context	23.6	11,156	<.001	M = W	In	M	7.71 (0.34)
						W	7.86 (0.30)
				M > W	Out	M	7.08 (0.28)
						W	5.43 (0.25)

(*continued*)

Table 2. continued

Effect	*F*	*df*	*p*	Contrast	Main Effect	Interaction	*M* (*SE*s)
Stage × context	243.1	21,156	<.001	In > Out	Chi	In	8.82 (0.28)
						Out	5.23 (0.21)
				In > Out	Ado	In	8.74 (0.26)
						Out	6.26 (0.21)
				Out > In	Adu	In	5.79 (0.22)
						Out	7.27 (0.22)

Note. n = 580; M = men; W = women; Chi = childhood, Ado = adolescence; Adu = adulthood.

Witnessed aggression

Main effects of gender, stage, and context were observed on witnessed aggression (Table 3). Men witnessed more aggression overall compared to women. Participants reported less witnessed aggression within the family than outside the family. Finally, participants witnessed the most aggression in adolescence, followed by childhood, and finally adulthood. These main effects were qualified by significant interactions: gender × context, gender × stage, and stage × context. Specifically, men and women witnessed similar amounts of aggression within the family, but men witnessed more aggression outside of the family than women. Compared to women, men witnessed more aggression overall in childhood and adolescence; however, men and women witnessed similar levels of aggression in adulthood. Across both men and women, participants witnessed more aggression within the family in childhood and more aggression outside of the family in adolescence and adulthood. Overall, men reported experiencing and witnessing more aggression compared to women, which was due to greater exposure outside of the family. EIV was highest in adolescence.

Exposure to Interpersonal Violence Predicting IED

Hierarchical binary logistic regression analysis controlling for age, gender, race, and SES was used to evaluate the independent effects of EIV variables on IED diagnosis. In Step one, covariates significantly predicted IED diagnosis (*Wald* $\chi^2 = 34.66$, $df = 4$, $p < .001$, *Nagelkerke* $R^2 = .08$). The addition of

Lhea and Lhwa predictors (total aggression exposure within and outside the family, respectively) significantly improved the model fit (Wald $\chi^2 = 232.96$, $df = 8$, $p < .001$, *Nagelkerke* $R^2 = .44$). Age ($B = 0.03$, $SE = 0.01$, *Wald* $\chi^2 = 8.59$, $p = .003$), Lhea within the family ($B = 0.05$, $SE = 0.01$, *Wald* $\chi^2 = 15.62$, $p < .001$), Lhea outside the family ($B = 0.05$, $SE = 0.01$, *Wald* $\chi^2 = 16.56$, $p < .001$), and Lhwa within the family ($B = 0.02$, $SE = 0.01$, *Wald* $\chi^2 = 4.26$, $p = .039$) significantly predicted IED diagnosis. Lhwa outside-family was not significantly associated with IED controlling for other variables in the model. Lhea and Lhwa variables were significantly intercorrelated (*p*s < .001); however, associations were within an acceptable range for simultaneous inclusion in the model (i.e., not multicollinear; range $r = .41$ to $.75$) Exposure to aggression across the three life stages was too highly correlated to examine in a regression model (*r*s = .67-.87, *p*s < .001). However, separate logistic regression analyses showed that exposure at each stage was associated with IED diagnosis (all *p*s < .001).

Table 3. Main Effects and Interactions on Lhwa.

Effect	*F*	*df*	*p*	Contrast	Main Effect	Interaction	*M* (*SE*s)
Gender	9.02	1,579	.003	M > W	M		9.42 (0.30)
					W		8.20 (0.27)
Stage	41.2	21,158	<.001	Ado > Chi > Adu	Chi		8.65 (0.23)
					Ado		9.54 (0.23)
					Adu		8.25 (0.20)
Context	47.2	11,158	<.001	In < Out	In		7.98 (0.24)
					Out		9.64 (0.23)
Gender × stage	12.0	21,158	<.001	M > W	Chi	M	9.51 (0.35)
						W	7.80 (0.31)
				M > W	Ado	M	10.31 (0.33)
						W	8.76 (0.30)
				M = W	Adu	M	8.45 (0.30)
						W	8.04 (0.27)
Gender × context	34.4	11,158	<.001	M = W	In	M	7.89 (0.36)
						W	8.08 (0.32)
				M > W	Out	M	10.96 (0.35)
						W	8.32 (0.31)

(*continued*)

Table 3. continued

Effect	*F*	*df*	*p*	Contrast	Main Effect	Interaction	*M* (*SE*s)
Stage × context	281.2	21,158	<.001	In > Out	Chi	In	9.08 (0.28)
						Out	8.22 (0.27)
				Out > In	Ado	In	8.88 (0.27)
						Out	10.19 (0.25)
				Out > In	Adu	In	5.98 (0.24)
						Out	10.52 (0.25)

Note. n = 582; M = men; W = women; Chi = childhood, Ado = adolescence; Adu = adulthood.

Gender Differences

Comorbidity

Women showed a higher rate of current and lifetime mood and anxiety disorders than men. Current trauma-related disorder was three times more prevalent in women compared to men (15.4% vs 5.8%; $p < .001$), and lifetime trauma-related disorder was twice as prevalent (24.4% vs 10.5%; $p < .001$). Men showed a higher rate of lifetime substance use disorder, conduct disorder, and oppositional defiant disorder. Men and women did not differ in comorbid personality disorder diagnoses, including Cluster B diagnoses (men: 33.7%; women: 38.9%; ps > .199).

Study variables

Few significant gender differences were observed in the primary variables of interest (Table 1). Women reported modestly more negative emotional reactivity (AIM) than men ($d = 0.40$). Otherwise, men and women had similar scores on measures of emotional dysregulation and SIP.

Potential Mediators: Bivariate Correlations

Bivariate correlations between potential mediator variables, EIV variables, and the LHA were examined to select variables for mediation analysis (Table 4). In the full sample, the ALS total and AIM negative intensity measures of emotion dysregulation correlated moderately with lifetime exposure to aggression and moderately-to-strongly with life history of aggression. Bivariate correlations were also assessed separately for men and women. AIM negative reactivity correlated with LHA in men ($r = .12$, $p = .044$) but not women ($r = .07$, $p = .201$). In men but not women, positive evaluations of

aggressive behavior (i.e., SIP-RED) correlated with aggressive behavior (men: $r = .17$, $p = .031$; women: $r = .04$, $p = .635$). Based on the robust correlations between ALS, Lhea and Lhwa scales, and aggression in both genders, and the gender difference in SIP-RED correlation, we included these two variables as potential mediators in subsequent path models.

Effect of Gender on Mediation Models of Exposure to Aggression Predicting IED

Experienced aggression within the family

Separate PROCESS Models (Model 59) were used to evaluate the effect of gender on direct and indirect effects and individual pathways of EIV. Covariates (age, race, SES) were included in the model. Unstandardized coefficients (B) and standard errors (SE) are reported. Bootstrapping (5,000 samples) was used to estimate model coefficients. Lifetime history of experienced aggression-in ($B = 0.22$, $SE = 0.05$, $p < .001$) and SES ($B = –0.01$, $SE = 0.00$, $p = .016$) predicted ALS ($F[6,339] = 6.36$, $p < .001$, $R^2 = .10$). The variables in the model did not significantly predict SIP-RED. The overall model including Lhea within-family, ALS, and SIP-RED significantly predicted IED diagnosis ($n = 346$; $–2LL = 324.2$, $df = 10$, $p < .001$, *Nagelkerke*

Table 4. Bivariate Correlations.

Measure	ALS Total[a]	AIM Neg Int[a]	AIM Neg React[a]	SEIPQ HA[b]	SEIPQ NER[b]	SEIPQ RED[b]	LHA[c]
Lhea in	.34**	.31**	.12*	.01	.00	–.01	.55**
Lhea out	.30**	.24**	.00	.04	.06	.01	.60**
Lhwa in	.24**	.24**	.06	.08	.10	.08	.46**
Lhwa out	.26**	.17**	-.05	.05	.07	.08	.45**
LHA	.50**	.42**	.08*	.15**	.12*	.10	–

Note. Pearson's correlations.

*Correlation is significant at the 0.05 level.

**Correlation is significant at the 0.01 level.

[a]$n = 475$.

[b]$n = 353$.

[c]$n = 346$.

R^2 = .47). Age (B = 0.04, SE = 0.01, p = .013) and race (B = 0.61, SE = 0.29, p = .034) significantly predicted IED, as did LHEA-in (B = 1.22, SE = .0.18, p < .001) and ALS (B = 0.87, SE = 0.16, p < .001) but not SIP-RED (B = 0.09, SE = 0.14, p = .556). Both the direct effect of Lhea on IED and indirect effect through ALS were significant. There was a trend of gender moderating the direct effect of LHEA-in on IED (χ^2 = 3.58, df = 1, p = .059). LHEA-in predicted IED in both men (B = 0.88, SE = 0.22, p < .001, *95% CI*: 0.46-1.31) and women (B = 1.55, SE = 0.28, p < .0001, *95% CI*: 0.99-2.10), but the effect was stronger in women. Gender did not moderate the relationship between ALS or SIP-RED and IED. The indirect effect of LHEA-in on IED through ALS was significant in both men (B = 0.14, SE = 0.08, *95% CI*: 0.02-0.32) and women (B = 0.24, SE = 0.10, *95% CI*: 0.09-0.48), and gender did not moderate the strength of this relationship.

Experienced aggression outside the family

LHEA-out (B = 0.19, SE = 0.05, p = .004), gender (B = 0.24, SE = 0.10, p = .0233), and SES (B = –0.01, SE = 0.00, p = .011) predicted ALS (F[6,341] = 5.65, p < .001, R^2 = .09). The variables in the model did not significantly predict SIP-RED. The overall model predicting IED was significant (n = 348; *–2LL* = 327.33, df = 10, p < .001, *Nagelkerke* R^2 = .46). LHEA-out (B =1.28, SE = 0.20, p < .001), ALS (B = 0.86, SE = 0.16, p < .001), age (B = 0.04, SE = 0.01, p = .012), and gender (B = 0.66, SE = 0.33, p = .044), predicted IED. In addition, gender significantly moderated the relationship between LHEA-out and IED (χ^2 = 5.69, df = 1, p = .017). The effect of LHEA-out on IED was significant in men (B = 0.81, SE = 0.21, p = .001, *95% CI*: 0.41-1.22) and women (B = 1.73, SE = 0.35, p < .001, *95% CI*: 1.05-2.42), but was stronger in women. The relationship between LHEA-out and IED was mediated by ALS in women (B = 0.21, SE = 0.10, 95% CI = 0.07 to 0.45) and men (B = 0.12, SE = 0.08, *95% CI*: –0.00 to 0.32). The indirect effect sizes did not differ significantly by gender.

Witnessed aggression inside the family

The overall model predicting IED was significant (n = 349; *–2LL* = 333.74, df = 10, p < .001, *Nagelkerke* R^2 = .45). Age (B = 0.01, SE = 0.01, p = .030) and SES (B = –0.01, SE = 0.00, p = .009) significantly predicted ALS. Gender did not moderate the relationship between Lhwa-in and ALS (B = –0.02, SE = 0.11, p = .842). The variables in the model did not significantly predict SIP-RED. In the overall model predicting IED, Lhwa-in (B = 1.11, SE = 0.17, p < .001), ALS (B = 1.08, SE = 0.17, p < .001), age (B = 0.04, SE = 0.01, p =.008), and race (B = 0.60, SE = 0.29, p = .037) significantly predicted IED. In addition, gender moderated the relationship between witnessing aggression in the

family and IED ($\chi^2 = 4.66$, $df = 1$, $p = .031$). Gender did not moderate the relationships between ALS or SIP-RED and IED. The direct effect of Lhwa-in on IED was significant in both men ($B = 0.74$, $SE = 0.21$, $p < .001$, *95% CI*: 0.32–1.15) and women ($B = 1.48$, $SE = 0.28$, $p < .001$, *95% CI*: 0.92–2.04). However, ALS did not significantly mediate the relationship between Lhwa-in and IED in either men or women.

Discussion

In a large sample of adults, we found evidence that EIV both within and outside the family predicts IED diagnosis. In addition, we found that EIV was a stronger risk factor for IED in women than in men. Finally, we found little evidence for differences in pathways mediating exposure to aggression and IED diagnosis in men and women.

In separate models, we examined effects of experiencing aggression within and outside the family and witnessing aggression within the family on IED diagnosis. EIV was a risk factor for IED in both men and women, which is consistent with previous research linking violence exposures and aggressive behavior (Dodge et al., 1990; Fanning et al., 2014; Farrell et al., 2020). In all three models we found evidence that EIV is a stronger risk factor for recurrent problems with aggression in women compared to men. Thus, although men and women are exposed to similar amounts of aggression in the family and men are exposed to more aggression outside the family, the impact of exposure on recurrent aggression appears to be greater in women. Previous research finds higher conditional probabilities of PTSD in women who experience violence compared to men (Breslau, 2009). As such, the current findings accord with prior findings that women experience greater risk of PTSD in general and specifically related to interpersonal violence. Although we did not evaluate PTSD symptoms directly, PTSD diagnosis is characterized by negative affective states and increased reactivity to perceived threats, consistent with our focus on affective dysregulation. However, we did not observe gender differences in affective dysregulation. As such, further research is needed to understand the factors contributing to increased risk in women.

We included ALS and SIP-RED in our moderated mediation models based on our preliminary data reduction strategy. These two mediators potentially reflect trauma-related changes in affect regulation and social learning effects, both of which we predicted might vary by gender. We found that aggressive behavior is significantly correlated with SIP-RED in men but not women. As such, men with more positive evaluations of aggression also reported more extensive life history of aggression. However, SIP variables (hostile attribution, NER, and response evaluation) were unrelated to interpersonal violence

victimization (EIV). Previous studies have linked SIP biases in adulthood with childhood victimization (e.g., Berlin et al., 2011; Pettit et al., 2010; Taft et al., 2008). However, other studies have found no association between SIP biases and violence exposure or small significant associations (Chen et al., 2012; Taft et al., 2008). Some earlier positive findings examined exposure to more severe forms of violence (i.e., physical violence) compared to the Lhea and Lhwa measures, which assess both verbal and physical aggression victimization. There is evidence that severe (compared to less severe) physical abuse is more strongly associated with SIP biases (Shahinfar et al., 2001). Taken together, this evidence suggests that the time since victimization and severity of victimization may affect the extent of SIP biases and therefore the impact of SIP on aggressive behavior in adulthood. Thus, although SIP variables showed a small, significant correlation with aggression in men, our findings do not support the notion that SIP biases explain the association between aggressive victimization, defined broadly as exposure to psychological, verbal, and physical aggression, and IED diagnosis.

In contrast, measures of emotion regulation correlated robustly with interpersonal violence victimization. Affective lability correlated moderately with victimization and strongly with aggression in both men and women. Affective lability constituted a significant indirect effect in models of the relationship between *experiencing* aggression (within and outside the family) and IED diagnosis. An indirect effect of ALS was not observed for witnessing aggression inside the family. Affective lability was associated with IED in all three path models but was associated with violence exposure only in the case of experiencing violence. Previous research has found that experiencing abuse within the family of origin was associated with increased risk of PTSD in adulthood, but that witnessing family violence only was not (Kulkarni et al., 2011). The relationship between experiencing interpersonal violence and affective lability is also consistent with differing conditional probabilities of PTSD related to different types of trauma and the tendency for *experienced* traumatic events to be associated with more persistent PTSD symptoms compared to events that are witnessed or learned of (Breslau, 2009). Our results indicate that experiencing aggression either within or outside of the family predicts IED and that this association is likely to be partially transmitted through disrupted affect regulation.

We found that the indirect effect of experiencing aggression outside of the family on IED through affective lability was significant in both women and men. Although the effect sizes did not differ from each other, the association was twice as strong in women. As indicated by behavioral genetics studies, individuals who experience violence within the family are at increased risk for problematic aggression due to both heritable and environmental factors.

However, increased risk related to heritable factors would be absent in cases where the violence perpetrator was not a family member, allowing environmental effects to be observed unconfounded. Thus, our finding that affect dysregulation statistically mediates the relationship between interpersonal violence experienced outside of the family and IED diagnosis supports the notion that changes in affect regulation related to EIV contributes to the development of IED. Because we focus on IED diagnosis rather than a continuous measure of trait aggression or aggressive acts, our results indicated that interpersonal violence exposure increases the risk for chronic, clinically significant aggressive behavior. Finally, we found significant evidence that gender moderated the direct effect of EIV on aggression independently of affective regulation. Accordingly, further research is required to understand the factors that explain this gender difference in risk.

The present findings have implications for clinical practice and intervention. First, the findings are relevant to conceptualizing and treating aggressive behavior in women, compared to men. Our findings suggest that exposure to violence may be a stronger risk factor for aggression in women compared to men. Future research should examine whether comorbid trauma-related disorders such as PTSD contribute more significantly to aggressive behavior in women. There is evidence that sex differences in stress neurobiology contribute to the higher observed prevalence of PTSD and depression in women, and these neural systems overlap with those implicated in aggressive behavior (Bangasser & Valentino, 2014). Further research is needed to understand how neurobiological systems contribute to aggressive behavior in men and women. Such information could inform future research into pharmacological interventions for aggression. Relatedly, potential sex differences in pharmacologic interventions for aggression are an important target for investigation. The current study contributes to a growing literature on the role of trauma in IED. The significant role of trauma exposure in clinically significant aggressive behavior suggests trauma-informed interventions may be well-suited for this clinical group. In recent years, trauma-informed psychotherapy interventions for aggression, including intimate partner violence, have been developed, and these interventions show promising results in initial evaluations (Hewage et al., 2018; Taft et al., 2016).

The current study has limitations worth considering. First, the measures of experiencing and witnessing aggression assess exposure to discrete instances of aggressive behavior. They do not assess the duration of exposure to a threatening or dangerous environment. Accordingly, they do not parse acute versus chronic exposure to violence. The measures further do not assess the specific age of exposure, but rather capture cumulative exposure within broad age ranges. Second, although participants were characterized clinically with

respect to diagnoses including PTSD, we did not include a continuous measure of PTSD symptom severity in our analyses. However, observed rates of trauma- and stressor-related disorder in our sample show a gender difference that is consistent with the previous literature. Personality disorders were prevalent in the current sample. However, we observed no gender differences in the rate of Cluster B personality disorders, which include borderline and antisocial personality disorders. We also observed no gender difference in the affect dysregulation measure we examined as a potential mediator, the ALS, consistent with prior research. Future research is needed to examine the role of PTSD symptoms in gender differences in risk for aggression. Third, although we test a series of moderated mediation models, true causal mediation requires evidence of the temporal ordering of variables, which we do not have in this cross-sectional study. Research suggests that various transactional and bidirectional processes contribute to aggressive behavior, including coercive parent-child interactions and passive, active, and evocative gene-environment correlations (Burt, 2009b; Pettit & Arsiwall, 2008). It was not possible given the study design to evaluate these transactional effects; however, though novel, our findings are consistent with a well-replicated literature on gender and trauma and provide evidence for a plausible mechanism for the development of chronic aggression. A further potential limitation is that it was not possible to study the effects of specific types of EIV in this study. However, experienced aggression within the family during childhood (LHEA) likely reflects childhood maltreatment and/or sibling aggression, whereas experienced aggression within the family during adulthood likely reflects intimate partner violence. Witnessing aggression within the family likely captures observing family violence. Experiencing aggression outside of the family may reflect the broadest ranging of EIV, encompassing community violence, school violence, and dating and sexual violence. Further research assessing specific types of violence is needed to replicate and extend these findings. Furthermore, relevant mediators may differ depending on the type of violence exposure.

In a large, clinically relevant sample of adult men and women, we observed that experiencing aggression both within and outside the family, and witnessing aggression in the family, uniquely predicted chronic and clinically significant aggressive behavior. These relationships were stronger in women than in men. Affective dysregulation partially mediated EIV and IED diagnosis.

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