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Exposure to Interpersonal Violence and Risk for PTSD, Depression, Delinquency, and Binge Drinking Among Adolescents: Data From the NSA-R

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Abstract

Interpersonal violence (IPV) is associated with a range of subsequent negative outcomes; however, research has yet to test whether IPV operates as a specific risk factor for separate psychopathology outcomes, such as posttraumatic stress disorder (PTSD) symptoms, depressive symptoms, delinquent acts, or binge drinking. To address this, cumulative exposure to IPV and non-IPV-related traumatic events, PTSD symptoms, depressive symptoms, delinquent acts, and binge drinking were measured 3 times over approximately 3 years among a nationally representative sample of adolescents aged 12–17 (N = 3,614 at Wave 1). Results demonstrated that cumulative IPV exposure predicted subsequent PTSD, depression, delinquency, and binge drinking (βs = .07, .12, .10, and .09, respectively; all ps < .01) when all cross-relationships (e.g., the effect of delinquency on future binge drinking) were in the model. Exposure to non-IPV traumatic events generally did not confer vulnerability to subsequent psychopathology outcomes. Overall, findings from this study advance the literature in this area by exploring consequences for adolescents following cumulative IPV exposure.

Interpersonal violence (IPV), including sexual assault, physical assault, and witnessed violence (Hedtke et al., 2008; Kilpatrick et al., 2000; Resnick et al., 1993) is an important public health concern, as epidemiological studies suggest that exposure to IPV is extremely common among adolescents, with 21–80% of adolescents reporting exposure to some form of IPV (see, Finkelhor, Ormrod, & Turner, 2007c; Kilpatrick et al., 2000). This is problematic, given that exposure to IPV among adolescents is linked with a range of psychopathology outcomes, including posttraumatic stress disorder (PTSD), depression, substance use, and delinquency (Fergusson & Horwood, 1999; Kilpatrick et al., 2000, 2003. For example, Kilpatrick and colleagues (2000, 2003) found that exposure to IPV increased risk of PTSD, depression, and substance abuse, as well as comorbidity between these

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outcomes, in a U.S. sociodemographically representative cross-sectional sample of adolescents.

Although empirical evidence suggests that cumulative IPV exposure confers vulnerability for subsequent psychopathology outcomes, two major limitations persist. First, research has not controlled for cross-sectional relationships while linking IPV exposure with psychopathology outcomes. As prior research indicates correlations among depression, delinquency, and binge drinking regardless of IPV exposure (Bukstein, Glancy, & Kaminer, 1992; Fergusson, Lynskey, & Horwood, 1996; Marmorstein, Iacono, & Malone, 2010; Marti, Stice, & Springer, 2010; Wiesner & Kim, 2006; Wolff & Ollendick, 2006), the significant relationships may be mediated by the effect of IPV on the other outcomes. For example, whether IPV exposure predicts increases in problematic drinking after delinquency is included in the model is unclear. Given the link between IPV exposure and delinquency (McCart et al., 2007), and the link between delinquency and problematic drinking (e.g., binge drinking; Nation & Heflinger, 2006), a viable alternative explanation is that delinquency significantly contributes to these associations. Similarly, depression has been theorized to increase risk for delinquency (Wolff & Ollendick, 2006) and problematic drinking (Bukstein et al., 1992), which should be taken into account when examining these relationships.

Second, research has not consistently examined the effect of cumulative IPV exposure, although research suggests that multiple exposures to IPV is the norm, rather than the exception, among adolescents. Turner, Finkelhor, and Ormrod (2010) surveyed a nationally representative sample of 4,053 children aged 2–17 and found that 80% experienced at least one victimization, 66% were exposed to two or more, 30% were exposed to five or more, and 10% were exposed to 11 or more (also see, Finkelhor, Ormrod, & Turner, 2007a). Accounting for cumulative exposure to IPV, as opposed to a dichotomous exposed versus unexposed variable, is important because research has found that cumulative IPV exposure increased risk for PTSD, depression, substance use, and delinquency among adolescents (Finkelhor et al., 2007a; Finkelhor, Ormrod, & Turner, 2007b) and adults (Hedtke et al., 2008). These findings correspond well with other research demonstrating that risk of PTSD and depression increases with the frequency of lifetime exposure to traumatic and/or stressful events (Neuner et al., 2004).

In summary, numerous lines of research suggest that IPV exposure increases risk for subsequent PTSD, depression, delinquency, and problematic drinking, but this research has yet to address (a) whether IPV exposure confers risk for subsequent psychopathology when also including cross-relationships between psychopathology outcomes in the model, and (b) the specific relationship between cumulative IPV exposure and subsequent risk for psychopathology outcomes. The current study aims to fill these important gaps in the literature by examining the effect of IPV on risk for subsequent psychopathology (i.e., PTSD, depression, delinquency, problematic drinking), in the presence of the effects that these variables have on one another.

Method

Participants

The National Survey of Adolescents-Replication (NSA-R) is a longitudinal epidemiological study of adolescents ages 12–17 (*N* = 3,614 at Wave 1; 1,808 girls) residing in the United States. For detailed descriptions of the sampling and methodological procedures, refer to Wolitzky-Taylor et al. (2010). Data collection occurred between 2005 and 2010. The NSA-R sample consisted of a national household probability sample as well as an oversample of urban-dwelling youth (self-reported). During recruitment, 6,694 households were contacted in which parents completed a brief structured interview and were asked to identify at least one eligible adolescent. Of these, 1,268 (18.9%) parents refused adolescent participation; 188 (2.8%) adolescents refused to be interviewed following parental consent; 119 (1.8%) adolescent interviews were initiated, but not completed; and 1,505 (22.5%) identified eligible adolescents were unreachable or not available for interview. Thus, 3,614 cases resulted in complete adolescent interviews at Wave 1, including 2,459 in the national cross-section and 1,155 urban-dwelling adolescents. Mean age was 14.63 (*SD* = 2.70) at Wave 1. With regard to the racial/ethnic makeup of the sample, 65% were Caucasian, 15% were African American, 11% were Hispanic, 2% were Native American, 3% were Asian/Pacific Islander, and 3% described their racial/ethnic status as Other. Annual household income of the sample was $46,093 (*SD* = $8,775).

Measures

Interpersonal violence exposure and non-IPV traumatic event exposure—

Interpersonal violence exposure was assessed by self-reported behaviorally specific dichotomous questions (yes or no) regarding a series of events: (a) sexual assault (i.e., anal penetration, vaginal penetration, oral sex on the perpetrator, oral sex from the perpetrator, digital penetration, fondling of the adolescent, forced fondling of the perpetrator, or drug- or alcohol-facilitated sexual assault), (b) physical assault (i.e., attacked with a weapon; attacked with a stick, club, or bottle; attacked without a weapon; threatened with a weapon; attacked with fists), (c) abuse from a caregiver (i.e., spanked, slapped, pushed, locked in a closet, thrown against a hard surface, beaten up with fists or kicked, choked, burned on purpose, cut with sharp object, or threatened with a weapon), (d) witnessed community violence (i.e., witnessed shooting, witnessed stabbing, witnessed sexual assault, witnessed robbery, witnessed threatening with a weapon, witnessed hitting or kicking), and (e) witnessed domestic violence (i.e., witnessing pushing or shoving, punching or hitting, choking, hitting with objects, or threatening with weapon). Non-IPV traumatic event exposure was assessed by specific questions regarding motor vehicle accidents, other accidents, fires, natural disasters, and being bitten by dog. Specific wording of questions and details of this methodology are available in past publications (Kilpatrick et al., 2000, 2003). Consistent with the recently proposed criteria for the upcoming edition of the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*; Friedman, Resick, Bryant, & Brewin, 2011), a potentially traumatic event (PTE) was considered endorsement of any of the events regardless of whether the individual experienced fear, helplessness, or horror.

At Wave 1 (baseline), participants with exposure were asked how many times the event had ever happened to them. Two PTE variables (which included IPV and non-IPV) were created: (a) cumulative assaultive PTE history (sum of the number of sexual assault, physical assault, care-giver abuse, witnessed community violence, and witnessed domestic violence events that occurred), and (b) cumulative nonassaultive PTE history (sum of the other reported PTE events, e.g., accidents, natural disasters). These PTE variables were computed only for Wave 1.

Depression—Major depressive symptoms were assessed with self-report (yes or no) using criteria from the major depressive episode criteria according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., *DSM-IV*; American Psychiatric Association, 1994) over the past year. Specifically, adolescents were asked 13 questions about the presence of *DSM-IV* symptoms of depression, with thoughts of death and suicide separated into two items. Total depressive symptoms endorsed over the past year were summed for each wave in the present study. Internal consistency of the depression responses in the current study was, $\alpha = .88$ at Wave 1.

Delinquency—Delinquency was assessed using an adapted version of the National Youth Survey (Elliott, Huizinga, & Ageton, 1985), which asked adolescents if they had engaged in specific delinquent behaviors over the past year (yes or no). These delinquent behaviors included physical assault, selling drugs, burglary, motor vehicle theft, robbery, used force to obtain money or things from others, attacked someone with a weapon, attacked someone with intent to seriously hurt or injure, been arrested, or been to jail/juvenile detention. Total delinquent acts were summed for each wave in the present study, which had an internal consistency of $\alpha = .48$ at Wave 1. The low consistency of this measure is likely due to the fact that the various types of delinquent acts are not necessarily correlated (e.g., children who have been arrested do not necessarily sell drugs).

Binge drinking frequency—Problematic alcohol use frequency was assessed by asking adolescents how many days in the past 12 months they had consumed five or more alcoholic drinks (i.e., binge drink frequency; Courtney & Polich, 2009). Binge drinking was categorized as five or more drinks per drinking day following the Center for Disease Control Behavioral Risk Factor Surveillance System standard question/definition “Considering all types of alcoholic beverages, how many times during the past 30 days did you have five or more drinks on an occasion?” (see Stahre, Naimi, Brewer, & Holt, 2006).

PTSD symptoms—Current PTSD symptoms were assessed using the PTSD module of the NSA survey (Kilpatrick et al., 2000) and National Women Survey (Resnick et al., 1993), a structured diagnostic interview that assessed each *DSM-IV* symptom with a yes or no response indicating the presence of a symptom during the last 6 months. Symptoms endorsed were added to create a total sum of PTSD symptoms. Alpha in the present study was .80. The symptoms were not anchored to any specific index traumatic event. This measure demonstrates good concurrent validity and several forms of reliability (e.g., temporal stability, internal consistency, diagnostic reliability; Resnick et al., 1993; Ruggiero et al., 2006). The measure was validated against the PTSD module of the Structured Clinical

Interview for the *DSM* (SCID; Spitzer, Williams, & Gibbon, 1987) administered by mental health professionals Kilpatrick et al., 1998). The interrater kappa coefficient was .85 for the diagnosis of PTSD.

Procedure

Participant selection utilized a multistage, stratified, random-digit dial procedure within each region of the country. Trained interviewers at a survey research firm with significant experience managing survey studies administered the interview using a computer-assisted telephone interview system.

Participant protection—To protect the NSA-R participants, measures were developed to identify adolescents in potentially dangerous situations. Specifically, adolescents indicated if he or she (a) had been sexually abused in the past year, (b) had been hit or physically assaulted by a family member living in the household in the past year, or (c) had not disclosed the sexual or physical assault to anyone. If an adolescent answered yes to any of these questions, a clinician on the project team interviewed the adolescent.

Mean number of months between Wave 1 and Wave 2 assessment was 15.29 (*SD* = 4.58), between Wave 2 and Wave 3 assessment was 14.44 (*SD* = 2.67), and between Wave 1 and Wave 3 assessment was 29.02 (*SD* = 4.51). Because adolescents were oversampled in urban areas, cases were weighted to maximize representativeness of the sample to the 2005 U.S. adolescent population. A weight was created to restore the urban cases to their true proportion of the urban/suburban/rural variable, based on 2005 U.S. Census estimates. Next, weights were created to adjust the weight of each case based on age and sex based on 2005 U.S. Census estimates. Details of this weighting procedure have been provided elsewhere (Wolitky-Taylor et al., 2008).

Analytic Approach

First, preliminary analyses examined whether there were differences between participants who completed the baseline assessment and those who did not. Specifically, we examined differences in sociodemographic characteristics, including people living in household, children living in household, age of children in household, parent gender, parent age, parent marital status, parent income level, parent education level, parent employment status, parent race, adolescent gender, adolescent age, adolescent race, rural versus urban setting, parental concern with violent crime and child abuse, parent prevention of sexual assault and substance use, and school program for interpersonal violence and substance use. Details of this weighting procedure have been provided elsewhere (Wolitky-Taylor et al., 2008).

Descriptive statistics are first provided in Table 1. Hypotheses were tested with multiple regression analyses, in which Wave 1 characteristics (ethnicity, sex, age, IPV exposure, non-IPV exposure, PTSD symptoms, depression symptoms, delinquent acts, and binge drinking frequency) were simultaneously entered as predictors of the dependent variable. Separate regression analyses were conducted using Wave 2 (or 3) PTSD symptoms, Wave 2 (or 3) depression symptoms, Wave 2 (or 3) delinquent acts, and Wave 2 (or 3) binge drinking frequency as the dependent measures (see Tables 2 and 3). Multiple regression analyses

were chosen because entering all Wave 1 predictors simultaneously allowed us to test whether IPV continues to predict subsequent psychopathology (e.g., depression) while also including cross-relationships (e.g., the effect of delinquency on depression) in the model. To address the problem of attrition (see below), each regression analysis was completed twice: once using the original data (i.e., only completers), and once after imputing all missing values using multiple imputation with regression (using all study variables at Waves 1, 2, and 3 as predictor variables in the imputation models). As is shown in Tables 2 and 3, results varied little between analyses conducted on original and imputed data.

Results

Preliminary Analyses

Nonresponse—Of the 6,928 adolescents contacted to complete the assessment, 3,614 were measured at Wave 1 (52% response rate). Adolescents who refused to complete the baseline assessment significantly differed from those who completed the assessment on a number of factors for which survey response options varied from three to eight. One difference was that noncompleters reported a higher number of people living in the household, $F(7, 6920) = 2.22$, $p = .03$, where the response options for how many people lived in the household including the parent were *2–7*, *not sure*, and *refused*. The remaining were a higher number of children living in the household, $F(7, 6920) = 13.49$, $p = .01$; older age of children in the household, $F(7, 6748) = 3.01$, $p = .01$; more females, $F(2, 6925) = 6.31$, $p = .01$; more single parents, $F(6, 6921) = 3.70$, $p = .01$; more parents of Caucasian race, $F(7, 6920) = 2.63$, $p = .01$; more rural versus urban setting, $F(2, 6921) = 2.38$, $p = .03$; parents who were less concerned with violent crime, $F(5, 6922) = 2.31$, $p = .04$; parents who had not talked with the adolescent about how to avoid being molested or sexually abused, $F(2, 6925) = 3.41$, $p = .03$; parents who had not talked with the adolescent about how to avoid using alcohol and drugs, $F(3, 6924) = 4.96$, $p = .01$; and that a program had not been offered at the adolescent's school about alcohol or drug use, $F(3, 6924) = 3.14$, $p = .02$. Adolescents did not differ on any other sociodemographic measures; information on depression symptoms, PTSD symptoms, delinquent acts, and binge drinking were not collected from the parents at baseline.

Descriptive statistics—Cumulative IPV exposure, cumulative non-IPV PTE exposure, PTSD symptoms, and depression symptoms at each wave demonstrated acceptable levels of skewness (<2.31) and kurtosis (<7.0; cf. Kline, 2005). However, binge drinking frequency and delinquency were significantly skewed and kurtotic. Accordingly, both variables were log transformed, which improved both skewness (<3.9) and kurtosis (<11.0).

Table 1 provides descriptive information and the prevalence rates of the study variables. The prevalence of IPV exposure was 50%, 54%, and 56% at Waves 1, 2 and 3, respectively. The prevalence of non-IPV PTE exposure was 44%, 47%, and 47%. The prevalence of at least one delinquent act in the past year was 13%, 10%, and 10%. The prevalence of at least one depression symptom was 42%, 36%, and 34%. The prevalence of at least one binge-drinking episode in the past year was 13%, 20%, and 28%. The prevalence of at least one PTSD symptom in the past year was 43%, 42%, and 41%.

Attrition—Of the 3,614 adolescents measured at Wave 1, 2,511 were measured at Wave 2, and 1.653 were measured at Wave 3. Adolescents who completed all three waves (i.e., those who were not missing data from Waves 1, 2, or 3) had significantly lower Wave 1 depression symptoms (M = 1.08, SD = 1.93) compared to adolescents who did not complete each wave (M = 1.23, SD = 2.08), $F(1, 3613) = 4.63$, $p = .032$, $\eta_p^2 = .001$. Similarly, adolescents who completed all three waves had significantly fewer IPV exposures at Wave 1 (M = 1.21, SD = 1.85) compared to adolescents who did not complete each wave (M = 1.59, SD = 2.17), $F(1, 3613) = 31.98$, $p < .001$, $\eta_p^2 = .009$. Adolescents who completed all three waves also had significantly less non-IPV event exposures at Wave 1 (M = 0.80, SD = 1.22) compared to adolescents who did not complete each wave (M = 0.95, SD = 1.34), $F(1, 3613) = 12.16$, $p < .001$, $\eta_p^2 = .003$. Adolescents who completed all three waves also had significantly less delinquent acts at Wave 1 (M = 0.14, SD = 0.50) compared to adolescents who did not complete each wave (M = 0.25, SD = 0.70), $F(1, 3613) = 30.73$, $p < .001$, $\eta_p^2 = .008$ (analysis conducted on log transformed variable). Adolescents who completed all three waves (M = 1.61, SD = 9.03) did not significantly differ from adolescents who did not complete each wave (M = 1.74, SD = 12.31); $F(1, 3613) = 1.90$, $p = .17$, $\eta_p^2 = .001$ (analysis conducted log-transformed variables) in binge drinking at Wave 1. Completers did not differ from noncompleters in gender, $\chi^2(1, N = 3614) = .02$, $p = .89$, but participants belonging to a minority race or ethnicity were more likely to not complete all three waves, χ^2 (1, N = 3614) = 56.7, $p < .001$.

Regression Analyses

Tables 2 and 3 display summaries of the primary analyses. As can be seen, the number of Wave 1 IPV exposures significantly predicted depression symptoms, PTSD symptoms, and delinquent acts at Wave 2, but not binge drinking at Wave 2, with all cross-relationships in the model. Wave 1 IPV exposures significantly predicted Wave 3 depression symptoms, PTSD symptoms, delinquent acts, and binge drinking frequency with all cross-relationships in the model. Non-IPV PTEs only predicted Wave 2 and Wave 3 PTSD symptoms and no other psychopathology outcomes. As can be seen in the tables, results of analyses on the imputed data generally corresponded closely to analyses on the original data (i.e., particularly the β coefficient estimates of effect size), with the expected exception that the p values tended to be lower using the imputed data given the larger sample sizes in the imputed data.

Discussion

When including all cross-relationships between psychopathology outcomes in the model, cumulative IPV exposure continued to independently confer prospective vulnerability for PTSD symptoms, depression, delinquency, and binge drinking. These data suggest that cumulative IPV exposure may operate as a generalized risk factor for the subsequent development of depression, binge drinking, PTSD symptoms, and delinquency. By contrast, non-IPV related PTE was only weakly prospectively related to PTSD symptoms and did not predict any of the other psychopathology outcomes when the model included IPV-related

PTEs. These data generally echo other research suggesting that IPV events are a more potent risk factor relative to other types of PTEs (Kessler, 2000; Resnick et al., 1993).

One interesting and unexpected finding was that Wave 1 cumulative IPV exposure was not robustly related to Wave 2 binge drinking frequency; in fact, the analyses on imputed data showed a *negative* relationship, whereas Wave 1 cumulative IPV exposure was positively related to all other psychopathology outcomes at Waves 2 and 3. The current study cannot address specifically why Wave 1 cumulative IPV exposure was related to Wave 3 binge drinking, but not Wave 2 binge drinking. One possibility that could be explored with future research is that this delayed effect on drinking behavior develops only following initial unsuccessful attempts to cope with negative affect subsequent to IPV exposure. However, if this was the case then it might be expected that Wave 1 depression or PTSD symptoms would have predicted Wave 2 or Wave 3 binge drinking, which was not observed in these data. Another possibility may be that increased negative affect initially increases social withdrawal, which has a consequence of decreasing access to social events involving binge drinking. However, if this were the case then it might also be expected that delinquent acts might also have shown a delayed effect, such that social withdrawal would presumably also decrease access to deviant peer groups and behavior, but Wave 2 and Wave 3 delinquent acts increased as a function of Wave 1 cumulative IPV exposure. Alternatively, developmental processes and altered social context (e.g., going to college) could also potentially explain the finding, as binge drinking does tend to increase in later adolescence. Future research is necessary to replicate the current observation of a delayed effect and examine possible mediators and moderators of this finding.

Overall, the present findings provide strong evidence that cumulative IPV exposure confers risk for subsequent PTSD symptoms, depression, delinquency, and binge drinking. There is a rich theoretical and empirical literature that suggests that exposure to PTEs is associated with a host of negative outcomes, including the phenotypes included in the present study.

Additionally, data suggest that exposure to PTEs is also related to disruption in the broad areas of emotional, cognitive, personality, and relational functioning (Cicchetti, Rogosch, Howe, & Toth, 2010; Valentino, Cicchetti, Rogosch, & Toth, 2008). There are numerous theories regarding how exposure to traumatic stress becomes associated with posttrauma phenotypes. For example, it has been posited that PTE exposure alters cognitive appraisals, leading youth to overexaggerate the emotionality of situations and therefore leading to a behavioral reaction consistent with behavioral dysregulation (Rossman & Ho, 2000). Additionally, the data could also be interpreted in light of the notion of complex PTSD, whereby it is hypothesized that exposure to multiple forms of PTEs is associated with a greater number of symptoms that are presented simultaneously (Cook et al., 2005).

This study focused on prevalence and associations between IPV exposure and risk for subsequent detrimental consequences and did not focus on treatment or recovery of victims over time. Thus, we are unaware of each individual’s process of recovery, social support networks available, or services that were sought out. Second, a large amount of attrition did not occur at random, and there was differential agreement to participate in the study based on family characteristics. It cannot be certain how the attrition or differential response

affects the present results, thus caution is warranted when interpreting the results and future replication is necessary. Third, cultural factors may play a role in the pathways between IPV exposure and subsequent consequences, which cannot be ignored. Although the current study examined overall pathways, it is important for future literature to investigate sociodemographic differences in these trajectories, including differences by ethnicity, type of environment (urban vs. rural), and gender. Indeed, ethnicity was predictive of several psychopathology outcomes when controlling for traumatic event exposure, suggesting the need for future research to understand the mechanisms explaining this relationship. Other limitations were that measures were based on self-report from the adolescents, and it will be necessary to examine the present relationships using objective measures with other raters (e.g., parents). The observed relations only indicate correlations and causality cannot be established from these data, as there were no experimental manipulations.

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Table 1

Descriptive Statistics for Symptoms and Exposure for each Wave

	Wave 1 (*n* = 3,614)		Wave 2 (*n* = 2,511)		Wave 3 (*n* = 1,653)	
Variable	***M***	***SD***	***M***	***SD***	***M***	***SD***
Binge drinking[a]	1.67	10.66	2.71	14.12	4.08	18.06
Depression symptoms[b]	1.16	2.01	1.02	1.91	0.98	1.89
Delinquent behaviors[c]	0.20	0.62	0.17	0.61	0.17	0.62
PTSD[d]	1.45	2.04	1.72	2.96	1.58	2.94
IPV exposures[e]	1.42	2.04	1.74	2.47	1.88	2.73
Non-IPV PTE exposures[f]	0.88	1.29	1.02	1.64	1.03	1.56

Note. PTSD = Posttraumatic stress disorder; IPV = interpersonal violence; PTE = potentially traumatic event.

[a]Number of times consuming ≥5 drinks/day.

[b]Number of 2-week *DSM-IV* depression symptoms during 6 months prior to interview.

[c]Number of serious delinquent behaviors during12 months prior to interview.

[d]Number of *DSM-IV* PTSD symptoms during 6 months prior to interview.

[e]Number of lifetime IPV events prior to interview.

[f]Number of lifetime non-IPV traumatic events prior to interview.

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Table 2

Multiple Regression Analyses Predicting Psychopathology at Wave 2 From Wave 1 Measures

	Original data (*n* = 2,511)		Multiple imputation (*n* = 3,614)	
Predictor	**β**	***t***	**β**	***t***
Depression[a]				
Ethnicity	.01	−0.02	.01	0.51
Sex	.11	6.12***	.11	7.76***
Age	.01	0.06	−.01	−0.80
IPV	.11	5.42***	.11	6.10***
Non-IPV PTE	.01	0.14	.01	0.32
PTSD	.19	6.95***	.19	8.30***
Binge drinking	.01	−0.01	.01	0.13
Depression	.28	10.27***	.27	11.99***
Delinquency	−.02	−0.84	.01	0.24
Delinquency[b,c]				
Ethnicity	.04	1.92	.03	2.06
Sex	−.08	−4.33***	−.08	−5.26***
Age	−.06	−3.00**	−.06	−3.66***
IPV	.10	4.46***	.09	5.27***
Non-IPV PTE	−.01	−0.23	.01	0.25
PTSD	.09	3.03**	.09	3.84***
Binge drinking	.12	5.93***	.12	7.23***
Depression	−.01	−0.18	−.01	−0.44
Delinquency	.32	15.62***	.37	22.24***
Binge drinking[b,d]				
Ethnicity	−.03	−1.97*	−.04	−3.15**
Sex	−.04	−2.07*	−.04	−3.13**
Age	.17	9.71***	.16	11.51***
IPV	−.03	−1.29	−.04	−2.36**
Non-IPV PTE	.02	1.33	.03	2.07*
PTSD	.04	1.52	.04	1.87
Binge drinking	.49	26.59***	.52	34.55***
Depression	−.01	−0.42	−.01	−0.61
Delinquency	.01	0.31	.03	2.10*
PTSD[e]				
Ethnicity	.02	1.05	.03	2.11*
Sex	.09	5.00***	.09	6.61***
Age	.01	0.21	−.01	−0.74
IPV	.11	5.57***	.10	5.91***
Non-IPV PTE	.04	2.29*	.04	2.98**
PTSD	.28	10.62***	.28	12.91***

	Original data (*n* = 2,511)		Multiple imputation (*n* = 3,614)	
Predictor	**β**	***t***	**β**	***t***
Binge drinking	.01	0.74	.02	1.01
Depression	.20	7.53***	.20	9.20***
Delinquency	.01	0.44	.04	2.22

Note. IPV = Interpersonal violence; PTE = potentially traumatic event; PTSD = posttraumatic stress disorder. Minority coded 1 = Caucasian, 2 = other.

[a] For model using completer data, $R^2 = .27$, $F(9,2511) = 104.03$, $p < .001$.

[b] These variables were log transformed to account for nonnormality prior to analyses.

[c] For model using completer data, $R^2 = .21$, $F(9,2511) = 73.33$, $p < .001$.

[d] For model using completer data, $R^2 = .33$, $F(9,2511) = 135.81$, $p < .001$.

[e] For model using completer data, $R^2 = .30$, $F(9,2511) = 121.26$, $p < .001$.

Table 3

Results of Multiple Regression Analyses Predicting Psychopathology Indices at Wave 3

	Original data (*n* = 1,653)		Multiple imputation (*n* = 3,614)	
Predictor	**β**	***t***	**β**	***t***
Depression[a]				
Ethnicity	−.01	−0.15	−.01	−0.37
Sex	.08	3.74***	.10	6.49***
Age	−.01	−0.06	−.01	−0.17
IPV	.12	4.48***	.11	6.05***
Non-IPV PTE	.01	0.21	−.04	−0.88
PTSD	.20	5.66***	.20	8.61***
Binge drinking	−.06	−2.47*	−.04	−2.41*
Depression	.20	5.81***	.20	8.63***
Delinquency	.04	1.64	.04	2.20*
Delinquency[b,c]				
Ethnicity	.04	1.66	.01	0.57
Sex	−.09	−3.89***	−.11	−6.93***
Age	−.06	−2.25*	−.01	−0.74
IPV	.10	3.46**	.13	6.66***
Non-IPV PTE	.01	0.04	.01	0.28
PTSD	.08	2.06*	.08	3.10**
Binge drinking	.11	4.29***	.07	4.03***
Depression	−.04	−1.15	−.06	−2.31*
Delinquency	.23	8.99***	.26	14.84***
Binge drinking[b,d]				
Ethnicity	−.10	−4.86***	−.06	−4.08***
Sex	−.02	−0.98	−.04	−2.72**
Age	.19	8.57***	.22	14.60***
IPV	.09	3.52***	.08	4.57***
Non-IPV PTE	−.01	−0.40	−.04	−2.55*
PTSD	.05	1.60	.04	1.55
Binge Drinking	.38	16.45***	.37	23.12***
Depression	−.01	−0.19	−.01	−0.49
Delinquency	.03	1.36	.03	1.71
PTSD[e]				
Ethnicity	.01	0.55	.02	1.24
Sex	.07	3.19**	.07	4.85***
Age	−.03	−1.25	−.04	− 2.24*
IPV	.07	2.57*	.08	4.60***
Non-IPV PTE	.06	2.67**	.03	2.04*
PTSD	.33	9.50***	.29	12.19***

	Original data (*n* = 1,653)		Multiple imputation (*n* = 3,614)	
Predictor	**β**	***t***	**β**	***t***
Binge drinking	.03	1.15	.04	2.20*
Depression	.11	3.35**	.14	6.18***
Delinquency	−.01	−0.23	−.02	−1.07

Note. IPV = Interpersonal violence; PTE = potentially traumatic event; PTSD = posttraumatic stress disorder. Minority coded 1 = Caucasian, 2 = other.

[a] For model using completer data, $R^2 = .27$, $F(9,2511) = 104.03$, $p < .001$.

[b] These variables were log transformed to account for nonnormality prior to analyses.

[c] For model using completer data, $R^2 = .14$, $F(9, 1653) = 28.63$, $p < .001$.

[d] For model using completer data, $R^2 = .30$, $F(9, 1653) = 76.47$, $p < .001$.

[e] For model using completer data, $R^2 = .24$, $F(9, 1653) = 56.22$, $p < .001$.