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Substance Misuse, Executive Function, and Young Adult Intimate Partner Violence: Direct and Indirect Pathways

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Abstract

Intimate partner violence (IPV) is a significant issue in young adult relationships, with immediate and long-term health and well-being consequences. The factors contributing to IPV are complex and span from the level of individual neurobiology to the wider socioecological system. The interplay across these domains in predicting IPV has been understudied. We, therefore, aimed to examine the factors contributing to IPV risk among young adults, adopting a holistic approach considering direct and indirect contributions of socioecological influences in a cohort of high-risk subjects. Data were from a longitudinal birth cohort established in 1991 comparing the developmental trajectories of individuals prenatally exposed to substances including cocaine and nonexposed individuals. Using data from a subsample of 206 participants followed between 2010 and 2020, we implemented path analysis to examine direct and indirect pathways between prenatal drug exposure (PDE) and young adult IPV. We considered the contributions of childhood maltreatment,

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maternal education, ethnicity, early adolescent substance use, and late adolescent executive function. Sex-specific effects were also explored. There were no significant direct or indirect associations between PDE and IPV. There was evidence of an indirect effect of low maternal education on IPV via effects on early adolescent substance use and subsequent effects on executive function in late adolescence. There was tentative evidence of an effect of ethnicity on IPV risk and of sex differences in the pathways contributing to IPV risk among males and females. We highlight the importance of executive function in young adult IPV risk and suggest considering maternal education and early adolescent substance use as additional contributors to IPV risk. Preventing IPV among young adults may involve enhancing executive functioning and preventing early substance misuse. When examining pathways contributing to IPV risk, it is necessary to adopt a framework integrating the wider socioecological environment.

Keywords
domestic violence, alcohol and drugs, dating violence, predicting domestic violence, intergenerational transmission of trauma, anything related to domestic violence

Introduction

Intimate partner violence (IPV) is a common experience in adolescence and young adulthood. In the United States, IPV is estimated to occur in almost 21% of high school students (Mark et al., 2024) and between 10% and 50% of college students (Spencer et al., 2021). IPV contributes to immediate mental and physical health consequences but also has the potential to perpetuate cycles of adversity across generations (Doyle et al., 2022). As IPV is not the result of single, individual risk factors, a holistic perspective is required to better understand the pathways contributing to IPV and its consequences. An approach that considers prenatal vulnerability in conjunction with socioecological circumstances is valuable in understanding the development of IPV and related outcomes, as well as the prevention of IPV.

This study focuses on prenatal drug exposure (PDE) as a marker of heightened neurobiological risk, given its association with increased infant mortality, premature birth, and impairments in children's neurodevelopment, especially the development of executive functioning (EF) (Skranes & Løhaugen, 2022). PDE also increases the likelihood of an individual's own future substance use, including cocaine use specifically (Delaney-Black et al., 2011), consequently increasing IPV risk (Romero-Martínez et al., 2023a).

This study focuses on marginalized status as a marker of heightened socioecological risk that co-occurs with PDE and simultaneously contributes to IPV risk. The use of substances, including cocaine, in pregnancy is disproportionately endorsed among marginalized individuals with reduced access to resources and education, and who tend to face greater socioecological adversity (Alhusen et al., 2013; Beyer et al., 2015). Together, PDE and concurrent systemic adversity may contribute to pathways that heighten the risk of IPV in young adulthood and risk perpetuating multigenerational cycles of risk. However, it remains unclear how these multiple influences directly or indirectly predict IPV risk, and while substance use is an established predictor of IPV, the contribution of prenatal substance exposure to IPV remains unclear.

Prenatal Influences on Executive Function Development and IPV Risk

Numerous concurrent factors may hamper development during the prenatal period as the physiological connection between mothers and fetuses during pregnancy exposes fetuses to conditions surrounding their mothers (Barry et al., 2021). PDE, including exposure to cocaine specifically, may compromise fetal neurodevelopment (Lambert & Bauer, 2012; Zhao et al., 2022) by impacting the maturation of neural pathways required to support EF (Skranes & Løhaugen, 2022). Simultaneously, fetal exposure may later exacerbate an individual's sensitivity to adversity that continues to persist postnatally (Bauer et al., 2011; Chaplin et al., 2010).

The association between PDE and EF may be relevant to IPV risk pathways, given the well-established link between EF and IPV. Users of IPV consistently show neuropsychological impairment in cognitive flexibility, attentional control, and planning, impairments that meta-analyses find to be especially apparent among IPV users also reporting drug misuse (Romero-Martínez et al., 2023b). EF deficits compromise threat detection, regulating emotion and aggression, and disengaging from conflict (Bueso-Izquierdo et al., 2022; Chester & DeWall, 2019). While drug use exacerbates these effects, EF deficits compromise regulation independently of substance misuse (Romero-Martínez et al., 2023b). Examining the factors that may first impair EF development may be important for understanding and intervening with pathways later predicting IPV, given the robust association between EF and the regulatory functions essential for navigating interpersonal conflict.

PDE, and exposure to cocaine specifically, appears to be a predictor of deficits in EF and behavioral regulation (Ackerman et al., 2010). Longitudinal research has found associations between prenatal cocaine exposure (PCE) and poorer inhibition capacity among children aged 7.5 to

14 (Bridgett & Mayes, 2011), elevated stress responding (Chaplin et al., 2010), neurocognitive alterations predictive of future substance initiation (Rando et al., 2013), and the likelihood of future arrests and conduct disorders (Fals-Stewart et al., 2003). Regarding mid-adolescence, similar associations have been found between prenatal smoking and EF impairments (Knopik et al., 2022), prenatal alcohol consumption and affect regulation (Chu et al., 2022), and prenatal cannabis exposure on substance initiation and behavioral regulation among adolescents (De Genna et al., 2022). PDE may, therefore, be a meaningful predictor of EF deficits and, thus, later IPV.

However, evidence is not consistent regarding the interpersonal outcomes linked to PDE, especially when considering outcomes that extend beyond childhood into early adulthood (Godleski et al., 2022). Regarding cocaine specifically, associations between PCE and cognitive abilities in mid-to-late adolescence tend to be small and modulated by the effects of the confounding environment (Skranes & Løhaugen, 2022). Research regarding IPV as a specific outcome related to PDE is particularly lacking. Only one study has so far examined the effects of PDE on IPV in adolescence and did not find direct or indirect associations (Stover et al., 2018). Accordingly, the long-term effects of PDE, and of cocaine exposure specifically, are far from conclusive.

Furthermore, given that PDE typically occurs in a broader context of environmental adversity, examining associations between PDE and later outcomes requires integration of the concurrent socioecological context (Skranes & Løhaugen, 2022). Socioecological adversity, such as reduced access to education and healthcare, and the experience of stress during the prenatal period may directly contribute to substance use during pregnancy and simultaneously compromise fetal neurodevelopment. Substantial literature has provided evidence of a link between adverse prenatal conditions and EF impairments (McGowan & Matthews, 2018), which is likely a result of dysregulated hypothalamic-pituitary-adrenal (HPA) axis functioning, which, accordingly, impairs stress regulation, emotional reactivity, and fetal neuromaturation (Demers et al., 2021). Co-occurring socioecological adversity during the prenatal period, such as maternal marginalized status, may elevate IPV risk through neurobiological effects.

Postnatal Influences on Neurodevelopment and IPV Risk

Environmental adversity that persists postnatally may continue to hamper development and exacerbate impairment as parents in adverse socioecological circumstances during pregnancy are likely to face continued difficulty, risking child exposure to various forms of early life adversity (ELA) (Zhao et al., 2022). Continued substance misuse may also affect caregiving and increase

children's exposure to maltreatment and conflict (Carliner et al., 2016; Shadur & Hussong, 2020), ultimately contributing to IPV risk pathways.

By interacting with HPA functioning and stress regulation, ELA may directly impair the development of neural pathways essential for EF, especially with repeated exposure to stressors (Luby et al., 2020). Externalizing problems, such as behavioral and attentional problems at school, are common among children and adolescents exposed to ELA (Lui et al., 2023; Thornberry et al., 2003). ELA may also heighten vulnerability to future substance misuse (Kirsch et al., 2020). Substance use, particularly illicit drugs such as cocaine, or polysubstance use, is strongly associated with IPV (Kraanen et al., 2014; P. H. Smith et al., 2012; Romero-Martínez et al., 2023a). Substances can exacerbate mental health difficulties and conflict in relationships and impair EF skills necessary for navigating relationships (Fink et al., 2023; Taylor & Sullivan, 2021; Weiss et al., 2021). The effect of heightened neurobiological risk due to PDE, combined with the effects of postnatal adversity, may together heighten vulnerability to IPV, especially among the most marginalized (Alhusen et al., 2013; Bueso-Izquierdo et al., 2015; Miguel et al., 2019).

Familial and Socioecological Systemic Influences

Wider familial and socioecological factors may also increase IPV risk via systemic mechanisms. Circumstances surrounding marginalized families may limit parents' monitoring of their children and permit fewer opportunities for healthy role modeling (Cappa & Giulivi, 2019). Their children may be more likely to engage with peer groups endorsing early substance misuse and antisocial behavior, which are established risk factors for IPV (Franklin & Kercher, 2012; Knous-Westfall et al., 2012). Indeed, longitudinal work by Hentges et al. (2018) found increased substance use and aggression among adolescents who reported higher levels of impulsivity and who had also experienced parental rejection in earlier childhood. Thus, familial circumstances that limit parenting quality, in combination with EF deficits, may together contribute to pathways that increase the likelihood of IPV.

In the United States, unique systemic and cultural stressors such as racial discrimination and financial instability may contribute to externalizing behaviors, including IPV specifically (Bueso-Izquierdo et al., 2015). Limited access to the resources that may buffer against these stressors may perpetuate adversity over generations (E. P. Smith et al., 2022). Highlighting the importance of examining ethnicity as a factor in pathways contributing to IPV, Rice et al. (2022) claim that African American women are thrice as likely as Caucasian women to be killed by their partners. Qualitative explorations suggest that lingering policies and systemic barriers may make seeking adequate housing,

employment, and resources a challenge that disproportionately impacts ethnic minority communities (Kulkarni & Notario, 2024). These challenges may contribute to IPV by increasing interpersonal conflict among families, contributing to maladaptive substance misuse to cope with stress, and limiting individuals' ability to seek help or escape a dangerous situation (Stewart & Haselschwerdt, 2023). While these barriers are disproportionately faced by individuals representing marginalized sectors of the population, the precise pathway through which ethnic minority status, marginalization, and socioeconomic disadvantage impact IPV is not conclusive and requires further examination.

The Current Study

The pathways contributing to IPV in adolescence and young adulthood are complex and span multiple levels of influence. These range from neurobiological factors, such as PDE, to the family sphere, such as parenting quality, and wider socioecological factors, including access to education, and experiencing marginalization-related stressors. Few studies have attempted to examine their joint contributions to IPV risk pathways. Given the co-occurrence of multiple risk factors, marginalized communities may be disproportionately at risk of IPV and its consequences. Thus, we aimed to examine the direct and indirect influence of socioecological factors on pathways contributing to IPV among a high-risk cohort.

We adopt a holistic approach, specifically considering the contributions of prenatal neurobiological risk, represented by PDE, contributions of postnatal influences of childhood maltreatment, early adolescent substance misuse, and late adolescent EF, and contributions of marginalization, represented by maternal ethnicity and educational status. We also consider IPV use *and* victimization in young adult dating relationships as IPV is often bidirectional (Bates, 2016; Caetano et al., 2007). No direct association between PDE and IPV was predicted based on prior analyses within this cohort (Stover et al., 2018). However, PDE was expected to be associated with adolescent EF impairment, and impaired EF was hypothesized to increase IPV. Based on literature examining links between maternal education and development, low maternal education was expected to elevate the risk of young adult IPV, directly *or* indirectly (Cyr et al., 2022; Harding, 2015; Jackson et al., 2017). We predicted greater young adult IPV among minority groups based on literature examining the effects of marginalization-related stress on neurobiology and behavior (E. P. Smith et al., 2022). Early adolescent substance misuse was expected to predict young adult IPV, and PDE was expected to predict early adolescent substance misuse. Sex differences were also anticipated based on prior analyses involving this cohort, with males expected to show

greater early adolescent substance misuse and thus elevated IPV risk (e.g., Chaplin et al., 2010; Rando et al., 2013).

Methods

Participants

Data for the current study were drawn from an archived dataset for a longitudinal study that recruited mothers and infants born at Yale-New Haven Hospital from 1991 to 1996, originally including 523 mothers and their prenatally recruited infants (Mayes et al., 2005). This cohort represents a high-risk group of mothers from an underprivileged, inner-city context. For the present study, we used a subsample of 121 mother-child dyads from the original sample, who participated in longitudinal follow-up from infancy to early adulthood. In the original sample, subjects reporting opioid use were excluded, and no infants were breastfed.

For the adolescents' continuing participation, subjects between 15 and 18 years of age at the first follow-up timepoint were included if they had begun dating. Adolescents were excluded if they reported dependence on psychoactive substances (excluding nicotine) and they reported severe psychiatric symptoms requiring care or hospitalization.

Baseline and Prenatal Measures

Prenatal Drug Exposure. Data were collected using the Addiction Severity Index (McLellan et al., 1992) and urine toxicology (Supplemental Material). PDE was ordinally coded with increasing levels of severity, representing participants without any exposure, with exposure to non-cocaine substances, and with exposure to cocaine and other substances. Cocaine use represents the most severe level of PDE. Ordinal coding was used to represent PDE as a single risk factor increasing in severity rather than separate groups to improve statistical power. Infant birthweight and sex were recorded at birth.

Marginalization Measures. Maternal ethnicity and education were recorded as categorical variables, at prenatal care registration or delivery registration as indicators of marginalization. Mothers either identified as African American, Hispanic, or Caucasian and indicated whether they had completed high school.

Adolescent and Young Adult Measures

Childhood Maltreatment. At age 14, adolescents completed the self-report Childhood Trauma Questionnaire, recording histories of abuse (physical,

sexual, and emotional) and neglect (physical and emotional) up to the age of 14 (Bernstein et al., 2003). Internal consistency was good ($\alpha_{Cronbach} = .838$).

Executive Functioning. We used participants' self-reported EF from ages 18 to 21 using the adult version of the Behavior Rating Inventory of Executive Function (BRIEF) (Roth et al., 2013). BRIEF addresses nine domains of executive function: Inhibit, Shift, Emotional Control, Task Monitor, Self-Monitor, Working Memory, Plan/Organize, Organization of Task Materials, and Task Completion. BRIEF shows excellent internal consistency within this subsample ($\alpha_{Cronbach} = .969$), and good construct and discriminant validity among adolescents (Nyongesa et al., 2019). This timepoint was selected as few studies have examined associations between PDE and late adolescent/young adult EF and so that EF could be specified as a predictor of young adult IPV. Participants attended varying numbers of follow-up visits; each participant's average BRIEF score across their visits within this 3-year window was calculated. This was to avoid excluding participants with poorer adherence. We confirmed there were no differences in EF based on the number of visits attended to address potential bias (Supplemental Figure 1).

Intimate Partner Violence. The Conflict in Adolescent Dating Relationships Inventory (CADRI) was used to measure total IPV use and victimization occurring between ages 22 and 25 (Wolfe et al., 2001). CADRI is a 50-item self-report measure addressing experiences and use of dating violence behaviors, with 25 items pertaining to experiences and 25 to use behaviors. CADRI addresses 5 subtypes of conflict that can occur in relationships: threatening behaviors, relational aggression, and physical, verbal, and sexual abuse. Participants reported how frequently on a scale of "never (1)" to "often (4)," each item was experienced. CADRI has good internal consistency (use $\alpha_{Cronbach} = .74$; experience $\alpha_{Cronbach} = .80$).

Substance Misuse. The computerized Teen version of the Addiction Severity Index (T-ASI) (Kaminer et al., 2016) was administered between ages 14 and 29. Binary coding was used to indicate whether participants' first age of substance use began before age 18. Reliability of the T-ASI for capturing adolescent substance use has been estimated between 0.66 and 0.79 (Brodey et al., 2008). Urine toxicology was not used to confirm self-reported substance use. See Supplemental Material for further details on study measures and coding of categorical variables.

Statistical Analyses

Statistical analyses were conducted using R and Jamovi (2.3.19). Models were specified and tested iteratively to examine the direct and indirect pathways between PDE (at birth), childhood maltreatment history (reported at age 14), EF in late adolescence (reported between ages 18 and 21), early adolescent substance use before age 18 (reported between ages 14 and 29), and young adult IPV (reported between ages 22 and 25). Path analysis used the DWLS estimator given our modest sample size and inclusion of ordinal data, violating assumptions of multivariate normality. Standard errors were bootstrapped with 1,000 draws. Several path models were specified, pruned, and compared referring to the model chi-square, adjusted Goodness-Of-Fit Index, and standardized root mean squared residual. The results show only pruned model results; all alternate non-pruned model specifications and results are available in Supplemental Material S2.

Results

The full sample for this study included 121 participants (62 females, 59 males), 106 with substance use data (56 females; 56 males). *t*-Tests verified that the subsamples did not differ across measures (Supplemental Table 1). The mean young adult IPV reported was 70.92 ($SD = 14.91$), out of a maximum score of 100. Of the 121 participants, 54.1% were prenatally exposed to cocaine and other substances, 19% were exposed to substances other than cocaine, and 26.4% were nonexposed. Most participants were of African American descent (82.6%), 12.4% Caucasian, and the remaining Hispanic (Table 1).

PDE significantly varied based on maternal ethnicity and maternal education. African Americans reported more prenatal exposure to cocaine (61.0%) compared to Caucasians (13.3%). Cocaine exposure was also higher among participants with mothers who had not achieved at least a high school level of education (78.0%) compared to participants with mothers achieving at least a high school level of education (42.5%) (Supplemental Table 2). Young adult IPV, late adolescent EF, and childhood maltreatment did not significantly differ by PDE. Birthweight significantly differed across levels of PDE, with a lower average birthweight in the cocaine-exposed compared to nonexposed subjects ($\mu_D = 540$ g, $t[118] < 0.001$) (Supplemental Table 3).

Of individuals experiencing IPV, all participants reported bidirectional IPV. Young adult IPV was positively correlated with late adolescent EF impairment ($r = .362$, $p < .001$). PDE was positively correlated with early

Table 1. Participant Demographic Information Table.

	No Substance Exposure (*N* = 32, 26.4%)	Prenatal Exposure (Non-Cocaine) (*N* = 23, 19.0%)	Prenatal Exposure to Substances and Cocaine (*N* = 66, 54.5%)	Full Sample (*N* = 121, 100%)
Baseline Characteristics	*N* (%)	*N* (%)	*N* (%)	*N* (%)
Gender				
Female	15 (24.2)	15 (24.2)	32 (51.6)	62 (51.2)
Male	17 (28.8)	8 (13.65)	34 (57.6)	59 (48.8)
Maternal race/ethnicity				
African American	22 (22.0)	17 (17.0)	61 (61.0)	100 (82.6)
Hispanic	1 (16.7)	2 (33.3)	3 (50.0)	6 (5.0)
Caucasian	9 (60.0)	4 (26.7)	2 (13.3)	15 (12.4)
Maternal education				
Below high school	4 (9.8)	5 (12.2)	32 (78.0)	41 (33.9)
High school or above	28 (35.0)	18 (22.5)	34 (42.5)	80 (66.1)

Note. *N* = 121.

Table 2. Model Results for Associations Between PDE, Childhood Maltreatment, EF, and Young Adult IPV.

				95% CI		
Dep	Pred	β	SE	Lower	Upper	*p*
Young adult IPV	Late adolescent EF impairment	.338	0.096	0.142	0.533	< .001
	Childhood maltreatment	.076	0.086	−0.099	0.229	.379
Late adolescent EF impairment	PDE	.022	0.097	−0.148	0.219	.820
	Childhood maltreatment	.130	0.108	−0.065	0.370	.231
Childhood maltreatment	PDE	−.036	0.098	−0.244	0.141	.715

Note. Based on 1,000 bootstrap draws. β = standardized regression coefficient; 95% CI = 95% confidence interval; SE = bootstrapped standard error; IPV = intimate partner violence; PDE = prenatal drug exposure; EF = executive functioning.

adolescent substance misuse ($r = .199$, $p = .041$) and maternal high school education ($r = -.325$, $p < .001$) (full results in Supplemental Table 4).

No models found PDE to be directly or indirectly predictive of outcome variables. There were indirect associations between socioecological factors and outcome variables.

Associations Between PDE, Childhood Maltreatment, Adolescent EF, and Young Adult IPV

The pruned model specified indirect pathways between PDE and young adult IPV through childhood maltreatment and late adolescent EF. Birth weight was not found to be significantly associated with any of the variables.

This model ($N = 121$, parameters = 11) (results in Table 2) achieved an excellent fit ($\chi^2[1] = 0.276$; $p = .599$, Standardized Root Mean Squared Residual (SRMR) = 0.012, adjusted Goodness-of-Fit Index (aGFI) = 0.990) and accounted for 12.7%, 1.71%, and 0.13% of the variance in young adult IPV, late adolescent EF and childhood maltreatment, respectively. The only significant association was direct, between late adolescent EF and young adult IPV ($\beta = .338$, $p < .001$). PDE was not significantly associated with childhood maltreatment or late adolescent EF. Non-pruned model results are in Supplemental Table 5.

Contributions of Maternal Education, Ethnicity, and Early Adolescent Substance Use

This model specified indirect pathways between marginalization measures at birth and young adult IPV through earlier adolescent substance misuse and EF impairment. The final model was selected through iterative modeling to include only relevant pathways (non-pruned model results in Supplemental Table 6).

Figure 1 shows the pruned model. PDE, maternal education, and maternal ethnicity were specified as predictors of early adolescent substance misuse, and substance misuse was a predictor of late adolescent EF and young adult IPV. This model ($N = 106$, parameters = 12) achieved a good fit ($\chi^2[9] = 8.55$; $p = .480$, SRMR = 0.045, aGFI = 0.963) and accounted for 15.8%, 8.48%, and 8.27% of the variance in young adult IPV, late adolescent EF and early adolescent substance misuse, respectively. The direct effect of late adolescent EF on young adult IPV remained significant ($\beta = .354$, $p < .001$), as did the protective effect of Caucasian maternal ethnicity ($\beta = -.173$, $p = .034$). Early adolescent substance misuse predicted late adolescent EF impairment ($\beta = .291$, $p = .013$). Maternal high school education had a significant protective effect on early adolescent substance use

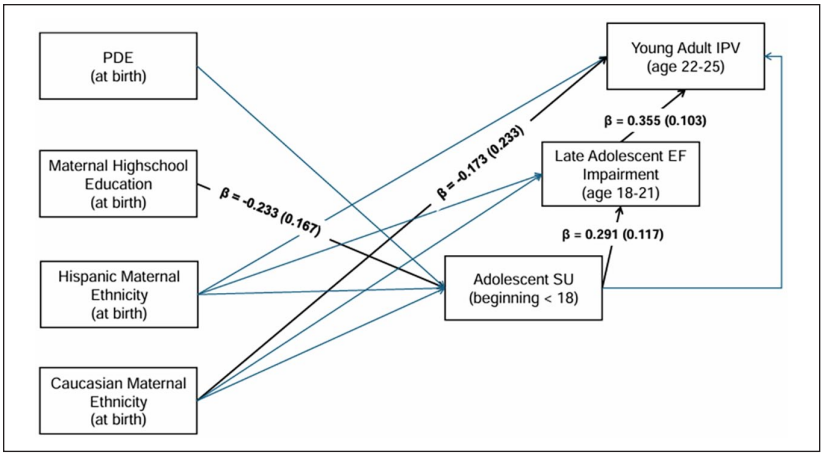

Figure 1. Simplified path diagram for model examining contributions of maternal ethnicity, maternal education, and early adolescent substnace misuse to young adult IPV. PDE = prenatal drug exposure; SU = subtance use; EF = executive function, IPV = intimate partner violence.

Table 3. Results for Model Including Maternal Education, Ethnicity, and Early Adolescent Substance Misuse.

				95% CI		
Dep	Pred	*b*	SE	Lower	Upper	*p*
Young adult IPV	Late adolescent EF impairment	0.355	0.103	0.150	0.559	< .001
	Early adolescent substance misuse	−0.058	0.111	−0.297	0.129	.600
	Hispanic maternal ethnicity	0.074	0.304	−0.285	0.907	.255
	Caucasian maternal ethnicity	−0.173	0.233	−0.987	−0.082	.034
Late adolescent EF impairment	Early adolescent substance misuse	0.291	0.117	0.129	0.478	.013
Early adolescent substance misuse	Maternal high school education	−0.233	0.167	−0.828	−0.157	.003
	Hispanic maternal ethnicity	0.025	0.191	−0.215	0.515	.535
	Caucasian maternal ethnicity	−0.127	0.319	−0.905	0.349	.256
	PDE	0.033	0.125	−0.189	0.299	.792

Note. Nine hundred eighty-nine successful bootstraps. IPV = intimate partner violence; PDE = prenatal drug exposure; EF = executive functioning.

(β = −.233, p = .003). PDE was not significantly associated with early adolescent substance use (Table 3).

Exploratory Sex-Specific Pathways

The full model for sex-specific pathways included all variables, amounting to 58 model parameters (Supplemental Table 7). A pruned model (Supplemental Material S2), with 28 parameters, achieved a good fit ($\chi^2[20] = 11.4$; p = .936, SRMR = 0.048, aGFI = 0.952), and among girls accounted for 4.2%, 20.1%, and 29.6% of the variance in young adult IPV, late adolescent EF and early adolescent substance misuse, respectively. Among boys, it accounted for 36.5%, 33.1%, and 18.6% of the variance in IPV, EF, and early adolescent substance misuse, respectively. Detailed sex-specific results are in the Supplemental Figure 2 and Supplemental Table 8.

Discussion

We examined the direct and indirect influence of socioecological and systemic factors on young adult IPV. Challenges that disproportionately face marginalized communities, such as limited access to education, exposure to ELA, and substance misuse, together may elevate young adult IPV risk. We adopted a holistic, multidomain approach to consider the interplay of these factors and to address limitations of existing research examining pathways contributing to IPV. Given the high proportion of participants exposed to cocaine prenatally in this cohort, we focus our discussion on literature examining PDE, including the exposure to cocaine specifically.

Replicating previous findings (Stover et al., 2018), there was no direct link between PCE and young adult IPV, nor an indirect association through socioecological factors. Other studies have not found robust associations between PCE and early adolescent outcomes like aggression (Godleski et al., 2022), and few studies have examined associations with late adolescent outcomes. In studies examining late adolescence, the relationship between PCE and EF is unclear. Among 21-year-olds, Willford et al. (2018) found no PCE-related differences in neural activity during an EF task. In contrast, Richardson et al. (2019) found a direct effect of PCE on emotion regulation at age 21. Additional cohorts maintained into late adolescence are needed to further understand the long-term effect of PCE. While it is challenging to isolate the effects of cocaine specifically, given its typical use with other substances, such isolation may be useful for highlighting effects driven uniquely by cocaine exposure as opposed to other substances, though this was not a primary aim of this study.

We provide further support for a link between EF and IPV, a relationship that has been well-supported by existing meta-analyses (e.g., Romero-Martínez et al., 2023b). EF impairment contributed to young adult IPV risk even after accounting for birth weight, maternal ethnicity, maternal education, and childhood maltreatment. The importance of EF in IPV is further highlighted by our finding that early adolescent substance misuse and maternal education were associated with IPV only via interactions with EF. During adolescence, substance misuse may compromise the development of regions critical to EF (Bava & Tapert, 2010; Hammond et al., 2014), which may heighten emotional reactivity and dampen regulation, elevating the risk of aggression and IPV (Fink et al., 2023; Weiss et al., 2021).

No direct effect of substance misuse on IPV was found, contrasting other studies supporting direct effects of substance use on IPV use and victimization (Mark et al., 2024). This may be partly explained by a limitation in our approach of using only self-reported substance use, leading to potential underreporting, and our exclusion of adolescents reporting dependence on psychoactive substances. Associations between substance misuse and EF are worth investigating further, given their bidirectional and cyclical relationship (Kim-Spoon et al., 2017; Luciana, 2020). The indirect effect of early adolescent substance misuse on IPV may simply reflect persistent EF deficits rather than a unique effect of substance misuse on IPV. Future research should, therefore, consider EF deficits existing prior to substance use to confirm the directionality between EF and substance use and disentangle their contributions to IPV risk.

Low maternal education indirectly increased IPV by predicting early adolescent substance misuse. Low maternal education, a proxy of marginalization, has previously been associated with maternal psychiatric symptoms and substance misuse, limited access to resources and healthcare, and other environmental adversity (Jackson et al., 2017; Rasmussen et al., 2022). Adversity linked to low maternal education and access to resources may impact caregiving capacity (Cappa & Giulivi, 2019; Jackson et al., 2017) and facilitate adolescents' early substance misuse and participation in peer groups promoting risk-taking and violence (Knous-Westfall et al., 2012; Lee & Vandell, 2015). These may impair EF development while simultaneously elevating adolescent IPV risk (Taylor & Sullivan, 2021). Parents' own substance misuse or conflict may also model harmful behaviors (Franklin & Kercher, 2012; Stover & Kahn, 2013), highlighting the importance of considering the contributions of marginalization and socioecological circumstances to EF development.

Like previous research using this cohort (Chaplin et al., 2015), analyses suggested sex differences in the contribution of childhood maltreatment to early adolescent substance use and, indirectly, to EF impairment. Girls with

heightened stress responses were more likely to report adolescent substance misuse. Epidemiological and clinical literature does indeed find that women with maltreatment histories are more at risk of using alcohol to cope with stress (Peltier et al., 2019; Verplaetse et al., 2018). However, these findings should be considered exploratory given the model's complexity and warrant future investigation with a more powerful sample.

Study Limitations

While we provide evidence of associations between maternal ethnicity and young adult IPV, particularly when comparing the risk of IPV among African American and Caucasian populations, we must consider the unbalanced sample. African American ethnicity may be a direct risk factor for IPV due to structural barriers and stressors linked to ethnicity (Cramer & Plummer, 2009; Field & Caetano, 2004). However, their elevated risk of IPV may simply reflect their over-representation in this cohort. We also did not directly examine the effects of sociocultural attitudes surrounding gender roles, aggression, and relationships, which may contribute to IPV, particularly among marginalized communities (Guerrero-Molina et al., 2023; Senkans et al., 2020). These norms may simultaneously discourage help-seeking and instead promote maladaptive coping strategies (Gage & Lease, 2021; Stewart & Haselschwerdt, 2023).

Given the high-risk, longitudinal nature of this study, attrition is another concern. PDE, particularly cocaine, is a risk factor for early mortality and is linked with socioecological factors that may hamper participation in longitudinal research (Goldstein et al., 2021). Attrition may explain our lack of association between PDE and behavioral outcomes; individuals with the most severe outcomes may no longer be participants. Repeated participation in longitudinal studies can also be laborious and demanding, particularly for adolescents with limited flexibility and mobility and who may lack the EF skills for planning and keeping appointments (Davis et al., 2016). Future research using high-risk cohorts should optimize strategies promoting accessibility and retention.

Our reliance on self-report measures, particularly for IPV and adolescent substance misuse, should also be considered. Individuals may be reluctant to fully disclose the extent of IPV and underage substance misuse, bringing the accuracy of these measures into question. While underreporting is likely, our analyses were still able to capture associations between adolescent substance use and EF and between EF and IPV. Thus, even at relatively lower severity levels, the relationships between EF and IPV and substance use and EF seem to be robust. Validation of adolescent substance use via toxicology may have been useful, but it may also have further deterred in-person follow-up visits.

Similarly, while the self-report BRIEF is considered reliable among adolescents, additional measures of EF such as the Wisconsin Card Sorting Test may be useful in providing validation for this self-report measure and should be considered for any future follow-up timepoints with this cohort.

Implications

Our findings highlight the importance of considering wider socioecological circumstances surrounding marginalized populations at risk of young adult IPV. Women with limited access to resources/education during the prenatal period may benefit from receiving greater support to meet their material, psychological, and emotional needs. Support schemes may include promoting or financially sponsoring registration for early prenatal care, and enrolling in psychoeducational/therapeutic programs or substance use interventions (Abatemarco et al., 2021; Goler et al., 2012; Shi & MacBeth, 2017). Larger-scale policy and healthcare system changes are likely necessary, as inaccessibility and unaffordability largely account for disparities in prenatal care registration and birth outcomes (Gadson et al., 2017). Fear of judgment and being reported to Child Protective Services may also hinder prenatal care enrollment among populations reporting substance misuse (Frazer et al., 2019; Roberts & Pies, 2011). Birth outcomes may be improved by reducing the stigma associated with maternal substance misuse and training healthcare workers to provide judgment-free spaces where expectant mothers can feel empowered and aware of care options for their circumstances (Weber et al., 2021).

Our finding of pathways between adolescent substance use and EF impairments, and later young adult IPV, suggest that interventions for IPV should also address these difficulties. Stover et al. (2022) highlight the importance of fostering EF capacities while simultaneously addressing substance misuse to more effectively reduce anger, hostility, and IPV. For adolescents, programs implemented in schools or the local community, involving parents, teachers, or mentors, may enable adolescents to improve relationship skills and reduce IPV risk when dating (De Koker et al., 2014). Alongside targeting relationship skills and EF, care providers should also consider the accessibility and affordability of interventions. Larger-scale systemic changes improving access to high-quality education, reducing financial stressors, and improving community safety are likely necessary to disrupt multigenerational cycles of socioecological stress and violence.

Conclusion

This study examined the socioecological and systemic factors indirectly and directly associated with IPV risk among a sample of high-risk individuals.

We contribute to existing literature emphasizing the link between EF and IPV risk. We additionally found that maternal education indirectly predicted young adult IPV risk by elevating the risk of early adolescent substance misuse and compromising late adolescent EF capacity. We, therefore, highlight the importance of considering IPV risk and substance misuse through a holistic framework rather than viewing these outcomes solely as products of neurobiological vulnerability. Alongside aiming to improve prenatal conditions, providing support to marginalized women with low income and less education may be a crucial avenue for preventing substance misuse and IPV, perhaps over multiple generations. Finally, as part of the growing approach in developmental psychopathology, we emphasize that psychopathology cannot be understood from a single monolithic perspective. To understand complex challenges, such as IPV, holistically and meaningfully, and to implement effective prevention strategies, we must consider the unique circumstances surrounding specific individuals and the dynamic systems they inhabit.

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Supplemental Material

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