THE ROLE OF CAREGIVER PRE- TO POSTNATAL SUBSTANCE USE, PARENTAL MONITORING, AND EARLY ADOLESCENT PEER PROCESSES ON LATE ADOLESCENT SUBSTANCE USE

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by

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ABSTRACT

We examined a developmental model for late adolescent substance use beginning with prenatal substance use. The model examined the role of caregiver prenatal and postnatal substance use on late adolescent substance use through three mediation pathways: early adolescent parental monitoring, early adolescent substance use, and early adolescent peer group delinquency behaviors. The sample consisted of 216 mother-child dyads with \( N = 116 \) in the sample with prenatal cocaine use and \( N = 100 \) in the sample without prenatal cocaine use. The sample was diverse (72% Black) and predominately low-income. A path analysis was conducted and results did not find a significant pathway from prenatal and postnatal substance use to adolescent substance use. However, results did find significant covariances between early adolescent parental monitoring, early adolescent peer group delinquency behaviors, and early adolescent substance use. Additionally, a significant pathway from early adolescent substance use to late adolescent substance use was found. Results from a multigroup analysis indicated that among adolescents with low levels of early adolescent peer group delinquency behaviors, lower postnatal substance use was associated with late adolescent substance use. A second multigroup analysis found that among male adolescents, higher prenatal substance use was associated with late adolescent substance use. The results emphasize the importance of caregiver substance use, parental monitoring, and peer group delinquency behaviors within a low-income, diverse sample.
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INTRODUCTION

Experimentation with substance use is part of the normative risk-taking process of the adolescent period. A national U.S. survey on substance use found that for 2021, 7.3% of eighth graders reported alcohol use, 4.1% reported marijuana use, and 9.4% reported nicotine use (including vaping) in the past 30 days. By 12th grade, these rates had increased to 25.8% for alcohol 19.5% for marijuana, 24.6% and for nicotine use (including vaping) (Substance Abuse and Mental Health Services Administration, 2022). Results also indicated that 10.2% of eighth graders and 32% of twelfth graders reported using illicit substances (Substance Abuse and Mental Health Services Administration, 2022).

Although substance use experimentation may be normative during adolescence, it is not without risk. Initiation of substance use in early adolescence (11-14 years of age) has been linked with a greater risk for developing a substance use disorder (DeWit et al., 2000; Richardson et al., 2013). Meanwhile, initiation of substance use in late adolescence (15-19 years of age) is associated with a more rapid transition between the time of initiation and the development of a substance use disorder (Behrendt et al., 2009). Results from a longitudinal study spanning 32 years found that participants with a greater number of substance use disorder symptoms as high school seniors were significantly more likely to have two or more substance use disorder symptoms and prescription drug use and misuse at age 50 (McCabe et al., 2022). Overall, studies support that both early initiation and higher frequency of substance use in adolescence is associated with a greater risk of developing a substance use disorder in later life.

Prenatal substance use as a significant risk factor for both earlier initiation and higher rates of substance use in adolescence (Glantz & Chambers, 2006; Richardson et al., 2013). Moreover, prenatal substance use is often a marker for continued postnatal use (Forray & Foster,
2015). However, only a handful of studies have examined the role of chronicity of substance use from pre-to-postnatal periods in predicting adolescent substance use (De Genna et al., 2015, 2017).

Caregiver substance use is also linked to lower parental monitoring, a significant proximal risk factor for adolescent substance use (Clark et al., 2015). Thus, parental monitoring may partially mediate the association between chronicity of substance use and adolescent substance use. Finally, robust evidence indicates that peer group substance use is a proximal predictor of adolescent substance use even in the context of parental substance use disorders (e.g., Eiden et al., 2016); however, peer group risk behaviors may also exacerbate the association between caregiver and adolescent substance use. On the other hand, adolescent engagement with prosocial peers may be protective, ameliorating the association between parental monitoring and adolescent substance use. To build on this existing evidence, this thesis will examine both main effects and interactions, which was tested using longitudinal data from the prenatal period to late adolescence (see Figure 1). These effects are explicated in more detail below.

**Role of Pre-to-Postnatal Substance use for Adolescent Substance Use**

*Prenatal Substance Use and Adolescent Substance Use*

Prenatal substance use is associated with a higher risk for adolescent substance use (Minnes et al., 2017; Weinberg, 2001). Several theoretical orientations, such as fetal programming (Barker, 1998; Kwon & Kim, 2017) and Developmental Origins of Health and Disease (DOHaD) (Gluckman et al., 2010; K. J. O’Donnell & Meaney, 2017) highlight the importance of the prenatal period on development. Additionally, translational neuroscience perspectives (Horn et al., 2018) highlight how prenatal substance use is a marker for toxic stress in the prenatal period. These theories state that high prenatal stress has long-lasting implications for children’s
developmental outcomes (Cao-Lei et al., 2016), including engagement in risk behaviors through changes in stress (Chaplin et al., 2015), arousal (Chaplin et al., 2015), and reward processes (Müller et al., 2013). Taken together, there is clear theoretical and empirical support for examining the association between prenatal substance use and adolescent substance use.

However, only a limited number of longitudinal studies with prospective, multi-method assessments of prenatal substance use have examined this association (Baer et al., 2003). In addition, studies that have examined this association have typically focused on the association between the prenatal use of one substance, such as cocaine, alcohol, marijuana, or tobacco, and adolescent substance use (Baer et al., 1998; Chaplin et al., 2015; Richardson et al., 2013), and results have been mixed. For instance, among studies examining associations between prenatal cocaine use and adolescent substance use, results have varied with some studies reporting prenatal cocaine use as a significant predictor of adolescent substance use (Richardson et al., 2013) use while others have found this association to not be significant (Bennett & Lewis, 2020). One explanation for these mixed results may be that the timing of prenatal cocaine use impacts how prenatal cocaine use is associated with adolescent substance use. For example, longitudinal studies have found prenatal cocaine use during the first trimester of pregnancy to be significantly associated with earlier adolescent initiation of alcohol and marijuana use but not of adolescent tobacco initiation (Richardson et al., 2013).

In contrast, Bennett and Lewis (2020) found that prenatal cocaine use was not a significant predictor of adolescent substance use in 15-17.5-year-olds. Instead, the authors found that proximal factors, such as peer substance use and adolescent depressive symptoms, predicted substance initiation and use (Bennett & Lewis, 2020). Meanwhile, other work supports that both prenatal substance use and proximal risk factors predict adolescent substance use (Day et al.,
Within the prenatal alcohol literature, there are consistent findings linking prenatal alcohol use to adolescent substance use (Dodge et al., 2009). Prenatal maternal alcohol consumption of three or more glasses has been found to be significantly associated with offspring alcohol use disorders between 14-21 years of age (Alati et al., 2006) and increased the likelihood of the adolescent consuming three or more glasses of alcohol at 14 years of age (Alati et al., 2008). In summary, studies examining substance use of a single substance are mostly supportive of the association between prenatal substance use and adolescent substance use.

However, persistent substance use in pregnancy often occurs in the context of multiple substances, making generalizability difficult when substances are each examined separately (Wells, 2009). Thus, indicators of polysubstance use may be more reflective of actual use patterns. In addition, prenatal substance use is often a marker of continued postnatal substance use that may be a proximal predictor of adolescent substance use via a number of different processes.

**Caregiver Postnatal Substance Use and Adolescent Substance Use**

Caregiver prenatal substance use often continues or increases after birth (Shisler et al., 2016; Tsantefski et al., 2014). Like prenatal substance use, postnatal substance use can also increase the risk of adolescent substance use (McDermott, 1984). For example, children of parents with alcohol use disorders have significantly higher risk of early initiation of alcohol and other substance use and greater frequency of use in adolescence (Eiden et al., 2016). One explanation for this association is the application of the Social Learning Theory (Bandura, 1969). It is theorized that within families with caregiver substance use, adolescents engage in numerous interactions with those who use substances and who have positive views on substance (Bahr et
Additionally, when a caregiver engages in substance use, there is more availability of substances. Taken together, the continued reinforcement of favorable substance use attitudes, combined with an access to substances, may result in an increase in adolescent substance use.

Although there is robust support in the literature of the association between caregiver substance use and adolescent substance use, most of the studies have been cross-sectional (McDermott, 1984). Additionally, the longitudinal studies that have examined caregiver postnatal substance use and adolescent substance use have typically been limited to timepoints throughout or around the adolescent period (Li et al., 2002). For example, a one-year study found that caregiver binge drinking significantly predicted adolescent alcohol initiation but this study did not include any measures of caregiver substance use prior to the year of the study (Rusby et al., 2018). Similarly, Hill et al (2005) found parental smoking to be associated with the initiation of daily smoking in adolescents aged 10-21 years, but the authors did not ask caregivers about prenatal or early childhood smoking habits. Although there are robust longitudinal studies on caregiver substance use, such as the Michigan Longitudinal Study, they began in early childhood and did not report on prenatal substance use (Buu et al., 2009). Taken together, there is clear support for the association of caregiver postnatal substance use and adolescent substance use; however, there is limited knowledge of how duration or chronic severity of prenatal to postnatal substance use may be associated with adolescent substance use.

Overall, there are limited number of studies examining the association of the chronicity of caregiver postnatal substance use and adolescent substance use. However, longitudinal studies that span from the prenatal to the adolescent period allow for multiple timepoint models to examine the association between substance use from prenatal-postnatal periods and adolescent substance use. For example, De Genna et al. (2022) not only found different patterns of change
over time in caregiver marijuana use in their longitudinal (pre-pregnancy to 16 years postpartum) study, but also found that these different trajectories predicted different levels of adolescent substance use. Results indicated for mothers who increased in marijuana use over time, adolescents had a significantly greater risk of developing a cannabis use disorder compared to adolescents whose mothers had chronic marijuana use (De Genna et al., 2022). Results also showed that adolescents with mothers who had little to no marijuana use over time had a significantly lower risk of using marijuana (De Genna et al., 2022). Likewise, a study examining the co-use of caregiver cigarettes and cannabis found that chronic co-use from pregnancy to 16 years postpartum was significantly associated with a higher risk for offspring substance use disorder by age 22 years (De Genna et al., 2017). This suggests that the cumulative exposure to caregiver substance use from birth to adolescence may be an important predictor of adolescent substance use.

**Role of Parental Monitoring on Adolescent Substance Use**

**Caregiver Postnatal Substance Use and Parental Monitoring**

Caregiver substance use may also have indirect effects on adolescent substance use via parenting processes. Among different aspects of parenting, one robust proximal predictor of adolescent substance use is parental monitoring (Eiden et al., 2016). Parental monitoring includes behaviors that facilitate parents’ knowledge of their children’s whereabouts and activities (Bornstein, 2002). These include the parenting knowing the adolescent’s peer group, being aware of where the adolescent spends free time, and knowing what activities the adolescent engages in (Yabiku et al., 2010). Caregiver substance use may interfere with parental monitoring, and indeed, higher rates of caregiver substance use have been linked to lower levels of parental monitoring (Chassin et al., 1996; Shorey et al., 2013). This relationship may be
explained due to high levels of caregiver substance use increasing the risk of negative family and personal consequences, which in turn decreases the effectiveness of parental monitoring strategies (Shorey et al., 2013). There is strong support in the literature for the association between higher caregiver postnatal substance use and lower levels of parental monitoring, which has been linked with higher adolescent substance use (Chassin et al., 1996).

**Parental Monitoring and Adolescent Substance Use**

High levels of parental monitoring are typically used to decrease the chance the child engages in unwanted activities (Racz & McMahon, 2011) and high levels of parental monitoring are predictive of less adolescent substance use (Dishion et al., 1999; Mills et al., 2021; Shorey et al., 2013; Yabiku et al., 2010). On the other hand, lower levels of parental monitoring are associated with a greater risk of adolescent substance use (Coley et al., 2008; Stice & Barrera, 1995; Walters, 2020). Results from a meta-analysis found strong support for parental monitoring as a proximal risk factor for adolescent marijuana use in cross-sectional and longitudinal studies (Lac & Crano, 2009). Additionally, lower levels of parental monitoring have been found to predict earlier adolescent alcohol initiation, increases in alcohol consumption, and a greater risk for developing alcohol misuse issues (DiClemente et al., 2001; Hayes & Australian Institute of Family Studies, 2004). A meta-analysis also supports a significant negative association between parental monitoring and delinquency (Hoeve et al., 2009) The mean effect size for parental monitoring and adolescent delinquency was strong (ESr = .23, ESr range −0.23 to −0.31, \( p < .001 \)), suggesting that parental monitoring is correlated with adolescent delinquency (Hoeve et al., 2009). Because parental monitoring is one of the most robust proximal predictors of adolescent substance use and caregiver substance use is associated with parental monitoring,
parental monitoring may partially mediate the association between caregiver and adolescent substance use (Latendresse et al., 2008).

In a study of 9th and 10th graders, parental monitoring was found to be a significant mediator between problematic caregiver substance use (adolescent reported) and adolescent alcohol use (defined as “more than a just a few sips”) (Finan et al., 2015; Shorey et al., 2013). Short-term longitudinal studies within families with caregiver substance use have also found support for lower levels of parental monitoring to be associated with higher adolescent substance use (Barnes et al., 2006). A longitudinal study from 13 to 17 ½ years of age found that parental monitoring significantly mediated the association between caregiver alcohol use and adolescent alcohol use (Latendresse et al., 2008). However, there are only a few long-term longitudinal studies that examine substance use from the prenatal period until adolescence. As such, there are limited studies that incorporate measures of caregiver substance use from pre-to-postnatal periods and include parental monitoring as a mediator of the association between caregiver substance use and adolescent substance use.

**Role of Peer Group Behaviors**

*Peer Group Behaviors*

Parents remain an important predictor of adolescent behavior, but throughout adolescence peers have an increasingly significant impact on adolescent behavior (Barnes et al., 2006). In addition, peer groups provide adolescence with a sense of identity, reinforcement of cultural norms, and stability (Sussman et al., 2007). Taken together, peer group behaviors are particularly predictive of adolescent behaviors due to the additive effects of peer and group influences. For instance, peer group delinquency is an especially salient predictor of adolescent substance use (Barnes et al., 2006; Dishion et al., 2002; Hawkins et al., 1992; Scalco et al., 2014). Peer group
delinquency may be a proximal risk factor for adolescent substance use due to a variety of peer processes, such as peer selection processes (adolescents with similarities have selected each other) and peer influences (adolescents engaged in behaviors), and availability of substances (Eiden et al., 2016).

Contrary to the robust literature on the association between peer group delinquency and adolescent substance use, there have been only a handful of studies on the impact of peer group prosocial behaviors. A recent longitudinal study concluded peer prosocial group behaviors were promotive of prosocial behaviors but did not find significant results to suggest peer group prosocial behaviors were protective against peer group delinquency (Walters, 2020). However, the majority of the studies on peer prosocial behaviors have found peer group prosocial behaviors to reduce the risk of substance use and delinquent behavior (Mason et al., 2019; Yabiku et al., 2010). Given the limited studies on the association between peer group prosocial behaviors and adolescent substance use, this thesis will build upon the existing work by examining the association between peer group prosocial behaviors and adolescent substance use in a longitudinal sample.

The Interaction of Parenting and Peers

However, peer group behaviors may not only be directly associated with adolescent substance use but peer group delinquency behaviors may also have additive or synergistic effects with low parental monitoring and prosocial peer behaviors may reduce the risk of low parental monitoring. There is a robust body of literature using longitudinal methods that provide support for both peer relationships and parenting practices to be predictive of adolescent substance use (Barnes et al., 2006) and for potential interactive effects of parenting and peers (Kiesner et al., 2010). However, little is known about how parental monitoring might protect against adolescent
engagement with delinquent peers, noted in earlier studies as an important next step (Barnes et al., 2006). Lower levels of parental monitoring are associated with an increase in unsupervised time, which may indicate that adolescents with lower levels of parental monitoring may be more vulnerable to peer influences. This pathway has been supported in longitudinal studies, which have found significant interactions between parental monitoring and peer delinquency when predicting adolescent alcohol misuse (Barnes et al., 2006).

On the other hand, high levels of parental monitoring have been associated with a reduced vulnerability to peer influences, such that higher levels of parental monitoring decreased the risk of the engagement with delinquent peers (Tornay et al., 2013). Studies have found support for a variety of ways that peer group behaviors, particularly peer group delinquency behaviors, interact with parental monitoring and adolescent substance use (Kiesner et al., 2010). The literature supports peer group behaviors as a mediator and peer group behaviors as a moderator of the association between parental monitoring and adolescent substance use. To build on these mixed results, this thesis will examine peer groups as both a mediator and a moderator of the association of parental monitoring and adolescent substance use within the same sample.

The Role of Biological Sex on Adolescent Substance Use

There has been support in the literature for biological sex differences regarding substance use. For example, women have been found to have an older age at onset of substance use compared to males and adult men have been found to engage in problematic alcohol use at higher rates than adult women (McHugh et al., 2018). A recent study examined patterns of substance use and found a unique pattern of substance use only among male adolescents and a second unique pattern of substance use only among female adolescents (Picoito et al., 2019). Among female adolescents, there was a group of adolescents who experiment with alcohol while
using tobacco frequently, and among male adolescents, there was a group of adolescents who had early initiation of substance use and then continued with polysubstance user (Picoito et al., 2019). Additionally, Picoito et al (2019) found different associations between these two unique groups and related adolescent measures, such as psychological symptoms, bullying, and parent-adolescent communication. Although this study was cross-sectional, it highlights the need to better understand different developmental pathways to male versus female adolescent substance use.

**Current Study**

I describe my conceptual model in described in detail below (see Figure 1). I propose to test the how peer group delinquency behaviors interact with parental monitoring, early adolescence substance use, and postnatal substance use. Additionally, associations of prenatal substance use and postnatal substance use on early adolescent substance use and the association of postnatal substance use on peer group delinquency behaviors was examined in the analytical model.

**Conceptual Model**

The conceptual model is consistent with research indicating higher caregiver substance use from prenatal period to early adolescence being associated with lower levels of parental monitoring (Chassin et al., 1996; Shorey et al., 2013; Hans, 2002), which in turn may provide more opportunities for engagement with peer groups with delinquent behaviors in early adolescence, increasing risk for late adolescent substance use (Kiesner et al., 2010; Mann et al., 2015). However, peer group delinquent behaviors may also co-occur with parental monitoring and adolescent substance use (Vaughan et al., 2022). Similarly, higher levels of parental monitoring have been associated with lower levels of adolescent substance use and lower levels
of peer group delinquency behaviors (Melotti et al., 2018). Additionally, peer group delinquency behaviors have been found to be highly correlated with adolescent substance use (Ferguson & Meehan, 2011). This may be explained by peer selection and influence processes; adolescents who use substances may seek out peer groups who also engage in substance use or adolescents within a peer group with substance use behaviors may be influenced to experiment and engage in substance use (Eiden et al., 2016).

Additionally, the proposed model will examine peer group delinquency behaviors as a moderator of the association between parental monitoring and LA substance use. Studies have found support for peer group delinquency behavior as a moderator of the association between parental monitoring and adolescent substance use (Kiesner et al., 2010). The interaction of parental monitoring and peer group delinquency has been shown to predict different rates of adolescent substance use. For example, Barnes et al. (2006) found that high peer delinquency in combination with low parental monitoring had the steepest rate of predicted alcohol misuse over time. Additionally, adolescents who engage in substance use and have substance using peers are more likely to receive reinforcement of the substance use behavior (Bahr et al., 2005). Because of this, I will also examine peer group delinquency behaviors as a moderator between the association of EA substance use and LA substance use.

**Hypothesis**

The conceptualized model described above reflects several hypothesized associations, which are described below.

1. I hypothesize that the association between prenatal substance use and late adolescent substance use will be mediated by continued postnatal substance use.
2. I also hypothesize that higher postnatal substance use will be associated with lower levels of EA parental monitoring, which in turn was associated with lower levels of LA substance use.

3. I hypothesize that higher postnatal substance use will be associated with higher levels of both EA substance use and EA peer group delinquency behaviors, which will each be associated with higher levels of LA substance use.

4. The model also includes hypothesized concurrent associations among the variables at EA as follows: there will be a significant association among all early adolescent variables, such that higher levels of parental monitoring will be associated with lower levels of EA substance use and EA peer group delinquency behaviors, and higher EA substance use will be associated with higher peer group delinquency behaviors.

5. I also hypothesize that peer group delinquency behaviors will moderate the association between EA variables and LA substance use. Among adolescents with moderate to high peer group delinquency behaviors, there will be a stronger association between the variables at EA (in the expected directions hypothesized as main effects) and LA substance use. Among adolescents with no to little peer group delinquency behaviors, I hypothesize there will be a weaker association between the variables at EA and LA substance use.
Figure 1

*Conceptual Model*

![Diagram showing the relationship between parental monitoring, peer group delinquency, and late adolescent substance use.]

**Figure 1**: Conceptual model. EA: early adolescence; LA: late adolescence.
METHODS

Participants

The sample consisted of 216 mother-child dyads who participated in an ongoing longitudinal study to follow the dyads from birth to late adolescence. Mothers were recruited from two hospitals, which mainly served a diverse, low-income urban population. To be eligible for the study, mothers had to be 18 years of age or older, have no prenatal illicit substance use other than cocaine or marijuana, no multiple pregnancy, and no major medical concerns in the infant. Approximately 4,800 mothers completed a screening interview after delivery. There were 340 eligible infant-mother dyads, 119 of which were enrolled into the prenatal cocaine-exposed (PCE) group. The PCE group included mothers who were positive for prenatal cocaine use on any indicator (see Measures). After a dyad was recruited and enrolled in the PCE group, a demographically similar non-cocaine exposed infant-mother dyad was recruited into the non-cocaine-exposed (NCE) group. The NCE group included 101 dyads matched on infant sex at birth, maternal education, and maternal race/ethnicity. Biological sex at birth was gathered at the first assessment from medical records and 49% of the sample was assigned male at birth. After the exclusion of 4 dyads due to infant medical problems, the final sample was 216 (116 PCE, 100 NCE, 106 male). A flowchart of this recruitment process is depicted in Figure 4.

Biological mothers ranged from 18 to 42 years old ($M = 29.53$, $SD = 6.06$) at the time of recruitment and about 72% of the mothers identified as Black. At the time of recruitment, 66% of mothers had never been married, 13% were married and 20% were divorced, separated, or widowed. Mothers ranged from completing 8 to 18 years of education ($M = 11.80$, $SD = .13$), with 71% of mothers reporting their education as high school or below. Approximately 76% of participants received Temporary Assistance for Needy Families by the time of the first visit
(years 2001-2004). Additionally at the time of recruitment, 27% of participants were unemployed and 69% were receiving Medicaid. During the EA visit, the youth was asked to self-identity their race/ethnicity and 57.2% of the youths identified as Black, 17.8% identified as Mixed Race, 8.6% identified as Hispanic, 6.6% identified as White, 2.0% identified as American Indian, 0.7% identified as Asian, and 7.1% identified as other. The institutions of the primary investigators and the hospitals involved in the study all received institutional review board approval. All enrolled participants completed informed written consent. After April 2003, all participants provided HIPAA authorization.

Eligible biological mothers who did not enroll in the study were more likely than those who did enroll to have children placed out of maternal care. When compared to mothers who were eligible for the study but did not enroll, mothers who enrolled were more likely to be within the age range of 18 to 25 years old during recruitment ($p < .001$) and were also more likely to have a high school or lower education ($p < .001$). There were no other significant differences on demographic variables for those eligible and enrolled in the PCE group and those eligible for the PCE group but did not enroll.

**Procedure**

Participating mothers completed the 1 month assessment when the infant was around 4-8 weeks old. If there was a change in custody in a dyad, the legal guardian was located and consented. Biological mothers were interviewed to obtain prenatal substance use information at the first assessment, in addition to the primary caregiver under circumstances of custody change.

Follow-up assessments took place every 6 months until the early school age ($M = 5.52$ years, $SD = 0.36$) and again during middle childhood ($2^{nd}$ grade). The last two assessments took place in EA ($M = 13.26$ years, $SD = 0.82$) and LA ($M = 14.90$, $SD = 0.87$). The compensation for
study assessments increased in value over time and included small gifts, checks, and gift cards.

For a detailed description of the recruitment methods and procedures, please see Eiden et al. (2015).

**Figure 2**

*Recruitment Flowchart*

![Recruitment Flowchart](image)

*Note.* The recruitment to enrollment flowchart of the study. Women who did not complete the first assessment within the 1 month timeframe were kept in the study.

**Measures**

*Caregiver Substance Use*

Substance use during pregnancy was measured using multiple methods including a screening questionnaire after delivery, the Timeline Followback interview (TLFB; Sobell & Sobell, 1992) at each assessment, maternal and infant urine assays, and maternal hair assays after delivery (Eiden et al., 2015). Maternal and infant urine collected by the hospital at delivery were assayed for cocaine, benzodiazepines, opiates, and tetrahydrocannabinol and these data were extracted from the medical records. Results from urine assays were marked as positive if any
drug or metabolite was greater than 300g/ml (Eiden et al., 2014). Maternal hair samples were assayed for cocaine use; after an initial screening, gas chromatography/mass spectrometry was used as confirmation for positive cocaine use. Caregiver report of substance use was assessed with a trained interviewer administering the TLFB. The TLFB is an established reliable and valid method to capture substance use over time, substance use patterns, and has been shown to be reliable to other measures of self-report. (Brown et al., 1998).

During assessment, biological mothers were provided with a calendar that covered the duration of their pregnancy and 1 month postpartum. During the postnatal assessments, caregivers were provided a calendar that covered the previous 6 months. To aid in recall, caregivers were asked to first identify days within the duration of the calendar that personal events, such as birthdays, vacations, and holidays, occurred. The TLFB data provided the average number of days of cocaine use per week, the average number of cigarettes smoked per week, and the average number of standard drinks consumed per week.

Following previous studies (Conradt et al., 2014), a polysubstance use count variable was created for prenatal substance use. Each substance (cocaine, cigarettes, marijuana, and alcohol) was dichotomized into a dummy-coded variable. Cocaine use was coded as positive (1) if mothers self-reported cocaine use during the TLFB or if maternal hair, maternal urine, or infant urine were positive for cocaine use. Marijuana use was coded as positive (1) if the mother self-reported marijuana use during the TLFB. Cigarette use was coded as positive (1) if the mother self-reported cigarette use during the TLFB. Prenatal alcohol use was coded as positive (1) if the mother self-reported any alcohol use during the TLFB. Based on these assessments, 53.7% of mothers had prenatal cocaine use, 31% had prenatal marijuana use, 45.8% had prenatal alcohol
use, and 56.9% had prenatal cigarette use. The final prenatal substance use variable was created by taking the count of the four dummy-coded risk variables.

We did not have any biomarker assessments of postnatal substance use. To maintain both the rank-order of participants and potential dose-response effects of postnatal substance use, a cumulative risk variable was created from proportion scores based on maternal self-reports alone (Moran et al., 2017). The proportion scores was computed by transforming continuous variables (e.g., number of days used cocaine, number of standard drinks/week) into proportions by dividing the participant’s substance use by the maximum value of each substance in the sample. For each timepoint, a proportion score of 0 reflected no use of the substance and a score of 1 reflected the maximum use (relative to the sample) of the substance. Thus, the rank-order in the amount of substance use across participants was maintained in addition to reflecting severity of substance use.

First, proportion scores of the four substances were created within each timepoint. Postnatal caregiver cocaine use (number of days cocaine was used) was divided by the maximum number of days cocaine was used to create a proportion score. Postnatal caregiver marijuana use (number of days marijuana was used) was divided by the maximum number of days marijuana was used to create a proportion score. Postnatal caregiver alcohol use (average number of standard drinks per day) was divided by the maximum alcohol use to create a proportion score. Postnatal caregiver cigarette use (average cigarette use per day) was divided by the maximum cigarette use to create a proportion score. The sum of the four proportion scores (potential range: 0-4) was calculated to create a timepoint risk variable. The final cumulative postnatal substance use risk variable was created by computing the average of all timepoints to reflect cumulative caregiver substance use from birth to EA.
**Parental Monitoring**

Parental monitoring was assessed with child reported data at EA. The 9-item report measured the degree to which caregivers had knowledge of their child’s acquaintances, behaviors, and locations (Grundy et al., 2007; Sturge-Apple et al., 2003). The items included “how often does your parent know what you do with your free time?” and “how often does your parent know who your friends are?” Each question was answered on a scale of 1 to 5, with 1 being “never” and 5 being “always.” The score used in analysis was the mean of the 9 items computed by taking the average of all items \( M = 3.98, SD = .81 \) and higher scores indicate higher parental monitoring. This measure had an internal consistency of .85, indicating adequate reliability.

**Peer Group Delinquency Behaviors**

Adolescent report on the Peer Delinquency Scale (Loeber et al., 1998) was used to measure peer group delinquency behaviors in EA. Each question was given a scale of 1 to 5, with 1 being “none of them” and 5 being “all of them.” The questions asked over the past six months how many of the adolescent’s friends engaged in each behavior, such as “skipped school without an excuse?” and “stolen something worth more than $100?” The peer delinquency score was created by taking the mean of the 12 items \( M = 1.23, SD = .35 \), with higher scores reflecting more peers with delinquent behaviors. This measure had an internal consistency of .85 in this sample, indicating adequate reliability. For the multigroup analysis to examine moderation by peer delinquency, the original variable was dichotomized into no to low peer group delinquency (0, termed low delinquency) and moderate to high peer group delinquency (1, termed high delinquency). No to low peer group delinquency status indicated the adolescent reported either “none of them” to each of the 12 items (an average score of 1) or “none of them”
to 11 questions and a “few of them” for only one item (an average score of 1.08). High peer
group delinquency status indicated the adolescent reported a “few of them” or higher for two or
more items on the scale (an average score of 1.17 or higher). Therefore, the cut-off value for
dichotomization was 1.09, which was below the mean (\(M = 1.23\)) and the median (1.17) but
resulted in a more meaningful interpretation. This also allowed the mode (1.08), which
represented answering “a few of them” to only one item, to be included within the no to low peer
group delinquency group.

*Adolescent Substance Use*

Adolescent alcohol and cigarette use was assessed in EA and LA with a self-reported
survey based on the Youth Risk Behavior Surveillance Survey (Grunbaum et al., 2001). The
following questions were used in the current thesis. “During the past 30 days, on how many days
did you have more than a few sips of beer, wine, or liquor?” “During the past 30 days, how many
days did you use smokeless (chewing) tobacco?” “During the past 30 days, on how many days
did you smoke cigarettes?” “During the last 30 days, on about how many days did you use an
electronic or e-cigarette?” “During the last 30 days, on how many occasions (if any) have you
used marijuana (weed, pot) or hashish (hash, hash oil)”? Other substance use was measured with
20 items assessing other substance use in the past month (e.g., heroin, cocaine) and response
options had a range from 1 to 7, with 1 being “never” and 7 being “40 or more times” (Substance
Abuse and Mental Health Services Administration, 2007). In addition, adolescent hair samples
were collected and sent to Omega Laboratories for testing of cocaine, marijuana, opioids,
methamphetamine, and phencyclidine. First, a screening analysis was done that checks for drug
use on a small portion of the hair sample. The hair samples were screened with an enzyme-linked
immunosorbent assay methodology, which has been reported as a reliable method for drug
testing. If a sample had a positive screen, the sample was examined for the corresponding metabolites via chromatography/mass spectrometry testing. The combination of a positive screening (positive for the parent drug) and confirmation (positive for the corresponding metabolites and if no metabolites of the drug, then positive for the drug a second time) resulted in a positive drug screening. Omega Laboratories followed the standard industry cut-off levels, which were modeled after the thresholds proposed in the 2004 Substance Abuse and Mental Health Services Administration guidelines. Hair analysis can measure substance use as soon as 5-10 days after use. Since the average rate of hair growth is ½ inch per month, a 2 inch hair sample can identify substance use from the past 4 months (Eiden et al., 2014). Hair analysis completed by Omega Laboratories did not produce a positive screen from an environmental exposure to a drug, as metabolite are an internal by-product of drug use and cannot be produced from the environment.

In EA, the frequencies of past 30 day substance use was as follows: 1.9% tobacco, 4.4% alcohol, 10.6% marijuana, 2.0% stimulants, .6% tranquilizers, 2.0% cough medicine, and 17.2% cocaine. Overall in EA, 79.2% of adolescents had no substance use in the past 30 days, 16.4% used one substance, 2.5% used two substances, and 1.9% used 3 three substances. In LA, the frequencies of past 30 day substance use was as follows: 12.7% tobacco, 5.% alcohol, 31.5% marijuana, 2.5% stimulants, .7% (sniffing) glue, 2.1% cough medicine, 11.5% cocaine, and .7% bath salts. Overall in LA, 66.4% of adolescents had no substance use in the past 30 days, 22.4% used one substance, 7.7% used two substances, 2.8% used 3 substances, and .7% used four substances. Because of the low base rates for individual substance use and the corresponding zero-inflated count variable, I create a dichotomized variable for all substances (0 = no use of the substance in the past 30 days; 1 = positive hair sample or any self-reported use of the substance).
Tobacco products (cigarettes, e-cigarettes, and chewing tobacco) were counted as one substance. Next, I created a dummy coded variable for each timepoint (0 = no use of any substances in the past 30 days; 1 = positive hair sample or self-report of one or more substances). In EA, 20.8% (N = 33) of adolescents used at least one substance and in LA, 33.6% (N = 48) of adolescents used at least one substance.

**Analytical Plan**

**Figure 3**

*Analytic Model*

![Analytic Model Diagram]

*Note.* The analytical model. Moderation by peer group delinquency was tested through multigroup analysis. EA: early adolescence, LA: late adolescence, SU: substance use. Not displayed in the figure but included in model testing were the within time associations among the EA variables.

First, descriptive statistics for demographics and the variables in the model were examined. Next, path analysis with weighted least squares-means and variance adjusted (WLSMV; *Mplus* Version 8.8) was used to test the conceptual model in Figure 1. This analytic
model is depicted in Figure 5. All path analysis was conducted in Mplus Version 8.8 (Muthen & Muthen, 1998). The model will include child biological sex at birth as a dummy-coded (males = 0) control variable for EA and LA substance use. The analytic model was tested in two steps. First, I examined the hypothesized main effects model. Second, I conducted multiple group analyses examining the role of peer delinquency as a potential moderator of EA to LA associations. Finally, I conducted multiple group analyses with child sex as the moderator to examine if the causal paths from prenatal and postnatal substance use to LA substance use differed for boys and girls. Given the categorical outcome variable of EA and LA substance use, I used the WLSMV estimator to examine model fit of the main effects model (Asparouhov & Muthen, 2006). I used the DIFFTEST function in Mplus to compare the unconstrained with constrained models in the multigroup analysis. The DIFFTEST function also utilized the adjusted $\chi^2$ value and is the recommended method for nested tests that use a robust weighted least squares estimator (Asparouhov & Muthen, 2006; Muthen & Muthen, 1998; Suh, 2015).

**Power Analysis**

For the overall model, a power analysis was conducted using G*Power version 3.1.9.7 (Faul et al., 2007) examining the power to detect small to large regression parameters in the path analysis. I first examined power to detect main effects with effect sizes ranging from small/medium to large with our given sample size of 216. Power calculations were conducted with a sample size of 216, and small ($f^2 = .02$), medium ($f^2 = .15$), and large ($f^2 = .35$) effect sizes, at a significance criterion of $\alpha = .05$. Power to detect small effects was low (power = .23). However, power to detect medium or large effects was adequate (greater than .90). A post-hoc power analysis done retrospectively, as opposed to prospectively for a study aiming to replicate previous work, will not identify if a lack of significance was due to low power or due to a small
effect since the post-hoc power analysis is a function of the tested $p$-value (Lenth, 2007). Based on a $p$-value cut-off of .05, 10 degrees of freedom, and a two-tailed test, the highest power calculated from a post-hoc analysis of a non-significant main effect would be .52 (Lenth, 2007). However, the table provided by Lenth (2007) also indicated that a $p$-value of .01 would reflect a post-hoc power analysis of .81 and a $p$-value of .001 would reflect a post-hoc power analysis of .98.

For the moderation effects, a second power analysis was conducted using G*Power version 3.1.9.7 (Faul et al., 2007) to determine power to detect interaction effect sizes ranging from small to large to test the study hypotheses. I examined the necessary sample size for adequate power (.80) with three predictors (independent variable, the moderator, and the interaction term). A-priori power calculations were conducted for small ($f^2 = .02$), medium ($f^2 = .15$), and large ($f^2 = .35$) effect sizes, and at a significance criterion of $\alpha = .05$ for power of .80. Power to detect small effects required a sample size greater than was available in this study ($N = 550$). However, the required sample size to detect medium or large effects was adequate ($N$ of at least 77 participants). A post-hoc power analysis for non-significant interactions was not conducted, as when study outcomes and variable correlations have already been observed, the post-hoc power analysis does not result in the actual power to detect statistical significance (Baranger et al., 2022; Zhang et al., 2019).
RESULTS

Descriptive Statistics

The descriptive statistics of the continuous and categorical study variables can be found in Table 1. Adolescents with high prenatal substance use also had higher rates of postnatal substance use. Female adolescents ($M_{female} = .27, SD_{female} = .45$) were more likely to use substances in EA than male adolescents ($M_{male} = .14, SD_{male} = .35, p < .001$). Female adolescents were also more likely to use substances in LA ($M_{female} = .42, SD_{female} = .50$) than male adolescents ($M_{male} = .25, SD_{male} = .44, p < .001$). Substance use in EA was significantly associated with substance use in LA, $X^2 (1, N = 134) = 9.69, p = .002$. Bivariate correlations indicated that higher peer group delinquency was associated with lower levels of parental monitoring and with substance use in EA. There were no significant bivariate correlations between any of the other EA variables and LA substance use. Higher prenatal substance use was associated with non-biological parent care at any time point between birth and early school age. The correlations among study variables are displayed in Table 2.

Missing data

There was a significant association between a report of non-biological parent care placement and missing data in early adolescence ($p = .02$) and in late adolescence ($p < .001$). Of the 64 participants who were in non-biological parent care, 24 (37.50%) had missing data in early adolescence and 33 (51.56%) had missing data in late adolescence. However, there were no associations between non-biological care status and late adolescent substance use ($p = .49$). There were no other significant group differences between adolescents with missing versus complete data on study variables or on demographic variables. Given these differences, non
biological care status was included as an covariate in model testing and the data met criteria for being missing at random (Rubin et al., 2007).

**Table 1**

*Descriptive Statistics of Study Variable*

<table>
<thead>
<tr>
<th>Continuous Variables</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
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<tr>
<td>Postnatal Substance Use</td>
<td>0.25</td>
<td>0.06</td>
</tr>
<tr>
<td>EA Parental Monitoring</td>
<td>3.98</td>
<td>0.64</td>
</tr>
<tr>
<td>EA Peer Group Delinquency</td>
<td>1.1</td>
<td>0.02</td>
</tr>
<tr>
<td>Prenatal Substance Use</td>
<td>1.86</td>
<td>1.63</td>
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</table>

<table>
<thead>
<tr>
<th>Categorical Variables</th>
<th>Percent (Group = 0)</th>
<th>Percent (Group = 1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EA SU</td>
<td>79.2% (No SU)</td>
<td>20.8% (SU)</td>
</tr>
<tr>
<td>LA SU</td>
<td>66.4% (No SU)</td>
<td>33.6% (SU)</td>
</tr>
<tr>
<td>Non-Biologic Care Placement</td>
<td>70.4% (No)</td>
<td>29.6% (Yes)</td>
</tr>
<tr>
<td>Sex at Birth</td>
<td>49.1% (Male)</td>
<td>50.9% (Female)</td>
</tr>
</tbody>
</table>

*Note.* Means and standard deviations reported for continuous study variables. For categorical study variables, the percentage of participants in each group is reported.

**Table 2**

*Correlations Among Study Variables*

<table>
<thead>
<tr>
<th>Variables</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
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<td>1. NBCP (0 = No)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Prenatal Substance Use</td>
<td>.21**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Postnatal Substance Use</td>
<td>-.13</td>
<td>.50***</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. EA Parental Monitoring</td>
<td>.06</td>
<td>-.09</td>
<td>.11</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. EA Peer Group Delinquency</td>
<td>.01</td>
<td>.001</td>
<td>&lt;.001</td>
<td>-.29***</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. EA SU (0 = No SU)</td>
<td>-.20**</td>
<td>.13</td>
<td>.22**</td>
<td>.22**</td>
<td>.22**</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>7. LA SU (0 = No SU)</td>
<td>.09</td>
<td>.04</td>
<td>.03</td>
<td>-.16</td>
<td>.18</td>
<td>.42***</td>
<td>1</td>
</tr>
</tbody>
</table>

*Note.* EA: Early Adolescence; LA: Late Adolescence; SU: Substance Use; NBCP: Non-biologic care placement. *** = \( p < .001 \), ** = \( p < .01 \), \( p < .05 \).
Model Testing

The analytic model included casual paths from prenatal substance use to postnatal substance use and postnatal substance use to EA substance use, EA peer delinquency, and EA parental monitoring. The analytic model also included causal paths from the EA variables to LA substance use. The model included the hypothesized direct paths from prenatal substance use to LA substance use and from postnatal substance use to LA substance use. Additionally, the covariance was specified for EA parental monitoring and EA substance use, EA parental monitoring and EA peer group delinquency behaviors, and EA substance use and EA peer group delinquency behaviors. After preliminary analysis identified non-biologic care a predictor of missingness and its significant correlation with prenatal substance use, the analytical model specified a causal path from prenatal substance use to non-biologic care placement. After exploratory analysis indicated significant sex differences within adolescent substance use, the sex at birth was added to the model to control for sex differences.

Results indicated this model fit the data well, $\chi^2(113) = 9.85, p = .71$, RMSEA = $.001$, 90% CI [.001, .05], CFI = 1.00, TLI = 1.00. This model is depicted in Figure 6. Results indicated that there was stability from prenatal to postnatal substance use ($b = .50, p < .001$). However, neither prenatal ($b = .03, p = .82$) nor postnatal ($b = .20, p = .11$) substance use were predictive of EA substance use. Results also showed that prenatal ($b = .02, p = .86$) and postnatal ($b = -.08, p = .60$) substance use were not predictive of LA substance use. Contrary to hypotheses, postnatal substance use was also not a significant predictor of EA parental monitoring ($b = .05, p = .68$), or EA peer group delinquency behaviors ($b = .01, p = .97$). Sex at birth was a significant predictor of EA substance use ($p = .04$). The probability of a male engaging in substance use at EA was 7.35% and the probability of a female engaging in
substance use at EA was 17.06%. Sex at birth was not a significant predictor of LA substance use ($p = .23$), in the context of all other variables in the model. EA substance use was a significant predictor of LA substance use ($b = .45, p = .004$). The probability of an adolescent who did not use substances in EA engaging in LA substance use was 19.77% and the probability of an adolescent who used substances in EA engaging in LA substance use was 32.64%. Neither EA parental monitoring ($p = .70$) nor EA peer group delinquency behaviors ($p = .08$) were significant predictors of LA substance use. Prenatal substance use was a significant predictor of non-biologic care placement ($p = .04$). The covariance among the residuals of all EA variables was significant. Both lower parental monitoring in EA and higher EA peer group delinquency behaviors were associated with substance use in EA. Lastly, higher peer group delinquency behaviors were associated with lower levels of parental monitoring.

**Figure 6**

*Hypothesized Main Effects Model*
Note. The figure shows the path results from the final model. The standardized coefficient of each pathway is reported. The black lines represent significant pathways and the gray lines represent non-significant pathways. EA: Early Adolescence; LA: Late Adolescence; SU: Substance Use.

*p < .05. **p < .01. ***p < .001.

**Moderation by Peer Group Delinquency**

Multiple group analysis with peer group delinquency as a moderator indicated a significant difference in the path from postnatal substance use to LA substance use. The model constraining the path from postnatal substance use to LA substance use was a significantly worse fit than the fully unconstrained model, $\Delta \chi^2(1) = 8.23, p = .004$. Among those with low EA peer group delinquency, lower postnatal substance use was associated with LA substance use ($b = -.58, p = .01$). Among those with high EA peer group delinquency, there was no significant association between postnatal substance use and LA substance use ($b = .21, p = .24$). There were no other significant differences across levels of peer group delinquency behaviors.

**Exploratory Analyses with Sex at Birth**

I conducted a second multigroup analysis to examine sex differences. The association between prenatal substance use and LA substance use was significantly different for males versus females. The model constraining the path from prenatal substance use to LA substance use was a significantly worse fit than the fully unconstrained model, $\Delta \chi^2(1) = 4.71, p = .03$. Among males, higher prenatal substance use was associated with LA substance use ($b = .43, p = .04$). Among females, prenatal substance use was not associated with LA substance use ($b = -.20, p = .25$). The association between postnatal substance use and LA substance use was also significantly different for boys versus girls. The model constraining the path from postnatal
substance use to LA substance use was a significantly worse fit than the fully unconstrained model, $\Delta \chi^2(1) = 5.18, p = .03$. However, postnatal substance use was marginally associated with LA substance use among boys ($b = -.58, p = .06$) and not associated among girls ($b = .22, p = .22$).

There was also a sex difference in the association of parental monitoring and peer group delinquency in EA. The model constraining the covariance between EA parental monitoring and EA peer group delinquency behaviors was a significantly worse fit than the fully unconstrained model, $\Delta \chi^2(1) = 8.12, p = .004$. Among females, there was a significant covariance between the residuals of EA parental monitoring and EA peer group delinquency ($b = -.43, p < .001$) indicating that among females, higher parental monitoring was associated with lower peer group delinquency behaviors in EA. However, among males, this covariance was non-significant, ($b = -.07, p = .48$). There were no other significant sex differences.
DISCUSSION

The main goal of this study was to examine the association between caregiver substance use and late adolescent substance use via parental monitoring and peer processes, and to examine the role of peer group delinquency as a potential moderator of these associations. I examined the fit of a conceptual model beginning from the prenatal substance use to postnatal substance use and examined pathways from postnatal substance use to LA substance use substance use through EA peer group delinquency behaviors, EA parental monitoring, and EA substance use, followed by examination of peer group delinquency as a moderator of these associations. Finally, an exploratory goal was to examine if the hypothesized associations were different based on sex at birth.

Caregiver substance use to Adolescent Substance Use

Although the conceptual model fit the data well, results were largely unsupportive of an association between caregiver substance use and adolescent substance use either directly or indirectly via parental monitoring for the sample as a whole, as there were sex differences. However, results were supportive of the path from prenatal substance use to postnatal substance use. This finding supported my hypothesis that prenatal substance use is a marker for continued postnatal substance use. The literature shows that tobacco, alcohol, and marijuana all have high rates of continued use from the prenatal to postnatal periods (Eiden et al., 2023). Although I utilized a count measure for prenatal substance use and a proportion score reflecting severity of substance use as the measure of postnatal substance use, results were similar to a previous study that dummy-coded substance use (0 = no substance use, 1 = substance use) and found maternal prenatal substance use significantly predicted maternal postnatal substance use (Smith et al., 2007). Similarly, a recent study found a significant correlation between prenatal polysubstance use and...
use and postnatal polysubstance (Bierce et al., 2023). Although my results are reflective of a count measure of prenatal substance use, other studies examining both individual substances and their trajectories have found similar results. For example, a study examining the average time to substance use abstinence in pregnancy found significant relapse rates for alcohol, cocaine, marijuana, and tobacco (Forray & Foster, 2015). Prenatal abstinence rates for tobacco use were found to be lower, possibly due to cigarettes being substituted for other substances (Forray & Foster, 2015). Further research examining pre- to postnatal substance use has found that although most women decrease substance use during pregnancy, there were significant increases in tobacco use by 9 months postpartum (Shisler et al., 2016). Another study found prenatal alcohol, tobacco, and marijuana use to each be significantly correlated with postnatal alcohol, tobacco, and marijuana use (Eiden et al., 2020).

Contrary to hypothesis, prenatal substance use did not predict EA substance use. The literature on the association between prenatal substance use and adolescent substance has been small, but most studies indicate a significant association (Glantz & Chambers, 2006). It is possible that my non-significant results were due to aggregating prenatal substance use by time and substance. For example, first-trimester prenatal cocaine use and family alcohol use was found to be a significant predictor of adolescent marijuana and alcohol use, whereas adolescent tobacco use was predicted by current maternal tobacco use (Richardson et al., 2013). However, when controlling for adolescent mental health, adolescent peer substance use, and neighborhood characteristics, prenatal cocaine use was not found to be a significant predictor of adolescent substance use (Warner et al., 2011). A third study reported no significant association between prenatal cocaine use and any adolescent substance use, however there were significant relationships between prenatal cocaine use and adolescent marijuana and tobacco use (Lester et
al., 2012). A large portion of the literature has only examined a single substance or examined multiple substances individually, as Lester et al (2012) did. A further examination into measurement differences indicates that strong family relationships may weaken the association between prenatal substance use and adolescent substance use and that prenatal marijuana use may specifically predict adolescent marijuana and tobacco use. (Glantz & Chambers, 2006). Instead of creating a measure of prenatal polysubstance use, most studies have examined the relationship between adolescent substance use and prenatal alcohol, marijuana, tobacco, and cocaine use separately, such as in Richardson et al (2013). Thus, it is difficult to generalize the findings of these studies. Although my measure of prenatal substance use reflected polysubstance use, it did not reflect timing of prenatal substance use nor the quantity of each substance.

Contrary to my expectations, neither prenatal nor postnatal substance use significantly predicted LA substance use. Therefore, my hypothesis of postnatal substance use mediating the relationship between prenatal substance use and LA substance use was not supported. Also contrary to my hypothesis, results suggested that postnatal substance use did not predict EA substance use. Although the association between postnatal substance use and EA substance use was significant at the bivariate level, when controlling for prenatal substance use, postnatal substance use did not account for significant variance in EA substance use. This could be a result of my measure of postnatal substance use being computed as an average across birth to EA. Most of the literature has found postnatal substance use to predict adolescent substance use, however most of longitudinal literature has been limited to the adolescent years. For example, Hill et al (2005) found that parental smoking during adolescence predicted adolescent smoking from the ages of 10 to 21 years old. Another longitudinal study recruited participants who ranged from 7th
to 10th grade and examined adolescent alcohol use at both baseline and 5 years later, but parental alcohol use was only measured at the baseline visit (Brook et al., 2010). Although the Brook et al (2010) study was longitudinal, the significant association between parental and adolescent alcohol use was cross-sectional. Rusby et al (2018) followed adolescents during the transition to high school and found parental marijuana use predicted adolescent binge drinking and parental binge drinking predicted both adolescent alcohol and marijuana initiation. However, Rubsy et al (2018) only examined current parental substance use. Similarly, a study examining the transition to middle school found parental substance use at baseline to be associated with adolescent substance use 18 months later (Li et al., 2002). However, the study by Li et al (2002) measured parental substance use differently and categorized the variable based on the adolescent having one parent who used substances, two parents who used substances, or no parents who used substances. Therefore, it is possible that my non-significant association between postnatal substance use and adolescent substance use is due to examining postnatal substance use from birth to adolescence instead of examining postnatal substance use concurrently, during adolescence. In addition, an aggregate measure of cumulative substance use to caregiver postnatal substance use may not capture changes over time in caregiver substance use, severity of substance use to any one substance (e.g., binge drinking), or the context of use that may affect child exposure to substance use. Recent work has found different patterns of caregiver substance use over time and that these different patterns of use significantly predict adolescent substance use (De Genna et al., 2015, 2017, 2022). De Genna et al (2022) found that children of chronic marijuana users were more likely to both use marijuana and have a Cannabis Use Disorder than children of non marijuana users. Mothers who increased in marijuana use over time had adult children with a greater risk of Cannabis Use Disorder than children of chronic marijuana use.
(De Genna et al., 2022). Adolescents whose mothers either quit tobacco use postpartum or increased smoking over time were more likely to engage in cigarette use when compared to adolescents whose mothers did not use cigarettes (De Genna et al., 2016). However with my methods, high levels of postnatal substance use could either indicate high levels of substance use continually from birth to EA or indicate extremely high levels in early childhood with lower levels in adolescence. Alternatively, low levels of postnatal substance use could indicate low levels throughout the postnatal period or indicate low levels overall with a high level of substance use only in EA.

Additionally, results supported the hypothesis that engaging in EA substance use predicts engaging in LA substance use. This has been replicated in other studies, for example within a sample of middle and high school students, adolescent alcohol use significantly predicted adolescent alcohol use 5 years later (Brook et al., 2010). Longitudinal work has found consistent support for an increase in substance use across adolescence (Derefinko et al., 2016) and Chassin et al. (2004) found past year use substance use to increase from early adolescence to late adolescence. Although the current study had a higher number of adolescents using substances in LA (N = 48) compared to in EA (N = 33), overall the percentage of those using substances was smaller compared to national samples. For example the Monitoring the Future study reported that approximately 20% of 8th graders engaged in alcohol use in the past 30 days, whereas almost 50% of 12th graders engaged in alcohol use in the past 30 days (Johnson et al., 2003). Monitoring the Future found similar increasing trends for other substances, such that around 20% of 8th grades used cigarettes in the past 30 days whereas 27% of 12th graders used cigarettes in the past 30 days (Johnson et al., 2003).
**Parental Monitoring**

Contrary to my hypothesis, I did not find postnatal substance use to be associated with EA parental monitoring and parent monitoring was not predictive of LA substance use, although higher parent monitoring was associated with a lower likelihood of EA substance use. However, it is possible that differences in the measurement of parental monitoring may explain this result. For example, parental monitoring only consisted of three questions in the study by Shorey et al (2013). Another study that examined parental monitoring within families with parental substance use found that parental control may be more protective against maldaptive behavior than parental warmth or parent-child relationship (Suchman et al., 2007). Suchman et al (2007) found that among mothers who used substances, higher levels of parental control was associated with lower levels of child externalizing behaviors. Specifically, higher parental control was defined as having an easier time imposing rules without being intrusive to the child (Suchman et al., 2007). My selected measure of parental monitoring may instead reflect the general knowledge aspect of parental monitoring and may not reflect, or correlate with, measures of parental control.

Parental monitoring was associated with EA substance use, but when controlling for EA substance use, parental monitoring did not account for additional variance in LA substance use. These results support findings that lower levels of parental monitoring and higher levels of adolescent substance use co-occur (Vaughan et al., 2022). It is possible that parental monitoring in LA may have had the expected co-occurring association, but it is also possible parental monitoring may predict substance use initiation instead of current use. For example, a study completed by Barnes et al (2006) had adolescents report on parental monitoring at multiple
timepoints. Barnes et al (2006) found parental monitoring to be a significant predictor of initial levels of adolescent substance use but not of its trajectories.

This may also be explained by differences in sample characteristics. For example, Shorey et al (2013) found parental monitoring to be a significant predictor of adolescent substance use, but only 30% of the participants in the study identified as Black compared to 72% in the current study. Black families are often part of extended family networks, comprised of biological and non-biological members, that support each other (Wallace & Fisher, 2007). It is possible that adolescents in the current study had an adult within their extended family network they turned to for parental involvement, which could explain the lack of association between postnatal substance use and EA parental monitoring. The study instructed adolescents to answer the parental monitoring questionnaire about the adult they feel closest to, but this may have been different than the caregiver who was engaging in substance use. This could also explain why, contrary to my hypothesis, EA parental monitoring was not associated with LA substance use. Additionally, a social system framework explains that Black adolescents and their parents are likely to be very aware of the higher rates of substance use related arrests for Black Americans compared to White Americans, which may lead to lower rates of substance use among Black adolescents (Terling Watt & McCoy Rogers, 2007).

**EA Peer Group Delinquency Behaviors**

In contrast to my hypothesis, postnatal substance use was not associated with EA peer group delinquency behaviors and EA peer group delinquency behaviors were not found to be predictive of LA substance use. A past study concluded that due to the comparatively lower levels of substance use in Black adolescents, compared to other racial groups, EA peer group delinquency behaviors may not be a strong predictor of LA substance use for Black adolescents.
A comprehensive narrative review acknowledged that racial differences have not been included in most studies on peer delinquency behaviors and adolescent substance use (Hoeben et al., 2016). Of the studies that were identified as examining racial differences between peer delinquency behaviors and substance use, results were mixed (Hoeben et al., 2016). More recent work has found that Black adolescents may be less susceptible to peer influences and instead driven by family oriented socialization processes (Rowan, 2016). It is also possible our results were not supportive of prior literature due to measurement differences. Barnes et al (2006) measured peer delinquency behaviors by asking adolescents “how often has your closest friend…” before each item. However, this study measured peer group delinquency behaviors by asking adolescents to report how many friends within their friend group had done each item. Measuring peer group behavior is important, as both peer selection and peer influence processes can change adolescent behavior (Henneberger et al., 2021). A study examining social identity theory found that adolescents were more likely to engage in behaviors that matched their ingroup, but that the association was stronger when adolescents reported higher levels of identification with the group (Tarrant, 2002). Additionally, a review examining the effects of peers on adolescent smoking found that close friends and peer groups have different influences on adolescent behavior; for example, one study found close friends to predict substance use initiation whereas peer groups predicted current substance use (Simons-Morton & Farhat, 2010). As explained by social identity theory, adolescents engaged in behaviors that aligned with the social norms of their group (Simons-Morton & Farhat, 2010).

In support of my hypothesis, lower levels of EA peer group delinquency behaviors were associated with higher levels of EA parental monitoring. Although this association has been strongly supported in the literature, the findings were not always generalizable. For example, in a
study completed by Crosnoe et al (2002), over 60% of the participants were white and had an average parental education level greater than high school. On the other hand, a study conducted by Dillon et al (2008) consisted of diverse and low income participants but they were recruited largely from the justice system or mental health agencies. Another study replicated findings within an Iranian sample but half of the adolescents were also recruited from the justice system, and as such those adolescents the justice system had significantly higher levels of peer group delinquency behavior than adolescents who were not in the justice system (Alboukordi et al., 2012). This finding extends past work by examining peer group delinquency and parental monitoring in a diverse, low-income sample outside of the criminal justice system.

**Peer Group Delinquency as a Moderator**

Contrary to my hypothesis, EA peer group delinquency levels did not change the association between EA substance use and LA substance use. However, high EA peer group delinquency was associated with EA substance use. Other analytical methods have found significant associations with peer group delinquency behaviors and adolescent substance use, but the studies did not report diverse samples. For example, Cambron et al (2018) utilized longitudinal latent growth modeling and found higher peer group delinquency behaviors predicted higher smoking and alcohol use from 5th to 9th grade, but only 25% of the participants were Black. Van Ryzin & Dishion (2014) hypothesized that substance use is a core aspect of social interaction within a peer group with delinquent behaviors, and the study found that peer group delinquency behaviors mediated the association between EA (12-13 years old) and LA (16-17 years old) substance use. Although this study conducted assessments at similar timepoints, only 30% of the study identified as Black (Van Ryzin & Dishion, 2014). Therefore, it is also possible that peer group delinquency behaviors follow a different pattern within Black
adolescents. Another potential explanation is that peer selection occurred during EA, but peer socialization did not take place between EA and LA (Simons-Morton & Chen, 2006). Additionally, EA peer group delinquency levels did not have an effect on the association between EA parental monitoring and LA substance use. This study expands upon current work by highlighting how peer delinquency behaviors may influence Black adolescents differently than White adolescents, as well as how parental monitoring may have a stronger role within Black families. Past research has found that compared to White parents, Black parents are more likely to supervise their children and Black adolescents report it is fear of punishment and their parent’s disapproval as reasons for not engaging in substance use (Terling Watt & McCoy Rogers, 2007).

In support of my hypothesis, EA peer group delinquency levels did effect the association between postnatal substance use and LA substance use but in an unexpected direction. Results from the multigroup analysis indicated that among adolescents with high peer group delinquency behaviors, the association between postnatal substance use and LA substance use was not significant. However, among adolescents with low peer group delinquency behaviors, higher levels of postnatal substance use predicted a lower probability of EA substance use. This unexpected result may also be due to differences in sample characteristics, specifically by the extended family network experienced by Black families, such that as caregiver substance use increases, the more likely an adolescent is to turn to biological and non-biological family members.

**Sex at Birth**

Results indicated that females had a higher probability of engaging in substance use in EA but not LA. Findings on gender and sex differences on adolescent substance use have been mixed,
but research has consistently found that substance use among female adolescents has significantly increased over the past several decades. For example, the Substance Abuse and Mental Health Statistics National Survey found a 20% increase in the rates of marijuana initiation among female adolescents from the 1960s to the 1990s (Kloos et al., 2009). Within early adolescence, a more recent large-scale study found no significant differences in substance use rates for alcohol, tobacco, marijuana, or illicit drugs but did find males to have significantly higher rates in late adolescence (Young et al., 2002). A study on the National Longitudinal Study of Adolescent Health found that females had a higher level of alcohol use from ages 12 to 15 (Biehl et al., 2007). Biehl et al (2007) also found that both males and females increased in substance use until the age of 18, but males had a steeper increase during late adolescence. The National Longitudinal Study of Adolescent Health was a majority White sample, with about 20% of participants identifying as Black (Biehl et al., 2007). Research has found Black adolescents to be less likely to engage in substance use than White adolescents. For example, results from a study also examining the National Longitudinal Study of Adolescent Health found that White adolescents were more likely to use alcohol than Black adolescents (Chen & Jacobson, 2012). This may explain why the current study did not find a sex difference in LA substance use, as the lower rates of substance use among Black adolescents, combined with the increasing trend of substance use among females, resulted in equal substance use rates in LA.

Among males, prenatal substance use had a positive association with LA substance use but postnatal substance use had a negative (and marginally significant) association with LA substance use. It is possible that child sex interacts with prenatal substance use differently than with postnatal substance use. For example, research has found that among adolescents with prenatal cocaine use, males had a blunted pressure response to stress whereas females showed an
increased emotional response to stress (Chaplin et al., 2015). Chaplin et al (2015) explained that a blunted response to stress increased the risk of substance use, as adolescents may turn to substances to increase their arousal and sensation seeking. However, an increased emotional response to stress was found to increase the risk of internalizing symptoms (Chaplin et al., 2015). However, the negative association between postnatal substance use and LA substance use in males was not expected and may reflect demographic differences.

There was a significant association of higher EA parental monitoring and lower EA peer group delinquency in female adolescents, but not in male adolescents. A study of low-income, urban Black adolescents found that adolescent self-report of parental monitoring was only a significant predictor of peer group delinquency for female adolescents (O’Donnell et al., 2012). However, the study did find that real-time reports of parental monitoring predicted peer group delinquency for male but not female adolescents (O’Donnell et al., 2012). O’Donnell et al (2012) explains that this indicated that effective parental monitoring techniques may be different for males versus female adolescents, with the perception of immediate parental monitoring being more effective for male adolescents.

**Strengths**

A major strength of the current study was the robust measurement of caregiver substance use during pregnancy and the postpartum period (birth until EA). Additionally, the longitudinal design allowed changes in substance use across the adolescent period to be examined. The longitudinal designed also allowed specific hypothesis regarding the effect of early adolescent
risk factors, such as peer group delinquency behaviors, and protective factors, such as parental monitoring, on late adolescent substance use.

**Limitations**

A main limitation of the current study was that postnatal substance use was analyzed as an aggregate measure and therefore the effect of different trajectories of postnatal substance use over time could not be modeled. However, the rank-order of postnatal substance use was maintained. On a similar note, prenatal substance use did not include the quantity of substance consumed nor the specific trimester. Instead, prenatal substance use was a composite of the number of different substances used during pregnancy. The cumulative risk index of prenatal substance use represented that the risk from prenatal substance use increased as the number of substances used in pregnancy increased, rather than from the quantity or timing of the substance (Ettekal et al., 2019). Although substance use has been found to be highly heritable (Deak & Johnson, 2021; McGue et al., 2000) this study was not able to examine the genetic influence of substance use. However, the heritability of substance use may not be fully detected during adolescence as studies have found stronger heritability estimates for adults than for adolescents (McGue et al., 2000). Additionally, a study examining substance use heritability within Monitoring the Future found illicit substances to have lower than expected heritability (McGue et al., 2000). The authors theorized that availability and access to illicit substances, along with other environmental factors, impact heritability rates (McGue et al., 2000).

Another limitation was that the study conceptualized peer group delinquency behaviors as a risk factor, and therefore as a moderator. However, the study could have also been conceptualized with parental monitoring a moderator, where high levels acted as a protective factor. Prior cross-sectional research has found support for parental monitoring as a moderator on
the association of peer substance use and adolescent substance use (Kiesner et al., 2010). Future research should examine parental monitoring as a moderator between EA peer group delinquency behaviors and LA substance use.

**Conclusions**

Despite these limitations, this study adds to the literature examining pathways from pre- and postnatal substance use to adolescent substance use. The results indicated the importance of examining caregiver substance use, parental monitoring, peer group delinquency behaviors, and adolescent substance use in a diverse, low-income sample. Specifically, the results highlight that the expected cascading effect of postnatal substance use on late adolescent substance use via parental monitoring may not be present among Black families. These results have implications for the timing of prevention programs aimed at reducing adolescent substance use. Prevention programs focused on increasing parental monitoring may be more effective during early adolescence, as results indicated higher levels of parental monitoring decreased adolescent initiation of substance use. Future work should also examine the extended family model within Black families to better understand how a close adult outside of the family unit may be protective against caregiver substance use.
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