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Gendered Differences in the Effects of Adverse Childhood Experiences
on Adolescent Substance Use

Emley A. Holcombe

A thesis submitted to the faculty of
Brigham Young University
in partial fulfillment of the requirements for the degree of
Master of Science

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ABSTRACT

Gendered Differences in the Effects of Adverse Childhood Experiences on Adolescent Substance Use

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Master of Science

Adolescence is a high-risk period for substance use, and the prevalence of adolescent substance use is a public health concern. Contributing factors for adolescent substance use are adverse childhood experiences (ACEs). ACEs are potentially traumatic childhood events that have negative associations with health and risk behaviors. The purpose of this study is to examine how the accumulation, timing, and duration of early ACEs (by age 5) impacts adolescent substance use. In addition, this study examines differences in these relationships by gender. Data from the Fragile Families and Child Wellbeing Study (FFCW) were used for the logistic regression analyses. The results generally showed significant relationships for early cumulative ACEs and early ACE timing and duration variables for the full and female sample when considering bivariate models, recency of trauma, and demographic variables. For male samples, statistical significance was only reached for extreme early cumulative ACEs and extreme early ACE timing and duration variables in all models. No significant relationships existed between early ACEs (accumulation, timing, or duration) and adolescent substance use when considering other major predictors of adolescent substance use at year 15. There were also no significant gender differences for early ACEs and adolescent substance use (accumulation, timing, or duration). Future studies should consider the impact of mediating variables on the relationship between early ACEs and adolescent substance use.

Keywords: adverse childhood experiences, adolescence, substance use, gender

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TABLE OF CONTENTS

TITLE PAGE	i
ABSTRACT.....	ii
ACKNOWLEDGEMENTS	iii
TABLE OF CONTENTS.....	iv
INTRODUCTION	1
LITERATURE REVIEW	3
Adverse Childhood Experiences (ACEs).....	3
ACEs and Adolescent Substance Use.....	4
ACEs and Gender	5
Other Major Predictors of Adolescent Substance Use.....	8
THE CURRENT STUDY.....	9
DATA AND METHODS	10
Sample.....	10
Adolescent Substance Use	11
Adverse Childhood Experiences.....	12
Individual ACEs.....	12
Cumulative ACEs by age 5.....	16
Timing and duration of ACEs by age 5	16
Cumulative ACEs at year 9.....	17

Gender.....	17
Demographics	17
Other Major Predictor Variables.....	18
ANALYTIC STRATEGY	20
RESULTS	21
DISCUSSION.....	26
Limitations	31
CONCLUSION.....	32
REFERENCES	34
TABLES	50
Table 1. Sample Characteristics by Youth’s Gender	50
Table 2. ACEs and Adolescent Substance Use by Youth’s Gender	51
Table 3. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Accumulation of ACEs (by Year 5) and Gender	52
Table 4. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Accumulation of ACEs (by Year 5 and in Year 9) and Gender	53
Table 5. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Accumulation of ACEs (by Year 5 and in Year 9), Demographics, and Gender	54
Table 6. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Accumulation of ACEs (by Year 5 and in Year 9), Demographics, Major Predictors of Adolescent Substance Use (in Year 15), and Gender	55

Table 7. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Timing and Duration of ACEs (by Year 5) and Gender.....	56
Table 8. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Timing and Duration of ACEs (by Year 5), Cumulative ACEs (in Year 9), and Gender	57
Table 9. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Timing and Duration of ACEs (by Year 5), Cumulative ACEs (in Year 9), Demographics, and Gender.....	58
Table 10. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Timing and Duration of ACEs (by Year 5), Cumulative ACEs (in Year 9), Demographics, Major Predictors of Adolescent Substance Use (in Year 15), and Gender.....	59
APPENDIX A. DISTRIBUTIONS OF ADOLESCENT SUBSTANCE USE	60
Table 11. Distribution of Adolescent Substance Use Variables by Youth’s Gender	60
Table 12. Distribution of Adolescent Substance Use as a Count Variable by Youth’s Gender	60

INTRODUCTION

The developmental time of adolescence is a high-risk period for substance use (Gray and Squeglia 2018). Indeed, about 10 percent of 8th graders, 19 percent of 10th graders, and 32 percent of 12th graders report having used illicit substances in 2020 (“Monitoring the Future” 2021). For youth ages 16-17, approximately 10 percent report binge drinking in the past month (*Behavioral Health Barometer* 2019). The prevalence of substance use among adolescents constitutes a public health concern (“Results from the Annual National Youth Tobacco Survey” 2022). Scholars have found that adolescent substance use is associated with various negative health and behavior outcomes in adolescence, such as anxiety (Hines et al. 2020; Lee et al. 2020), delinquency (Jones and Pierce 2020; Staff et al. 2020), and poor academic performance (Ghanem 2021; Houtepen et al. 2020).

Previous studies have identified important risk factors for substance use during adolescence, including peer substance use (Hoffmann 2021; Schuler et al. 2019; Trucco 2020), parent-child relationships (Mak and Iacovou 2019; Rusby et al. 2018), victimization (Davis et al. 2019; Davis et al. 2020; Kim et al. 2019), behavioral problems (Green et al. 2019; Kozak et al. 2019), and sensation seeking (Charles et al. 2016; Jensen, Chassin, and Gonzales 2017). In addition to these risk factors, studies suggest that exposure to adverse childhood experiences (ACEs) are also linked to substance use (Felitti et al. 1998; Leza et al. 2021; Loudermilk et al. 2018; Tang et al. 2021). ACEs are potentially traumatic childhood events that have negative associations with health and risk behaviors (Felitti et al. 1998). ACEs include experiences such as sexual, physical, and emotional abuse; physical and emotional neglect; parental separation or divorce; parental incarceration; household alcohol or drug use; and household mental illness.

According to the 2016 National Survey of Children’s Health, a nationally representative

study of children in the United States, more than twenty percent of children experience at least one ACE with vulnerable groups more likely to report greater exposure (Crouch et al. 2019). The consequences of ACEs have received substantial attention from researchers as numerous studies have found associations between ACE exposure and negative health and behavioral outcomes (Houtepen et al. 2020; Lee et al. 2020; Rojo-Wissar et al. 2021). However, few studies have considered how the accumulation, timing, and duration of ACE exposure are related to adolescent substance use or how gender impacts those relationships. The current study can provide a greater understanding of how gender may shape the relationship between the accumulation, timing, and duration of ACEs experienced in early childhood and substance use in adolescence. This knowledge can have significant implications for trauma prevention policies and intervention efforts.

The accumulation, timing, and duration of early ACEs has been associated with a variety of negative repercussions throughout the life course (Friedman et al. 2015; Hunt, Slack, and Berger 2017; Jimenez et al. 2016; Merrick et al. 2017; Rojo-Wissar et al. 2021; Strine et al. 2012). Recently, scholars have considered the role of ACEs in adolescent substance use. ACEs significantly increase the risk of cigarette, alcohol, and marijuana use among adolescents (Brown and Shillington 2017; Fite et al. 2015; Yilmaz, Lo, and Solakoğlu 2015). While few studies have examined gender differences in ACEs, research suggests that ACE exposure is different for boys and girls (Baglivio and Epps 2016; Fang, Chuang, and Lee 2016; Schilling, Aseltine, and Gore 2007; Strine et al. 2012), and that ACE outcomes vary by gender (Fang et al. 2016; Leban and Gibson 2020; Pierce and Jones 2021). This growing body of gender and ACE literature points toward the potential importance of gender in understanding the consequences of ACEs across the life course. Thus, further research is needed to explore how gender shapes the relationship

between ACEs and adolescent substance use. The current study adds to the existing literature on ACEs by using data from the Fragile Families and Child Wellbeing Study (FFCW), a national urban birth cohort, to examine how the accumulation, timing, and duration of ACEs in early childhood may be related to adolescent substance use and how those relationships may differ by gender.

LITERATURE REVIEW

Adverse Childhood Experiences (ACEs)

Extensive research indicates that ACEs are associated with negative health outcomes among adolescents, such as anxiety (Elmore and Crouch 2020; Kim, Galván, and Kim 2021), depression (Houtepen et al. 2020; Lee et al. 2020), sleep problems (Park et al. 2021; Rojo-Wissar et al. 2021), and suicidality (Li et al. 2021; Thai et al. 2020). ACEs are also associated with poor academic performance (Ghanem 2021; Houtepen et al. 2020) and school suspension or expulsion (Bell et al. 2021; Pierce, Jones, and Gibbs 2022). For example, Pierce and colleagues (2022) found that adolescents with a cumulative ACE score of four or more were almost four times more likely to have experienced school suspension or expulsion. In addition, other researchers have found that greater exposure to ACEs increased the risk of violence among youth ages 13-15 (Salo, Appleton, and Tracy 2021). Other studies have found that ACEs are associated with delinquency (Fagan and Novak 2018; Jones and Pierce 2020), violent behavior (Baglivio, Wolff, and Epps 2021; Salo et al. 2021), and other involvement in crime (Brockie et al. 2015; Garrido, Weiler, and Taussig 2018).

Two main themes have emerged from ACE studies. First, there is evidence of a “graded” or “dose-response” relationship between ACEs and life outcomes in that the number of ACEs experienced incrementally increases the likelihood of experiencing negative physical, mental,

and behavioral outcomes in adolescence and adulthood (Brockie et al. 2015; Felitti et al. 1998; Friedman et al. 2015; Merrick et al. 2017). Second, ACEs are highly interrelated, meaning that exposure to one ACE is likely to be associated with exposure to additional ACEs (Baglivio and Epps 2016; Mersky, Janczewski, and Topitzes 2017).

Recent literature has also suggested that the timing and duration of ACEs is especially important when it comes to childhood and adolescent outcomes. Indeed, substantial research has documented that exposure to ACEs as early as age 5 or younger is associated with worse developmental outcomes, including social delays, behavioral delays, and below average literacy skills, compared to children who have experienced no ACEs (Cprek et al. 2020; Jimenez et al. 2016; McKelvey, Selig, and Whiteside-Mansell 2017; Schroeder, Slopen, and Mittal 2020). In some studies, the longer the duration of ACE exposure for children, the larger the deficit in social and behavioral development (Cprek et al. 2020; Schalinski et al. 2016). These findings suggest the importance of ACE accumulation, timing, and duration for adolescent outcomes.

ACEs and Adolescent Substance Use

Much of what we know about the positive relationship between ACEs and substance use we have learned from studies utilizing adult samples (for examples see Allem et al. 2015; Merrick et al. 2017; Strine et al. 2012). General findings have shown that experiencing trauma is also associated with substance use in adolescence (Bender et al. 2015; Carliner et al. 2016; Cicchetti and Handley 2019). Recently, researchers have used the ACE model to understand adolescent substance use and have found that ACEs increase the risk of substance use in adolescence (Afifi et al. 2020; Brown and Shillington 2017; Giordano et al. 2014; Kühn et al. 2020; Leban and Gibson 2020; Ramos-Olazagasti et al. 2017; Yilmaz et al. 2015). Specifically, scholars have found associations between ACEs and adolescent cigarette use (Afifi et al. 2020;

Ofuchi, Zaw, and Thepthien 2020), vaping (Afifi et al. 2020; Ofuchi et al. 2020), alcohol use (Afifi et al. 2020; Ramos-Olazagasti et al. 2017), binge drinking (Afifi et al. 2020; Duke 2018), and marijuana use (Afifi et al. 2020; Chatterjee et al. 2018). In addition, scholars have identified a dose-graded relationship between ACEs and adolescent substance use outcomes in that increased exposure to ACEs increases substance use behaviors (Brockie et al. 2015; Ofuchi et al. 2020; Scheidell et al. 2018).

While prior work has shown that ACEs have a cumulative effect on adolescent substance use through the dose-graded relationship, key limitations remain. Specifically, there is little research to date that has considered the relationship between accumulation of ACEs and adolescent substance use in conjunction with the impact of timing and duration of ACEs. In addition, few studies have examined the impact of gender on the relationship of ACEs (accumulation, timing, and duration) to adolescent substance use. Many studies focus exclusively on accumulation of ACEs and overlook the impact of timing and duration which is problematic because early childhood is a sensitive period of development where traumatic experiences are more influential and associated with more severe negative outcomes later in life (Ogle, Rubin, and Siegler 2013). Early exposure to trauma can interrupt normal neurological development (Putnam 2006; Thomason and Marusak 2017). Moreover, when exposure to ACEs becomes frequent or prolonged, the stress on the body and brain become toxic and disrupt normal brain functioning, leading to lifelong mental and physical strain (Agorastos et al. 2018). Some evidence also suggests that longer durations of ACE exposure are associated with more severe outcomes (Cprek et al. 2020; Schalinski et al. 2016). These essential aspects of timing and duration are often overlooked when examining the outcomes of ACE accumulation.

ACEs and Gender

A small but growing body of literature has considered the role of gender in ACE exposure and outcomes. ACEs are not gender-neutral experiences. Several studies have found greater overall ACE exposure for females compared to males (Baglivio and Epps 2016; Baglivio et al. 2014; Fang et al. 2016; Felitti et al. 1998). In the original ACE study, Felitti and colleagues (1998) found that a higher percentage of women reported experiencing 2, 3, or 4+ ACEs compared to men. Moreover, other ACE studies have generally found that females report more instances of abuse in every ACE category except for physical abuse (Baglivio and Epps 2016; Fang et al. 2016; Schilling et al. 2007; Strine et al. 2012). Studies have also shown that ACEs are linked to different health and behavior outcomes for boys and girls (Leban and Gibson 2020; Pierce and Jones 2021).

One hypothesized link between ACEs and adolescent substance use is the self-medication hypothesis. This hypothesis suggests that individuals who exist in painful emotional extremes, either feeling too much or not at all, use substances to relieve painful emotions or to control emotions (Khantzian 1997). Similarly, Agnew's (2006) General Strain Theory (GST) focuses on the relationship between emotions and coping behaviors. GST is a theoretical perspective that can provide an explanation for gendered differences and perspectives in strain. Although gender was not the original focus of GST, gender became an important theoretical addition. GST proposes that males and females experience different types of strain and that they cope with or respond to that strain differently (Agnew 2006). Gendered responses to strain are due to different gender socialization between males and females. A recent study found that traditionally masculine women and traditionally feminine women were socially conditioned to respond differently to strain based on their internalized gender norms (Isom Scott and Mikell 2019). Agnew (1992) has suggested that one source of strain is the presence of negative stimuli,

including abuse or trauma.

Research supports this theory with evidence that males and females experience different types of strain and that their responses to strain differ by gender (Broidy 2001; Broidy and Agnew 1997; Jang 2007; Moon and Morash 2017). However, not all studies support these findings. There is mixed evidence as one of the first empirical tests of GST did not find differences by gender (Hoffmann and Su 1997). In a sample of adolescents, males and females reported different strain with males more likely to experience physical punishment than females (Hay 2003). In general, females report greater ACE exposure than males (Baglivio and Epps 2016; Fang et al. 2016; Kappel et al. 2021; Strine et al. 2012). Females, as opposed to males, are also more likely to respond to strain with self-destructive coping mechanisms such as disordered eating and substance use (Francis 2014; Piquero et al. 2010). When males experience negative emotions due to strain, they are more likely to experience emotions like anger. This anger is directed outward and towards others. Females, on the other hand, respond to strain with emotions such as sadness, anxiety, depression, and anger (Broidy and Agnew 1997; Sharp, Brewster, and Love 2005). These emotions, including anger, are directed inward, causing a different response than the male response (Broidy and Agnew 1997; Jang 2007). When confronted with these emotions, many women turn to coping methods such as substance use (Sharp, Peck, and Hartsfield 2012).

Although few ACE studies examine gender differences, Leban and Gibson (2020) recently examined gendered differences between ACEs and adolescent substance use. Their research indicated that the risk for adolescent female substance use remained significant while the risk for adolescent male substance use was non-significant when accounting for other risk factors besides ACEs. Another recent study examined gendered differences in accumulation,

timing, and duration of ACEs as they related to youth delinquency (Pierce and Jones 2021). In this study, early and prolonged ACEs were significantly related to delinquency for females, but not males. (Pierce and Jones 2021).

Agnew also noted the importance of strain timing and duration in his theory of GST. Agnew (2006) theorized that high magnitude strains were more likely to cause engagement in negative coping behaviors due to threshold effects. High magnitude strains can be long duration strains, early strains, and accumulation of strains which contribute to a threshold effect (Agnew 2006). A threshold effect in this instance represents the point at which the balance of motivations and constraints toward substance use shifts in favor of substance use due to abuse (Agnew and Messner 2015). This threshold effect is similar to the physiological concept of allostatic load. Allostatic load refers to a state of being where individuals experience a system imbalance in the body due to strain (McEwen and Stellar 1993). Allostatic load is a result of chronic, cumulative, or repeated stressors such as ACEs (Finlay et al. 2022). When environmental challenges, such as abuse, surpass the individual's ability to cope, allostatic overload occurs (Finlay et al. 2022). In this state, stress response systems in the body are continuously activated in a flight or fight response leading to body dysregulation and eventually diseases such as mental illness (Finlay et al. 2022). Accordingly, ACEs are associated with elevated allostatic load and poorer health outcomes in adulthood (Finlay et al. 2022; McEwen and Stellar 1993). The theoretical background of threshold effects and biological impact of allostatic load increases understanding of how accumulation, timing, and duration of childhood abuse impacts adolescents. This research illustrates the importance of more complex ACE analysis that includes consideration of accumulation, timing, duration, and gender.

Other Major Predictors of Adolescent Substance Use

Other major predictors of adolescent substance use are behavioral problems (Green et al. 2019; Kozak et al. 2019), sensation seeking (Charles et al. 2016; Jensen et al. 2017), diagnosis of attention-deficit/hyperactivity disorder (ADHD) (Molina et al. 2018; Rhodes et al. 2016), peer substance use (Hoffmann 2021; Schuler et al. 2019; Trucco 2020), lack of parental supervision (Allen et al. 2016; Kristjansson et al. 2020), and distant parent-child relationships (Mak and Iacovou 2019; Rusby et al. 2018). Because these variables can impact adolescent substance use, examining these variables in addition to early ACEs gives a more nuanced and complete understanding of adolescent substance use.

THE CURRENT STUDY

While the consequences of ACEs in adulthood have been well-documented, significantly less research has considered the effects of ACE accumulation, timing, and duration on adolescent substance use, and even fewer have examined the role of gender. The purpose of this study is twofold: first, I examined how the accumulation, timing, and duration of early ACEs influence adolescent substance use; second, because few studies to date have examined how the effect of ACEs may vary across groups, I then explored how these patterns vary by gender. Specifically, the current study investigates the following hypotheses:

Hypothesis 1: Adolescents who experience more cumulative ACEs by age 5 will be more likely to engage in substance use.

Hypothesis 2: Adolescents who experience earlier ACEs (timing) and experience those ACEs over a longer period of time (duration) will be more likely to engage in substance use.

Hypothesis 3: The effects of ACEs (cumulative, timing, and duration) by age 5 on adolescent substance use will vary by gender.

DATA AND METHODS

Sample

The current study uses data from the Fragile Families and Child Wellbeing Study (FFCW). The FFCW Study contains longitudinal data from 4,898 children born in 20 US cities sampled from hospitals starting in 1998-2000. Individuals were selected from a stratified, multistage sampling technique of cities and hospitals. Sampling from hospitals was done in an effort to increase response rates, especially from unwed fathers. Data used in this analysis was from the focal child's birth, year 1, year 3, year 5, year 9, and year 15. Year 15 data was collected from 2014-2017 when the focal child was in adolescence. This data set is representative of children born to unmarried parents from cities in the US with a population greater than 200,000. To learn more about fragile families, the majority of births sampled were to unwed parents with a 3 to 1 ratio of births to unwed parents compared to married parents.

Initial data was collected in the hospital by an interview with the parents shortly after the child's birth. Follow up interviews were done after 1, 3, 5, 9, and 15 years. These interviews were done both over the phone and with in-home assessments. Interviews were conducted with mothers, fathers, primary caregivers, as well as the focal child in later years. This dataset provides an important source for ACE research because oversampling was done for unmarried parents, poor families, and minority families who are at a higher risk for ACE exposure (Reichman et al. 2001).

In order to estimate the gendered effects of early ACEs on youth substance use, I combined data from the focal child's birth with core mothers and father surveys at years 1, 3, and 5, primary caregiver surveys from years 3, and 5, and child surveys from year 15. I also included the caregiver survey and child survey from year 9 to statistically adjust for recency of ACEs in

the models. Response rates across the years averaged about 85 percent (Schroeder et al. 2020). Within the years of data compiled for this analysis, missing data on the demographic variables is below 5 percent. However, missing data is closer to 25 percent for the child maltreatment measures as this data was obtained during in-home assessments. To address missing data, I used multiple imputation to produce and merge 25 data sets with 100 burn-ins using a chained equation method of multiple multivariate data imputation (Graham, Olchowski, and Gilreath 2007). This method of addressing missing data is consistent with several other studies that have used Fragile Families data to study ACEs (Pierce, Jones, and Holcombe 2022; Quader, Gazmararian, and Suglia 2022). Knowing that the missing data are not missing at random and that imputing on non-random missing variables can produce biased estimates and standard errors, I utilized a standard conservative approach (Allison 2001). I used the measure I created for adolescent substance use to impute values for all the independent variables. Following guidance from Von Hippel (2007), I ultimately excluded cases with imputed values for the outcome from the analyses.

Adolescent Substance Use

Adolescent substance use was measured with a series of questions asked to the focal child in year 15 that captured a range of substance use behaviors that included alcohol use, tobacco use, marijuana use, illicit drug use, and prescription drug misuse. For alcohol, the adolescent was asked if they, “ever had a drink of beer, wine, or liquor, not just a sip or a taste of someone else's drink, more than two or three times in your life when you were not with your parents.” The adolescent was also asked if they had “ever smoked an entire cigarette,” “ever tried marijuana,” “ever tried illegal drugs besides marijuana,” and “ever used prescription drugs (not prescribed).” Possible responses to all substance use questions were “yes” or “no.” The distributions of use or

misuse for each substance is included in Table 11 (see Appendix A). According to this distribution, most adolescents reported engaging in marijuana use (21.65 percent) and alcohol use (17.00 percent). The third highest reported substance use among adolescents was tobacco at 5.38 percent. A count from 0 to 5 was then created by adding together the substance use variables showing how many substances a youth reported using or misusing. The distribution of adolescent substance use as a count variable is shown in Table 12 (see Appendix A). Because very few in the sample reported using or misusing 3 or more substances, a dichotomous variable representing adolescent substance use was created to indicate whether the focal child had used any of the substances (0 = no substance use, 1 = any substance use).

Adverse Childhood Experiences

Individual ACEs. For the ACE measures, I used the CDC-Kaiser Study (Felitti et al. 1998) and subsequent ACE research (Pierce, Jones, and Holcombe 2022; Hunt et al. 2017) as a framework. I examined eight categories of ACEs during the early childhood FFCW years (1, 3, 5) and at year 9 (which is coded separately as a measure of recency): physical abuse, emotional abuse, physical neglect, emotional neglect, household substance use, parental incarceration, parental intimate partner violence, and household depression and/or anxiety. For all 8 ACE categories, a dichotomous variable was created representing exposure to that ACE in either years 1, 3, or 5 (coded as 1) or no exposure to that ACE in years 1, 3, and 5 (coded as 0). Similarly, a dichotomous variable was created for each ACE category in year 9 representing exposure to that ACE in year 9 (coded as 1) or no exposure to that ACE in year 9 (coded as 0).

Select measures from the Parent-Child Conflict Tactics Scale (CTS-PC) were used to determine physical and emotional abuse and neglect (Straus et al. 1998). In FFCW, CTS-PC subscales were coded in the following scale: never happened, once, twice, 3 to 5 times, 6 to 10

times, 11 to 20 times, and more than 20 times. To calculate the degree of maltreatment, I used the midpoint of each category and then total them. These totals were recoded into a dichotomy indicating whether a family scored in the top 10th percentile for the total number of acts toward the child as done in previous studies (Hunt et al. 2017; Pierce and Jones 2021).

Physical neglect is measured at years 3, 5, and 9. All 3 years were measured by asking the mother and father whether she or he: “left child home alone, but thought some adult should be with him/her,” “was not able to make sure (child) got the food he/she needed,” “wasn’t able to take child to a doctor or hospital,” and “were so drunk/high that you had a problem taking care of your child.” After calculating the top 10th percentiles, dichotomous measures were created for physical neglect by year 5 and physical neglect in year 9.

Emotional neglect was measured at years 3, 5, and 9. Each year was measured with one item to the primary caregiver asking if they were “so caught up with your own problems that you were not able to show love to your child.” After calculating the top 10th percentiles, dichotomous measures were created for emotional neglect by year 5 and emotional neglect in year 9.

Physical Abuse is measured at years 1, 3, 5, 9. At year 1, the mother and father were asked if they, their partner, or the other parent had, “spanked the child in the past month.” At years 3, 5, and 9, the primary caregiver was asked how many times in the past year he or she: “shook child,” “hit child on bottom with a hard object,” “spanked him/her on the bottom with your barehand,” “slapped child on the hand, arm, or leg,” and “pinched child.” At year 9, the father was also asked how many times in the past year they had “spanked child on the bottom with bare hand,” “slapped child on hand, arm, or leg,” and “pinched child.” In year 9, the focal child was also asked how often “mom spanked or hit you,” “father spanked or hit you,” and “social father spanked or hit you.” Dichotomous measures for physical abuse by year 5 and

physical abuse in year 9 were created based on the top 10th percentiles.

Emotional Abuse was measured at years 3, 5, and 9 by asking the primary caregiver or parent how many times he or she had “swore or cursed at a child,” “shouted, yelled, or screamed at child,” “said you would send child away or would kick child out of the house,” “called him/her dumb or lazy or some other name like that,” and “threatened to spank or hit child but did not actually do it.” In year 9, the focal child was also asked how often their mom or social father “shouted, yelled, screamed, swore, or cursed at you.” Dichotomous measures for emotional abuse by year 5 and emotional abuse in year 9 were created based on the top 10th percentiles.

Household substance use was measured in years 1, 3, 5, and 9 for mothers, biological fathers, and mother’s current partner (when applicable). At year 1, mothers were asked: if they had smoked marijuana or used cocaine or other hard drugs in the past month; if drinks or drugs has interfered with how they manage daily since the birth of their child; if drinking or drugs interfered with personal relationships since the birth of the child; and if they’ve sought help or been treated for a drug or alcohol problem since the birth of the child. If the mother responded yes to any of these questions, household substance use was coded “yes” for that year. To determine maternal substance use in the following years, mothers were asked in years 3, 5, and 9 if they had used a series of drugs in the previous year: sedatives, tranquilizers, amphetamines, analgesics, inhalants, marijuana, cocaine, LSD, and heroine. Heavy drinking was also measured in year 5 by asking if the mother often had 4 or more drinks in one day almost “every day,” “a few times a week,” or “a few times a month.” To determine drug and alcohol use for fathers and mothers’ current partners, the mother was asked in years 5 and 9 if the father or current partner had “problems with job/family/friends because of alcohol/drug use.”

Using these questions, I created a dichotomous variable representing household substance

use to indicate whether the child's biological mother, biological father, or mother's current partner had used substances between survey years 1 and 5 (0 = no drug and/or heavy alcohol use, 1 = some drug and/or heavy alcohol use). Following the same procedure, household substance use at year 9 was coded dichotomously (0 = no drug and/or heavy alcohol use, 1 = some drug and/or heavy alcohol use).

Parental incarceration was measured based on reports from mothers and fathers during years 1, 3, 5, and 9. Mothers and fathers reported on whether the mother, father, or mother's current partner had spent any time in prison or jail or were currently in prison or jail at years 1, 3, 5, and 9. The variable for parental incarceration by year 5 was coded dichotomously (0 = no parental or partner incarceration, 1 = any parental or partner incarceration). At year 9, parental incarceration was also coded dichotomously.

Parental interpersonal violence was measured at years 1, 3, 5, and 9 using a combination of physical, emotional, and sexual violence experienced by a mother. While this variable does not measure direct abuse, the child would be exposed to domestic violence that his or her mother experienced from her romantic partner (either the child's biological father or current partner). The following questions were asked among years 1, 3, 5, and 9 to the focal child's mother and indicates exposure to parental interpersonal violence: how often the child's biological father or mother's current partner (1) "tries to keep you from seeing or talking with your friends or family," (2) "tries to prevent you from going to work or school," (3) "withholds money, makes you ask for it, or takes it," (4) "slaps or kicks with a fist or object," (5) "pushes, grabs, or shoves, you," (6) "hits you with a fist or dangerous object in front of child," (7) "throws something at you," (8) "has a physical fight with you in front of child," (9) "forces you to have sex or do sexual things," (10) "withholds sex to try and control your behavior," and (11) "if you were ever

cut, bruised, or seriously hurt in fight.” If the mother reported any of the emotional, physical, or sexual abuse measures at year 1, 3, or 5, parental interpersonal violence was coded as yes (1 = yes, 0 = no). Similarly, a dichotomous variable was created for parental interpersonal violence at year 9.

To measure *household depression and/or anxiety*, I used a scale determining if the mother or biological father (data was not available for mother’s current partner) meets anxious criteria at year 1 and year 3 per the Composite Interview Diagnostic Interview (CIDI) (Kessler et al. 1998). The CIDI is a standardized, reliable assessment of mental disorders to measure generalized anxiety disorder and major depression (Patten 1997). Similarly, the CIDI was used to determine mother’s and biological father’s depression at years 1, 3, and/or 5 and 9. These results were then dichotomized to create overall parental anxiety and/or depression through years 1, 3, and 5 (0 = no parental depression or anxiety, 1= parent had depression and/or anxiety). A similar dichotomous variable was created for parental depression and/or anxiety at year 9.

Cumulative ACEs by age 5. To create a cumulative ACE measure, I examined each specific ACE across years. The scale utilized the previously created dichotomized measures for each individual ACE exposure in years 1, 3, or 5. The dichotomized individual ACEs through year 5 were added together to create a scale from no exposure (coded 0) to exposure to all 8 ACEs (coded 8). To be consistent with CDC-Kaiser ACE literature, ACE scores of 4 through 8 were combined into one category of “4 or more.”

Timing and duration of ACEs by age 5. Following previous studies, I measured timing and duration of ACEs by first dichotomizing the ACE index score at each year, independently, to represent high ACE scores (2+ ACEs at that year) versus no/low adversity (0-1 ACEs at that year) (Schroeder et al. 2020). Next, I created a five level category variable: (1) no or low

adversity at each year (< 2 ACEs at year 1, year 3, and year 5; reference category), (2) high early adversity (≥ 2 ACEs in year 1 and/or year 3 but not year 5), (3) high late adversity (≥ 2 ACEs in year 3 and year 5 or year 5 only but not year 1), (4) intermittent high adversity (≥ 2 ACEs in year 1 and year 5 but not year 3), and (5) chronic high adversity (≥ 2 ACEs in year 1, year 3, and year 5).

Cumulative ACEs at year 9. I also examined ACEs reported in year 9 as a way to measure recency of ACE exposure. Individual ACEs for this year were previously coded (0 = no exposure, 1 = exposure to that type of ACE). These items were added up to form a scale from 0 (no exposure) to 8 (exposure to all 8 ACEs) at year 9. ACE scores of 4 through 8 were combined into one category (4 or more). To maintain proper time order, ACEs recorded at year 9 must be experienced after the year 5 survey.

Gender

Gender was measured using a constructed variable indicating the focal child's gender as male or female at birth. A dichotomous variable was created based on this with 1 indicating female and 0 indicating male.

Demographics

The demographic variables I included in certain analyses are adolescent age, mother's age, mother's marital status, adolescent race/ethnicity, poverty status, and mother's education. Adolescent age was based on a constructed variable at year 15 determining how old the adolescent was at the time of their primary caregiver's interview and remained a continuous variable. Mother's age was measured at the focal child's birth and remained a continuous variable. Mother's marital status was also measured at the focal child's birth and coded as 0 for not married to the biological father at child's birth and 1 for married to the biological father at

the child's birth. Adolescent race/ethnicity was based on a question in year 15 asking the youth to self-describe their race/ethnicity. Answers were recoded as "White," "Black," "Hispanic," and "Other." The variable for poverty status was based on a constructed variable indicating the household relationship to the poverty line at the focal child's birth. It was recoded as 1 for families above the poverty line and 0 for families below the poverty line at the focal child's birth. For mother's education, the variable was measured at the focal child's birth and recoded into three categories: "less than high school," "high school or equivalent," and "greater than high school."

Other Major Predictor Variables

I also included major predictors of adolescent substance use in some analyses. These major predictor variables are adolescent self-control, ADHD diagnosis, adolescent peer substance use, parental supervision, mother-child closeness, and father-child closeness. These measures were added to some models to better understand how these more recent factors impact adolescent substance use when still considering early ACE exposure. These major predictor variables were all measured using data from year 15. However, data was not gathered in the FFCW dataset on the timing of these major predictor variables in year 15. Therefore, time order cannot be distinguished for these major predictor variables in year 15 and adolescent substance use in year 15.

Adolescent self-control variable was created from an abbreviated Dickman's impulsivity scale (Dickman 1990) by asking the following questions: (1) "I don't spend enough time thinking over a situation before I act," (2) "I often say whatever comes into my head without thinking first," (3) "I often get into trouble because I don't think before I act," (4) "I often say and do things without considering the consequences," (5) "The plans I make don't work out because I

haven't gone over them," and (6) "I often make up my mind without taking the time to consider the situation from all angles." These questions were reverse coded and then added together to create a scale for adolescent self-control from 0 to 18 with higher scores representing more impulsivity.

Adolescent ADHD diagnosis was from one question in year 15 asking the primary caregiver if the adolescent had been diagnosed by a doctor with ADHD. Adolescent peers' substance use was created from 5 questions asked to the focal child about peer use of alcohol, marijuana, or other drugs: (1) "Friends drank alcohol more than two times without their parents," (2) "Friends tried marijuana," (3) "Friends tried other drugs to get high," (4) "Friends asked you to go drinking with them," and (5) "Friends given or sold marijuana to you." The answers were reverse coded and added together to create a scale from 0 to 10 for peer substance use with higher values representing greater peer substance use.

Parental supervision came from three questions asked to the adolescent about how often their primary caregiver (1) "knows what you do during your free time," (2) "knows what you spend money on," as well as (3) "how often you spend time alone in your home without an adult present?" These questions were added together to create a scale for parental supervision ranging from 0 to 6, with higher scores representing more parental supervision. For mother-child closeness and father-child closeness, two questions were asked to the child at year 15: (1) "How close do you feel with your biological mother/father?" and (2) "How well do you and your mom/biological father dad share ideas/talk?" These questions were reverse coded and added together respectively to create two scales: a mother-child closeness scale and a father-child closeness scale. These scales ranged from 0 to 6 with higher scores representing more closeness between mother-child or father-child.

ANALYTIC STRATEGY

The distributions and means of the demographic variables and other major predictors of adolescent substance use for the analytic sample from FFCW are presented in Table 1 reported by the full sample as well as by gender of the adolescent. In Table 2, the distributions of cumulative ACEs by year 5, categories of ACE timing and duration, cumulative ACEs at year 9, and adolescent substance use are presented for the full analytic sample from FFCW study as well as the female-only and male-only subsamples.

Tables 3, 4, 5, and 6 assess the impact of cumulative ACEs (by year 5) on adolescent substance use for the full analytic sample, females only, and males only using logistic regression with coefficient results recorded as odds ratios. Table 3 shows the bivariate relationship between early cumulative ACEs and adolescent substance use for the full sample (Model 1), for females only (Model 2) and for males only (Model 3). Table 4 adds on to Table 3 by including ACEs at year 9 to investigate the effect of cumulative ACEs by year 5 on adolescent substance use in the presence of more recent ACEs for the full sample (Model 1), females (Model 2), and males (Model 3). Table 5 adds demographic variables, again, for the full sample (Model 1), females (Model 2), and males (Model 3). Finally, Table 6 includes all the variables from Table 5 plus major predictors of adolescent substance use reported at year 15 for the full sample (Model 1), females (Model 2), and males (Model 3).

Tables 7, 8, 9, and 10 examine the effect of timing and duration of ACEs by year 5 on adolescent substance use for the full analytic sample (Models 1), females only (Models 2), and males only (Models 3) subsamples using logistic regression with coefficient results recorded as odds ratios. Similar to Tables 3-6, I present the results in a stepwise fashion that included the bivariate relationship between timing/duration of ACEs by year 5 (Table 7), cumulative ACEs at

year 9 (Table 8), demographic variables (Table 9), and major predictors of substance use from year 15 (Table 10). For all models in Tables 3-10, I also include difference tests on the untransformed regression coefficients (before the coefficients are transformed into odds ratios) to assess whether the models for males and females are statistically different from each other (Paternoster et al. 1998). All analyses were conducted with the Stata statistical software (version 17.0).

RESULTS

Table 1 shows characteristics for the full sample, females only, and males only. Female youth make up 48 percent of the sample and both male and female youth average just over 15.5 years old. At Wave 1, mothers were an average age 25 years old, and nearly 24 percent of the mothers were married to the biological father at the birth of the focal child. The child's ethnic/racial breakdown is 18 percent White, about 49 percent Black, 25 percent Hispanic, and 8 percent Other. Nearly 39 percent of the sample were above the poverty line and the mothers' educational breakdown includes 32 percent reporting a less than high school diploma, 32 high school graduate, and 36 percent reporting some college or more. The average self-control score for both male and female youth was about 8.8. About 23 percent of males in the sample had been diagnosed with ADHD compared to about 9 percent of females. The average peer substance score was about 1.29. The average score for parental supervision, mother-child closeness, and father-child closeness were approximately 4.4, 4.3, and 2.7, respectively.

Table 2 shows the distribution of ACEs and substance use for the full sample and then separated by gender. Overall, looking at the accumulation of early ACEs by age 5, about 11 percent had an early ACE score of 0, 19 percent had an ACE score of 1, 20 percent had an ACE score of 2, 20 percent had an ACE score of 3, and 31 percent had an ACE score of 4 or more.

These numbers deviated slightly when separated by gender. Similarly, for the timing and duration of ACEs, about 10 percent experienced no or low adversity, 21 percent experienced high early adversity, 14 percent experienced high late adversity, 7 percent experienced intermittent high adversity, and 49 percent experienced chronic high adversity. Again, these numbers deviate slightly by gender. At year 9, about 25 percent of sample had an ACE score of 0, 31 percent had an ACE score of 1, 23 percent had an ACE score of 2, 13 percent had an ACE score of 3, and 8 percent had an ACE score of 4 or more. On average, about 29 percent of adolescents engaged in substance use at year 15; more specifically 27 percent of females that reported substance compared to about 31 percent of males.

Results of logistic regressions examining associations between early cumulative ACEs and adolescent substance use are presented in Table 3. Models 1-3 in this table, as with subsequent models in Tables 4-9, show results for the full model, females only, and males only. In Model 1, for each additional ACE a child experienced, the odds of substance use increased: 2 ACEs (OR = 1.68, $p < .01$), 3 ACEs (OR = 1.80, $p < .001$), and 4+ ACEs (OR = 2.50, $p < .001$). Moreover, the effects of early ACEs on substance use were positive and significant for both females (2, 3, and 4+ ACEs) and males (3 and 4+ ACEs).

Table 4 adjusts for the effects of ACEs at year 9 on substance use in order to understand the influence of recency on the previous models (see Table 3). Overall, the effects of early ACEs are reduced across the full model, female model, and male model, though most relationships remain significant. In the full model, youth who experienced 2 ACEs by year 5 (OR = 1.60, $p < .01$), 3 ACEs by year 5 (OR = 1.65, $p < .01$), and 4+ ACEs by year 5 (OR = 2.15, $p < .001$) had an increased odds of substance use. Similar to Table 3, early ACEs were positively related to adolescent substance use for females and males, though only 4+ ACEs (OR = 2.29, $p < .001$)

were significant for males. For each additional ACE a child experienced at year 9, the odds of reporting substance use increased. Specifically, adolescents who experienced 3 ACEs (OR= 1.41, $p \leq .05$) and 4+ ACEs (OR= 1.63, $p \leq .01$) had an increased odds of substance use. When it comes to this relationship by gender, year 9 ACEs were only significant for females (3 and 4+ ACEs).

In Table 5, demographic variables are included in the models to adjust for their impact on adolescent substance use in addition to cumulative ACEs by year 5 and at year 9. Compared to the previous model (see Table 4), the same early ACEs (by year 5) and ACEs at year 9 remained statistically significant. Generally, however, the impact of ACEs by year 5 is reduced for the full model, female model, and male model. For cumulative ACEs by year 5 in the full model, youth who experienced 2 ACEs (OR = 1.50, $p < .05$), 3 ACEs (OR = 1.54, $p < .05$), and 4+ ACEs by year 5 (OR = 1.95, $p < .001$) had an increased odds of substance use. Similar to Tables 3 and 4, cumulative early ACEs of 2 (OR = 1.64, $p < .05$), 3 (OR = 1.75, $p < .05$), and 4+ (OR = 1.88, $p < .05$) were positively related to adolescent substance use for females. However, for males, only 4+ ACEs (OR = 2.02, $p < .001$) were significantly related to adolescent substance use. In the full model, adolescents who experienced 3 ACEs (OR= 1.34, $p < .05$) and 4+ ACEs (OR = 1.57, $p < .01$) had an increased odds of substance use. ACEs at year 9 were only significant for females for 3 (OR = 1.67, $p < .05$) and 4+ ACEs (OR = 1.82, $p < .05$). Adolescent age was significant and positively related to adolescent substance use for the full model (OR= 1.63, $p < .001$), females (OR = 1.54, $p < .001$), and males (OR = 1.74, $p < .001$).

Table 6 includes major predictors of substance use at year 15. When adjusting for these predictors of substance use at year 15 and the demographic variables from the previous models (see Table 5), early ACEs were no longer significant for the full model, the female model, or the

male model. Only 4+ ACEs at year 9 (OR = 1.50, $p \leq .05$) was significantly related to adolescent substance use in the full model. Adolescent age remained positively related to adolescent substance use in the full model (OR = 1.49, $p \leq .001$), females (OR = 1.40, $p \leq .001$), and males (OR = 1.57, $p \leq .001$). In addition, self-control and peer substance increased the odds of adolescent substance use in the full model (OR = 1.07, $p \leq .001$; OR = 1.79, $p \leq .001$), female-only (OR = 1.08, $p \leq .001$; OR = 1.75, $p \leq .001$), and male-only (OR = 1.07; $p \leq .001$; OR = 1.83, $p \leq .001$) models.

Logistic regression results examining associations between ACE timing and duration variables and adolescent substance use are presented in Tables 7-10. In Table 7, the majority of ACE timing and duration categories increased the odds of adolescent substance use in the full sample and the female-only sample: high late adversity (full OR = 1.57, female OR = 1.86, $p < .05$), intermittent high adversity (full OR = 1.95, female OR = 2.28, $p < .01$), and chronic high adversity (full OR = 2.48, female OR = 2.53, $p < .001$). Only chronic high adversity was significantly related to substance use for males (OR = 2.41, $p < .001$).

The models in Table 8 adjust for the effects of ACEs at year 9 (see Table 8). Overall, the effects of ACE timing and duration are reduced across the full model, female model, and male model, though most relationships remain significant. In the full model, youth who experienced high late adversity by year 5 (OR = 1.49, $p < .05$), intermittent high adversity by year 5 (OR = 1.77, $p < .01$), and chronic high adversity by year 5 (OR = 2.17, $p < .001$) had an increased odds of substance use. Some ACE timing and duration measures were positively related to adolescent substance use for females as well. Specifically, female youth who experienced intermittent high adversity by year 5 (OR = 2.02, $p < .05$) and chronic high adversity by year 5 (OR = 2.11, $p < .01$) had an increased odds of substance use. However, only chronic high adversity by year 5

(OR = 2.21, $p \leq .01$) was significant for males. For each additional ACE a child experienced at year 9, the odds of reporting substance use increased. Specifically, adolescents who experienced 2 ACEs (OR= 1.33, $p \leq .05$), 3 ACEs (OR= 1.50, $p \leq .01$), and 4+ ACEs (OR= 1.79, $p \leq .01$) had an increased odds of substance use. This was similar to the relationship between year 9 ACEs and substance use for females. Females who experienced 2 ACEs (OR= 1.56, $p \leq .05$), 3 ACEs (OR= 1.85, $p \leq .01$), and 4+ ACEs (OR= 2.12, $p \leq .01$) had an increased odds of substance use. The only significant relationship between year 9 ACEs and substance use for males was 4+ ACEs (OR= 1.55, $p < .05$).

In Table 9, demographic variables are included in the regression models with ACE timing and duration and cumulative ACEs in year 9 to adjust for their impact on adolescent substance use. The impact of the ACE timing and duration variables are generally reduced across the models with several no longer meeting the threshold for statistical significance. In the full model, youth who experienced intermittent high adversity (OR = 1.59, $p < .05$) and chronic high adversity (OR = 1.91, $p < .001$) had an increased odds of substance use. For females, intermittent high adversity (OR = 1.89, $p < .05$), and chronic high adversity (OR = 1.94, $p < .01$) had an increased odds of adolescent substance use. Chronic high adversity was the only timing and duration variable positively related to adolescent substance use for males (OR = 1.88, $p < .01$). In the full sample, cumulative ACEs at year 9 were significantly related to adolescent substance use for 3 ACEs (OR = 1.41, $p < .05$) and 4+ ACEs (OR = 1.70, $p < .01$). Although no year 9 ACEs were significantly related to adolescent substance use for the male sample, several cumulative ACEs at year 9 were significantly related to adolescent substance use for females. Specifically, 2 ACEs (OR = 1.51, $p < .05$), 3 ACEs (OR = 1.80, $p < .01$), and 4+ ACEs (OR = 2.03, $p < .01$). There was also a significant gender difference between males and females for 3 cumulative

ACEs at year 9 ($p < .05$) using a test of equality (Paternoster et al. 1998).

In Table 10, major predictors of adolescent substance use at year 15 were included in the models in addition to the demographic variables, ACE timing and duration variables, and cumulative ACEs in year 9. No early ACE timing and duration variables were statistically significant in either the full, female, or male models. However, 4+ ACEs in year 9 ($OR = 1.54, p < .05$) were significant in the full model. For the female sample, 3 ACEs in year 9 ($OR = 1.70, p < .05$) and 4+ ACEs ($OR = 1.82, p < .05$) in year 9 were significant. There were no significant gender differences between males and females for any ACE measures. Several demographic variables and major predictors of adolescent substance use were significant across models. I used the Paternoster (1998) test of equality which utilized the untransformed logistic-scale coefficients from the female sample and male sample to determine if there were statistically significant differences across all the models for cumulative ACEs by year 5 and year 9 (Tables 3-6) as well as timing and duration of early ACEs and year 9 ACEs (Tables 7-9). The only significant gender difference for an ACE variable was in Table 9 for 3 cumulative ACEs in year 9. There were also significant gender differences between several demographic variables (i.e., age and race) as well as several major predictors of adolescent substance use at year 15 (i.e., parental supervision and mother-child closeness).

DISCUSSION

The purpose of this study was to examine the relationship between ACEs and adolescent substance use as well as how this relationship differs by gender. As the majority of adolescents reported marijuana and alcohol use (see Table 11 in Appendix A), these findings may be particularly important for understanding the relationship between early ACEs and adolescent marijuana and alcohol use. This study addresses gaps in the literature regarding adolescent

substance use. First, few studies have simultaneously considered the impact of early ACE accumulation, timing, and duration. Second, there has been limited research considering how gender shapes these relationships. By specifically including early ACE accumulation, timing, and duration as well as gender differences in the analyses, I was able to examine the complexity and nuance between early ACEs and adolescent substance use. To evaluate these associations, I proposed and tested three hypotheses using a General Strain Theory perspective. First, I proposed that adolescents who experienced early strain or ACEs by year 5 would be more likely to engage in substance use. Second, I hypothesized that the timing and duration of early ACEs will have a significant impact on adolescent substance use. Finally, I hypothesized that the impact of early ACEs (accumulation, timing, and duration) on adolescent substance use would significantly vary by gender. This study and its associated hypotheses attempt to fill the existing knowledge gaps in our understanding of ACEs, substance use, and gender.

My first hypothesis that cumulative early ACEs are significantly related to greater substance use in adolescence was supported by Tables 3-5. For the bivariate analyses in Table 3, the models for the full sample and for the female sample indicated that early cumulative ACEs by age five—two, three, and four or more—were significantly related to greater substance use in adolescence as well as three and four or more early ACEs for the male sample. Although the significance of cumulative early ACEs generally decreased with consideration of more recent exposure to ACEs (at year 9) and demographic variables, several early ACEs continued to meet the threshold for statistical significance. Specifically, early cumulative ACEs of two, three, and 4 or more remained statistically significant in Table 5 (which included year 9 ACEs) and Table 6 (which included year 9 ACEs and demographic variables) for the full sample and the female sample. In Tables 5 and 6, experiencing 4 or more early ACEs was significantly related to

adolescent substance use for the male sample as well. The analysis results in Tables 3, 4, and 5 provide evidence that Hypothesis 1 is supported, and there is a significant relationship between early cumulative ACEs and adolescent substance use. This finding is supported by previous research on ACEs and adolescent substance use (Brown and Shillington 2017; Leban and Gibson 2020).

The analysis results in Table 6 do not support Hypothesis 1. For the models in Table 6, major predictors of adolescent substance use at year 15 were added to the analyses in addition to the previously included early cumulative ACEs, year 9 ACEs, and demographic variables. Across the full sample, the female sample, and the male sample, none of the early cumulative ACEs were significantly related to adolescent substance use. These results seemingly contradict the earlier findings in Tables 3-5 as well as the findings of previous studies. Other research has found a statistically significant relationship between ACEs and adolescent substance use even when statistically adjusting for other associated factors (Brown and Shillington 2017; Fite et al. 2015; Yilmaz et al. 2015). A review of 21st-Century literature focused on strain and adolescent substance use suggests that other major predictors of adolescent substance use (beyond strain) act as mediating variables between strain and adolescent substance use (Hoffmann and Jones 2022). Based on this review, it is probably that the major predictors of adolescent substance use included in the models of Table 6 are acting as mediators between early cumulative ACEs and adolescent substance use. It is important to note that the current study cannot establish time order for the major predictors of adolescent substance use and the adolescent substance use variables because they were measured in the same year (year 15). Because the time order of these variables is entangled together, it is beyond the capabilities of the current study to determine if the major predictor variables are indeed acting as mediating variables. This limitation of the

current study gives more weight to the previous findings in Tables 3-5 that there is indeed a statistically significant relationship between early cumulative ACEs and adolescent substance use.

Hypothesis 2 was supported by the results shown in Tables 7-9. Specifically, high late adversity, intermittent high adversity, and chronic high adversity were significantly related to adolescent substance use for the full model and the female model in Table 7. Year 9 cumulative ACEs were included in Table 8 analyses. High late adversity, intermittent high adversity, and chronic high adversity remained statically significant for the full model at the same significance thresholds. For females, the significance levels decreased with only intermittent high adversity and chronic high adversity remaining significantly related to adolescent substance use. With the additional of demographic variables in Table 9, intermittent high adversity and chronic high adversity remained statistically significant for both the full model and the female model. While the significance level did decrease as additional variables were added to the models, chronic high adversity remained statistically significant for males in Tables 7-9. The analysis results in Tables 7, 8, and 9 support Hypothesis 2 that the timing and duration of early ACEs significantly impacts adolescent substance use. Previous research accounting for the timing and duration of ACE variables on adolescent outcomes, specifically delinquency and social skills, support this conclusion (Pierce and Jones 2021; Pierce, Jones, and Holcombe 2022).

However, Hypothesis 2 was not supported by the models in Table 10. Similar to Table 6, major predictors of adolescent substance use at year 15 were added to the analyses in Table 10 in addition to timing and duration of early ACEs, year 9 ACEs, and demographic variables. Across all three samples, none of the ACE timing and duration variables were significantly related to adolescent substance use. Again, these results seemingly contradict the earlier findings in Tables

7-9. As previously explained, a recent literature review suggests that these major predictor variables often act as mediators between ACEs and adolescent substance use (Hoffmann and Jones 2022). But because the time order of the major predictors of adolescent substance use and the substance use variables cannot be distinguished, it is not possible to determine whether these specific variables act as mediators for the analyses shown in Table 10. Because previous studies which have found that other major predictors of adolescent substance use act as mediators between ACEs and adolescent substance use, it is probable that the early ACE timing and duration variables would be significantly related to adolescent substance use if the major predictors were treated as mediators.

Future studies may want to consider other major predictors of adolescent substance use as mediators between ACEs and adolescent substance use. Researchers may also consider the relationship between ACEs and adolescent substance use using middle childhood mechanisms as mediators. The developmental state of middle childhood marks a, “shift in cognition, motivation, and social behavior, with profound and wide-ranging implications for the development of personality, sex differences, and even psychopathology” (Del Giudice 2018:95). This crucial stage of development may also mediate the relationship between early ACEs and adolescent outcomes.

Finally, I examined whether the relationship between early ACEs (i.e., cumulative, timing/duration) and adolescent substance use varied by gender (Hypothesis 3). Overall, my results did not supported Hypothesis 3. There were no significant gender differences between males and females for early cumulative ACEs or timing and duration of early ACEs. Although this result did not support Hypothesis 3, there was a significant gender difference for 3 cumulative ACEs in year 9 as shown in Table 9. There were also several significant differences

between males and females for demographic variables and major predictors of adolescent substance use (see Tables 5-6 and Tables 9-10).

Using a GST framework, I expected to find significant gender differences in adolescent substance use when considering early ACE variables. Indeed, previous research (utilizing a different data set than FFCW) has found significant gender differences when examining ACEs and adolescent substance use as well as adolescent delinquency (Leban and Gibson 2020). In another study that did use FFCW data, significant gender differences were found when examining ACEs and adolescent delinquency (Pierce and Jones 2021). Overall, the findings of this study seem to contradict the GST framework. However, it is important to note that the foundational GST article proposing gender differences was theoretical and did not empirically test their claims of gender differences (Broidy and Agnew 1997). Whereas the first study to empirically examine gender differences according to GST principles found no statistically significant gender differences when examining the relationship between stressful life events and adolescent drug use (Hoffmann and Su 1997). These results reaffirm that gender differences may not exist for all strain outcomes.

Limitations

The current study had several limitations. One such limitation is the absence of a sexual abuse measure which was included in the original ACE study (Felitti et al. 1998) but was not collected in the FFCW study. Therefore, this measure could not be included in my conceptualization and measurement of ACEs. Research suggests that females experience higher rates of sexual abuse and sexual assault in childhood than males (Bryant, Coman, and Damian 2020; Mersky et al. 2021). This may impact my ability to accurately measure gender differences in ACE outcomes. Future studies that include sexual abuse measures when available to

understand the impact of sexual abuse on adolescent substance use as well as gender differences would be beneficial.

The study was also limited by the somewhat narrow definition of ACEs. I only included 8 categories of ACEs in the analysis, modeled on the original ACE study. However, further research has found several other categories of ACEs to be important factors including death in the family, exposure to gun violence, or racial trauma (Bernard et al. 2021; Choi et al. 2020; Rajan et al. 2019). There may be other categories of childhood trauma that significantly impact adolescent substance use that were not included in the analysis. Other analyses can explore how these different areas of childhood trauma impact adolescent substance use and subsequent gender differences.

I was also limited in the operationalization of the adolescent substance use variable. Based on the data, I was able to include several different measures of adolescent substance use, including alcohol use, tobacco use, and illicit drug use. When examining the distribution of the substance use measure as a count variable, there were very few adolescents who had participated in many different forms of substance use. The results were not meaningful when treating adolescent substance use as a count variable due to its distribution. Therefore, I operationalized adolescent substance use as a dichotomous variable indicating any substance use and no substance use. Using a dichotomous variable for substance use is less specific than a count variable and limits the information I was able to gather from the models, but it was necessary because of the distribution of the measure (see Appendix A).

CONCLUSION

Consistently, ACE research suggests that ACEs are significantly related to many negative outcomes in adolescence and beyond (Felitti et al. 1998; Houtepen et al. 2020; Li et al. 2021).

The findings of this study suggest that cumulative early ACEs as well as timing and duration of early ACEs are significantly related to adolescent substance use (see Tables 3-5 and Tables 7-9). However, more research is needed to fully understand how mediating factors like major predictors of adolescent substance use and middle-childhood mechanisms may impact this relationship (see Tables 6 and 10). The hypotheses of this study can be modified for future research that examines the relationship between early ACEs and adolescent substance use. While GST and some previous empirical evidence suggest that ACEs have gendered outcomes, this study did not corroborate these findings. In the emerging field of ACE research, the current findings regarding early ACEs and adolescent substance use as well as the possible impact of mediators and role of gender should be carefully considered.

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TABLES

Table 1. Sample Characteristics by Youth's Gender

	Category (min, max)	Full Sample (N = 3,444)		Females (N = 1,678)		Males (N = 1,766)	
		%	(SE)	%	(SE)	%	(SE)
Adolescent Gender (Base)	(0,1)	--	--	48.72	(.01)	51.28	(.01)
Adolescent Age ^a (Y15)		15.59	(.01)	15.58	(.02)	15.60	(.02)
Adolescent Self-Control Scale ^a (Y15)		8.82	(.07)	8.72	(.11)	8.92	(.10)
Adolescent ADHD Diagnosis (Y15)	(0,1)	16.25	(.01)	9.40	(.01)	22.75	(.01)
Peer Substance Use Scale ^a (Y15)		1.29	(.03)	1.35	(.05)	1.23	(.05)
Mother's Age at Focal Child Birth ^a (Base)		25.12	(.10)	25.23	(.15)	25.02	(.14)
Mother Married at Focal Child's Birth (Base)	(0,1)	24.42	(.01)	24.11	(.01)	24.71	(.01)
Adolescent Race/Ethnicity (Y15)	White	18.09	(.01)	18.31	(.01)	17.88	(.01)
	Black	48.94	(.01)	49.54	(.01)	48.37	(.01)
	Hispanic	24.95	(.01)	24.57	(.01)	25.30	(.01)
	Other	8.02	(.01)	7.58	(.01)	8.44	(.01)
Above Poverty Level (Base)	(0,1)	38.91	(.01)	39.09	(.01)	38.73	(.01)
Parental Supervision Scale ^a (Y15)		4.39	(.02)	4.47	(.03)	4.32	(.03)
Mother-Child Relationship Scale ^a (Y15)		4.31	(.03)	4.21	(.04)	4.41	(.04)
Father-Child Relationship Scale ^a (Y15)		2.65	(.04)	2.41	(.06)	2.88	(.06)
Mother's Education (Base)	Educ < HS	31.86	(.01)	32.26	(.01)	31.47	(.01)
	Educ = HS	31.84	(.01)	31.76	(.01)	31.92	(.01)
	Educ > HS	36.30	(.01)	35.98	(.01)	36.61	(.01)

Source: Fragile Families and Child Wellbeing Study

25 multiple imputed data sets with 100 burn-ins were used to adjust for missing data in the variables.

Reference categories: white and mother's education < HS.

ABBREVIATION: SE = standard error, Y15 = year 15, Base = baseline or focal child's birth, ADHD = Attention-deficit/hyperactivity disorder.

^a Mean Reported

Table 2. ACEs and Adolescent Substance Use by Youth's Gender

	Full Sample <i>(N = 3,444)</i>		Females <i>(N = 1,678)</i>		Males <i>(N = 1,766)</i>	
	%	(SE)	%	(SE)	%	(SE)
<u>Cumulative ACEs (by Y5)</u>						
0	10.52	(.01)	11.09	(.01)	9.97	(.01)
1	18.61	(.01)	18.23	(.01)	18.98	(.01)
2	20.14	(.01)	20.53	(.01)	19.77	(.01)
3	19.86	(.01)	20.92	(.01)	18.86	(.01)
4+	30.87	(.01)	29.22	(.01)	32.43	(.01)
<u>Cumulative ACEs (Y9)</u>						
0	24.96	(.01)	24.36	(.01)	25.54	(.01)
1	30.97	(.01)	31.38	(.01)	30.58	(.01)
2	23.20	(.01)	23.52	(.01)	22.89	(.01)
3	12.70	(.01)	13.05	(.01)	12.36	(.01)
4+	8.17	(.01)	7.69	(.01)	8.63	(.01)
<u>Timing/Duration of ACEs (by Y5)</u>						
No or Low Adversity	9.57	(.01)	10.15	(.01)	9.02	(.01)
High Early Adversity	20.78	(.01)	21.64	(.01)	19.95	(.01)
High Late Adversity	13.81	(.01)	13.08	(.01)	14.50	(.01)
Intermittent High Adversity	7.15	(.00)	7.74	(.01)	6.59	(.01)
Chronic High Adversity	48.69	(.01)	47.38	(.01)	49.93	(.01)
<u>Youth Substance Use (Y15)</u>						
Percentage Reporting Substance Use	28.86	(.01)	27.10	(.01)	30.54	(.01)

Source: Fragile Families and Child Wellbeing Study

25 multiple imputed data sets with 100 burn-ins were used to adjust for missing data in the variables.

Reference categories: zero ACEs by year 5, zero ACEs in year 9, and no or low adversity.

ABBREVIATION: SE = standard error, ACEs = adverse childhood experiences, Y5 = year 5, Y9 = year 9, Y15 = year 15.

Youth Substance Use comprised of alcohol use, tobacco use, marijuana use, illicit drug use, and prescription drug misuse.

Table 3. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Accumulation of ACEs (by Year 5) and Gender

	Model 1		Model 2		Model 3		Diff Test
	Full (<i>N</i> = 3,444)	(<i>SE</i>)	Females (<i>N</i> = 1,678)	(<i>SE</i>)	Males (<i>N</i> = 1,766)	(<i>SE</i>)	
<u>Cumulative ACEs (by Y5)</u>							
1	1.00	(.19)	0.89	(.24)	1.09	(.28)	<i>NS</i>
2	1.68**	(.28)	1.84*	(.45)	1.53	(.37)	<i>NS</i>
3	1.80***	(.31)	2.04**	(.49)	1.59*	(.37)	<i>NS</i>
4+	2.50***	(.40)	2.43***	(.56)	2.52***	(.56)	<i>NS</i>
Odds of Use for Baseline	0.24***	(.03)	0.21***	(.04)	0.26***	(.05)	<i>NS</i>

Source: Fragile Families and Child Wellbeing Study

Note. The results are from logistic regression models that regressed adolescent substance use on ACEs with coefficients in odds ratios. 25 multiple imputed data sets with 100 burn-ins were used to adjust for missing data in the variables.

Reference category: zero ACEs by year 5.

ABBREVIATION: SE = standard error, ACEs = adverse childhood experiences, Y5 = year 5, Diff Test = difference test, NS = not significant difference test.

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

* $p \leq .05$ for Paternoster et al.'s (1998) test of equality for regression coefficients for males and females using the untransformed coefficient

Table 4. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Accumulation of ACEs (by Year 5 and in Year 9) and Gender

	Model 1		Model 2		Model 3		Diff Test
	Full (N = 3,444)	(SE)	Females (N = 1,678)	(SE)	Males (N = 1,766)	(SE)	
<u>Cumulative ACEs (by Y5)</u>							
1	0.99	(.19)	0.86	(.23)	1.08	(.28)	NS
2	1.60**	(.27)	1.70*	(.42)	1.49	(.36)	NS
3	1.65**	(.29)	1.78*	(.44)	1.52	(.35)	NS
4+	2.15***	(.36)	1.98**	(.47)	2.29***	(.53)	NS
<u>Cumulative ACEs (Y9)</u>							
1	1.05	(.13)	1.07	(.21)	1.05	(.17)	NS
2	1.25	(.16)	1.45	(.29)	1.09	(.19)	NS
3	1.41*	(.20)	1.71*	(.37)	1.20	(.25)	NS
4+	1.63**	(.27)	1.88*	(.50)	1.43	(.32)	NS
Odds of Use for Baseline	0.22***	(.03)	0.19***	(.04)	0.25***	(.05)	NS

Source: Fragile Families and Child Wellbeing Study

Note. The results are from logistic regression models that regressed adolescent substance use on ACEs with coefficients in odds ratios. 25 multiple imputed data sets with 100 burn-ins were used to adjust for missing data in the variables.

Reference categories: zero ACEs by year 5 and zero ACEs in year 9.

ABBREVIATION: SE = standard error, ACEs = adverse childhood experiences, Y5 = year 5, Y9 = year 9, Diff Test = difference test, NS = not significant difference test.

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

* $p \leq .05$ for Paternoster et al.'s (1998) test of equality for regression coefficients for males and females using the untransformed coefficient

Table 5. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Accumulation of ACEs (by Year 5 and in Year 9), Demographics, and Gender

	Model 1		Model 2		Model 3		Diff Test
	Full (N = 3,444)	(SE)	Females (N = 1,678)	(SE)	Males (N = 1,766)	(SE)	
<u>Cumulative ACEs (by Y5)</u>							
1	1.00	(.19)	0.87	(.24)	1.09	(.29)	NS
2	1.50*	(.26)	1.64*	(.41)	1.38	(.35)	NS
3	1.54*	(.28)	1.75*	(.45)	1.33	(.33)	NS
4+	1.95***	(.34)	1.88*	(.47)	2.02**	(.50)	NS
<u>Cumulative ACEs (Y9)</u>							
1	1.01	(.13)	1.04	(.21)	1.00	(.17)	NS
2	1.20	(.15)	1.40	(.28)	1.03	(.18)	NS
3	1.34*	(.20)	1.67*	(.37)	1.08	(.23)	NS
4+	1.57**	(.27)	1.82*	(.49)	1.41	(.33)	NS
<u>Demographics</u>							
Adolescent Gender	0.85*	(.07)	--	--	--	--	
Adolescent Age	1.63***	(.08)	1.54***	(.11)	1.74***	(.12)	NS
Mother's Age at Focal Child's Birth	1.00	(.01)	0.99	(.01)	1.02	(.01)	*
Mother's Marital Status	0.71**	(.08)	0.79	(.13)	0.65**	(.11)	NS
Adolescent Race/Ethnicity							
Black	0.73*	(.09)	0.56**	(.10)	0.97	(.17)	*
Hispanic	0.89	(.12)	0.65*	(.12)	1.22	(.23)	*
Other	0.93	(.16)	0.78	(.20)	1.14	(.28)	NS
Above Poverty Line at Birth	0.90	(.09)	0.88	(.12)	0.91	(.12)	NS
Mother's Education							
High School or Equivalent	0.92	(.09)	0.95	(.14)	0.90	(.12)	NS
Greater than High School	0.84	(.09)	0.98	(.16)	0.73*	(.11)	NS
Odds of Use for Baseline	< 0.00***	(.00)	< 0.00***	(.00)	< 0.00***	(.00)	NS

Source: Fragile Families and Child Wellbeing Study

Note. The results are from logistic regression models that regressed adolescent substance use on ACEs with coefficients in odds ratios. 25 multiple imputed data sets with 100 burn-ins were used to adjust for missing data in the variables.

Reference categories: zero ACEs by year 5, zero ACEs in year 9, male, White, below poverty line at birth, mother not married to biological father at child's birth, mother's educ < high school.

ABBREVIATION: SE = standard error, ACEs = adverse childhood experiences, Y5 = year 5, Y9 = year 9, Diff Test = difference test, NS = not significant difference test.

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

* $p \leq .05$ for Paternoster et al.'s (1998) test of equality for regression coefficients for males and females using the untransformed coefficients

Table 6. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Accumulation of ACEs (by Year 5 and in Year 9), Demographics, Major Predictors of Adolescent Substance Use (in Year 15), and Gender

	Model 1		Model 2		Model 3		Diff Test
	Full (N = 3,444)	(SE)	Females (N = 1,678)	(SE)	Males (N = 1,766)	(SE)	
<u>Cumulative ACEs (by Y5)</u>							
1	0.91	(.21)	0.83	(.28)	1.00	(.30)	NS
2	1.26	(.26)	1.39	(.42)	1.18	(.34)	NS
3	1.21	(.26)	1.43	(.44)	1.03	(.29)	NS
4+	1.37	(.27)	1.36	(.41)	1.42	(.39)	NS
<u>Cumulative ACEs (Y9)</u>							
1	0.99	(.15)	0.96	(.22)	1.01	(.20)	NS
2	1.18	(.17)	1.39	(.32)	1.01	(.21)	NS
3	1.37	(.24)	1.63	(.44)	1.14	(.28)	NS
4+	1.50*	(.29)	1.74	(.53)	1.37	(.36)	NS
<u>Demographics</u>							
Adolescent Gender	0.73**	(.07)	--	--	--	--	
Adolescent Age	1.49***	(.09)	1.40***	(.12)	1.57***	(.13)	NS
Mother's Age at Focal Child's Birth	1.01	(.01)	0.99	(.01)	1.02*	(.01)	*
Mother's Marital Status	0.77	(.10)	0.86	(.17)	0.73	(.14)	NS
Adolescent Race/Ethnicity							
Black	0.74*	(.11)	0.58**	(.12)	0.94	(.19)	*
Hispanic	0.85	(.14)	0.70	(.16)	1.04	(.23)	NS
Other	0.74	(.15)	0.59	(.18)	0.94	(.27)	NS
Above Poverty Line at Birth	0.81	(.09)	0.81	(.13)	0.81	(.12)	NS
Mother's Education							
High School or Equivalent	0.92	(.10)	0.91	(.15)	0.94	(.15)	NS
Greater than High School	0.83	(.11)	1.00	(.19)	0.69*	(.12)	*
<u>Major Predictors (Y15)</u>							
Self-Control Index	1.07***	(.01)	1.08***	(.02)	1.07***	(.02)	NS
Adolescent ADHD	1.11	(.14)	1.01	(.23)	1.18	(.18)	NS
Peer Substance Use	1.79***	(.05)	1.75***	(.07)	1.83***	(.08)	NS
Parental Supervision Index	0.87**	(.04)	0.77***	(.05)	0.96	(.06)	*
Mother-Child Closeness	0.97	(.03)	1.01	(.04)	0.93	(.04)	*
Father-Child Closeness	0.95*	(.02)	0.97	(.03)	0.93**	(.03)	NS
Odds of Use for Baseline	< 0.00***	(.00)	< 0.00***	(.00)	< 0.00***	(.00)	NS

Source: Fragile Families and Child Wellbeing Study

Note. The results are from logistic regression models that regressed adolescent substance use on ACEs with coefficients in odds ratios. 25 multiple imputed data sets with 100 burn-ins were used to adjust for missing data in the variables.

Reference categories: zero ACEs by year 5, zero ACEs in year 9, male, White, below poverty line at birth, mother not married to biological father at child's birth, mother's educ < high school

ABBREVIATION: SE = standard error, ACEs = adverse childhood experiences, Y5 = year 5, Y9 = year 9, Y15 = year 15, ADHD = Attention-deficit/hyperactivity disorder, Diff Test = difference test, NS = not significant difference test.

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

*p<.05 for Paternoster et al.'s (1998) test of equality for regression coefficients for males and females using the untransformed coefficient

Table 7. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Timing and Duration of ACEs (by Year 5) and Gender

	Model 1		Model 2		Model 3		Diff Test
	Full (N = 3,444)	(SE)	Females (N = 1,678)	(SE)	Males (N = 1,766)	(SE)	
<u>Timing/Duration of ACEs (by Y5)</u>							
High Early Adversity	1.16	(.22)	1.23	(.33)	1.09	(.30)	NS
High Late Adversity	1.57*	(.31)	1.86*	(.53)	1.34	(.38)	NS
Intermittent High Adversity	1.95**	(.42)	2.28**	(.69)	1.68	(.53)	NS
Chronic High Adversity	2.48***	(.41)	2.53***	(.61)	2.41***	(.56)	NS
Odds of Use for Baseline	0.22***	(.03)	0.19***	(.04)	0.25***	(.06)	NS

Source: Fragile Families and Child Wellbeing Study

Note. The results are from logistic regression models that regressed adolescent substance use on ACEs with coefficients in odds ratios. 25 multiple imputed data sets with 100 burn-ins were used to adjust for missing data in the variables.

Reference category: no or low adversity.

ABBREVIATION: SE = standard error, ACEs = adverse childhood experiences, Y5 = year 5, Diff Test = difference test, NS = not significant difference test.

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

* $p \leq .05$ for Paternoster et al.'s (1998) test of equality for regression coefficients for males and females using the untransformed coefficient

Table 8. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Timing and Duration of ACEs (by Year 5), Cumulative ACEs (in Year 9), and Gender

	Model 1		Model 2		Model 3		Diff Test
	Full (N = 3,444)	(SE)	Females (N = 1,678)	(SE)	Males (N = 1,766)	(SE)	
<u>Timing/Duration of ACEs (by Y5)</u>							
High Early Adversity	1.09	(.21)	1.11	(.30)	1.07	(.30)	NS
High Late Adversity	1.49*	(.30)	1.72	(.49)	1.30	(.37)	NS
Intermittent High Adversity	1.77**	(.39)	2.02*	(.62)	1.56	(.50)	NS
Chronic High Adversity	2.17***	(.37)	2.11**	(.52)	2.21**	(.53)	NS
<u>Cumulative ACEs (Y9)</u>							
1	1.08	(.14)	1.12	(.22)	1.06	(.17)	NS
2	1.33*	(.17)	1.56*	(.31)	1.16	(.19)	NS
3	1.50**	(.22)	1.85**	(.39)	1.25	(.26)	NS
4+	1.79***	(.29)	2.12**	(.54)	1.55*	(.37)	NS
Odds of Use for Baseline	0.20***	(.03)	0.19***	(.04)	0.24***	(.06)	NS

Source: Fragile Families and Child Wellbeing Study

Note. The results are from logistic regression models that regressed adolescent substance use on ACEs with coefficients in odds ratios. 25 multiple imputed data sets with 100 burn-ins were used to adjust for missing data in the variables.

Reference categories: no or low adversity and zero ACEs in year 9.

ABBREVIATION: SE = standard error, ACEs = adverse childhood experiences, Y5 = year 5, Y9 = year 9, Diff Test = difference test, NS = not significant difference test.

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

* $p \leq .05$ for Paternoster et al.'s (1998) test of equality for regression coefficients for males and females using the untransformed coefficient

Table 9. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Timing and Duration of ACEs (by Year 5), Cumulative ACEs (in Year 9), Demographics, and Gender

	Model 1		Model 2		Model 3		Diff Test
	Full (N = 3,444)	(SE)	Females (N = 1,678)	(SE)	Males (N = 1,766)	(SE)	
<u>Timing/Duration of ACEs (by Y5)</u>							
High Early Adversity	1.02	(.20)	1.08	(.30)	0.94	(.27)	NS
High Late Adversity	1.47	(.30)	1.71	(.50)	1.26	(.36)	NS
Intermittent High Adversity	1.59*	(.36)	1.89*	(.61)	1.37	(.45)	NS
Chronic High Adversity	1.91***	(.34)	1.94**	(.50)	1.88**	(.48)	NS
<u>Cumulative ACEs (Y9)</u>							
1	1.04	(.14)	1.09	(.22)	1.01	(.17)	NS
2	1.26	(.16)	1.51*	(.30)	1.08	(.19)	NS
3	1.41*	(.21)	1.80**	(.39)	1.11	(.24)	*
4+	1.70**	(.29)	2.03**	(.53)	1.51	(.34)	NS
<u>Demographics</u>							
Adolescent Gender	0.86	(.07)	--	--	--	--	
Adolescent Age	1.64***	(.08)	1.55***	(.11)	1.74***	(.12)	NS
Mother's Age at Focal Child's Birth	1.00	(.01)	0.99	(.01)	1.02	(.01)	*
Mother's Marital Status	0.70**	(.08)	0.78	(.13)	0.66**	(.11)	NS
Adolescent Race/Ethnicity							
Black	0.76*	(.10)	0.59**	(.10)	1.00	(.18)	*
Hispanic	0.92	(.12)	0.68*	(.13)	1.25	(.24)	*
Other	0.99	(.17)	0.82	(.21)	1.21	(.30)	NS
Above Poverty Line at Birth	0.89	(.08)	0.88	(.12)	0.90	(.12)	NS
Mother's Education							
High School or Equivalent	0.93	(.09)	0.95	(.14)	0.91	(.12)	NS
Greater than High School	0.85	(.09)	0.97	(.16)	0.74	(.12)	NS
Odds of Use for Baseline	< 0.00***	(.00)	< 0.00***	(.00)	< 0.00***	(.00)	NS

Source: Fragile Families and Child Wellbeing Study

Note. The results are from logistic regression models that regressed adolescent substance use on ACEs with coefficients in odds ratios. 25 multiple imputed data sets with 100 burn-ins were used to adjust for missing data in the variables.

Reference categories: no or low adversity, no ACEs in year 9, male, White, below poverty line at birth, mother not married to biological father at child's birth, mother's educ < high school

ABBREVIATION: SE = standard error, ACEs = adverse childhood experiences, Y5 = year 5, Y9 = year 9, Diff Test = difference test, NS = not significant difference test.

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

* $p \leq .05$ for Paternoster et al.'s (1998) test of equality for regression coefficients for males and females using the untransformed coefficient

Table 10. Logistic Regression Results in Odds Ratios Predicting Adolescent Substance Use by Timing and Duration of ACEs (by Year 5), Cumulative ACEs (in Year 9), Demographics, Major Predictors of Adolescent Substance Use (in Year 15), and Gender

	Model 1		Model 2		Model 3		Diff Test
	Full (N = 3,444)	(SE)	Females (N = 1,678)	(SE)	Males (N = 1,766)	(SE)	
<u>Timing/Duration of ACEs (by Y5)</u>							
High Early Adversity	0.87	(.19)	0.90	(.29)	0.85	(.27)	NS
High Late Adversity	1.21	(.28)	1.36	(.48)	1.11	(.36)	NS
Intermittent High Adversity	1.18	(.32)	1.57	(.61)	0.92	(.35)	NS
Chronic High Adversity	1.38	(.28)	1.43	(.43)	1.36	(.38)	NS
<u>Cumulative ACEs (Y9)</u>							
1	1.00	(.15)	0.99	(.23)	1.02	(.20)	NS
2	1.21	(.18)	1.45	(.33)	1.04	(.21)	NS
3	1.39	(.24)	1.70*	(.45)	1.16	(.29)	NS
4+	1.54*	(.30)	1.82*	(.54)	1.40	(.36)	NS
<u>Demographics</u>							
Adolescent Gender	0.74**	(.07)	--	--	--	--	NS
Adolescent Age	1.49***	(.09)	1.41***	(.12)	1.58***	(.13)	NS
Mother's Age at Focal Child's Birth	1.01	(.01)	0.99	(.01)	1.02*	(.01)	*
Mother's Marital Status	0.77	(.10)	0.85	(.17)	0.73	(.14)	NS
Adolescent Race/Ethnicity							
Black	0.77	(.11)	0.60*	(.13)	0.96	(.20)	*
Hispanic	0.88	(.14)	0.73	(.17)	1.08	(.24)	NS
Other	0.77	(.16)	0.61	(.19)	0.98	(.29)	NS
Above Poverty Line at Birth	0.80*	(.09)	0.80	(.13)	0.81	(.12)	NS
Mother's Education							
High School or Equivalent	0.94	(.11)	0.92	(.15)	0.95	(.15)	NS
Greater than High School	0.84	(.11)	1.01	(.19)	0.70*	(.12)	NS
<u>Major Predictors (Y15)</u>							
Self-Control Index	1.07***	(.01)	1.08***	(.02)	1.07***	(.02)	NS
Adolescent ADHD	1.11	(.14)	1.02	(.23)	1.18	(.18)	NS
Peer Substance Use	1.79***	(.05)	1.76***	(.07)	1.83***	(.08)	NS
Parental Supervision Index	0.87**	(.04)	0.77***	(.05)	0.96	(.06)	*
Mother-Child Closeness	0.97	(.03)	1.01	(.04)	0.93	(.04)	*
Father-Child Closeness	0.95**	(.02)	0.97	(.03)	0.94*	(.03)	NS
Odds of Use for Baseline	< 0.00***	(.00)	< 0.00***	(.00)	< 0.00***	(.00)	NS

Source: Fragile Families and Child Wellbeing Study

Note. The results are from logistic regression models that regressed adolescent substance use on ACEs with coefficients in odds ratios. 25 multiple imputed data sets with 100 burn-ins were used to adjust for missing data in the variables.

Reference categories: no or low adversity, no ACEs in year 9, male, White, below poverty line at birth, mother not married to biological father at child's birth, mother's educ < high school

ABBREVIATION: SE = standard error, ACEs = adverse childhood experiences, Y5 = year 5, Y9 = year 9, Y15 = year 15, ADHD = Attention-deficit/hyperactivity disorder, Diff Test = difference test, NS = not significant difference test.

*p ≤ .05, **p ≤ .01, ***p ≤ .001

*p ≤ .05 for Paternoster et al.'s (1998) test of equality for regression coefficients for males and females using the untransformed coefficient

APPENDIX A. DISTRIBUTIONS OF ADOLESCENT SUBSTANCE USE

Table 11. Distribution of Adolescent Substance Use Variables by Youth's Gender

	Category (min, max)	Full Sample (<i>N</i> = 3,418)		Females (<i>N</i> = 1,670)		Males (<i>N</i> = 1,748)	
		%	(<i>SE</i>)	%	(<i>SE</i>)	%	(<i>SE</i>)
Alcohol Use	(0,1)	17.00	(.38)	15.93	(.37)	18.02	(.38)
Tobacco Use	(0,1)	5.38	(.23)	3.95	(.19)	6.75	(.25)
Marijuana Use	(0,1)	21.65	(.41)	19.52	(.40)	23.68	(.43)
Illicit Drug Use	(0,1)	1.61	(.13)	1.50	(.12)	1.72	(.13)
Prescription Drug Misuse	(0,1)	2.08	(.14)	1.98	(.14)	2.17	(.15)

Source: Fragile Families and Child Wellbeing Study

Note. Adolescent substance use variables in this table were not imputed and therefore have a different sample size than the analytic sample.

ABBREVIATION: SE = standard error.

Table 12. Distribution of Adolescent Substance Use as a Count Variable by Youth's Gender

	Full Sample (<i>N</i> = 3,418)		Females (<i>N</i> = 1,670)		Males (<i>N</i> = 1,748)	
	%	(<i>SE</i>)	%	(<i>SE</i>)	%	(<i>SE</i>)
0	71.18	(0.89)	72.99	(0.83)	69.45	(.95)
1	16.00	(0.89)	15.51	(0.83)	16.48	(.95)
2	8.72	(0.89)	8.38	(0.83)	9.04	(.95)
3	2.52	(0.89)	2.16	(0.83)	2.86	(.95)
4	1.17	(0.89)	0.66	(0.83)	1.66	(.95)
5	0.41	(0.89)	0.30	(0.83)	0.51	(.95)

Source: Fragile Families and Child Wellbeing Study

Note. The count of adolescent substance use is not imputed and therefore has a different sample size than the analytic sample.

ABBREVIATION: SE = standard error.