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## Childhood and Adolescent Risk Factors for Comorbid Depression and Substance Use Disorders in Adulthood

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### Abstract

The comorbidity of major depression and substance use disorders is well documented. However, thorough understanding of prevalence and early risk factors for comorbidity in adulthood is lacking, particularly among urban African Americans. With data from the Woodlawn Study, which follows a community cohort of urban African Americans from ages 6 to 42, we identify the prevalence of comorbidity and childhood and adolescent risk factors of comorbid depression and substance use disorders, depression alone, and substance use disorders alone. Prevalence of comorbid substance use disorders and major depression in adulthood is 8.3% overall. Comorbidity in cohort men is twice that for women (11.1% vs. 5.7%). Adjusted multinomial regression models found few differences in risk factors for comorbidity compared to either major depression or a substance use disorder on its own. However, results do suggest distinct risk factors for depression without a substance use disorder in adulthood compared to a substance use disorder without depression in adulthood. In particular, low socioeconomic status and family conflict was related to increased risk of developing major depression in adulthood, while dropping out of high school was a statistically significant predictor of adult-onset substance use disorders. Early onset of marijuana use differentiated those with a substance use disorder with or without depression from those with depression without a substance use disorder in adjusted models. In conclusion, comorbid substance use disorders and depression are highly prevalent among these urban African Americans. Insight into the unique childhood and adolescent risk factors for depression compared to substance use disorders is critical to intervention development in urban communities. Results suggest that these programs must consider individual behaviors, as well as the early family dynamic.

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#### Author Disclosure

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## Keywords

African American; Comorbidity; Longitudinal Data; Major Depression; Substance Use Disorders

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## 1. Introduction

Individuals with substance use disorders often have comorbid depression, according to data from national and community studies (Compton et al., 2007; Grant, 1995; Grant et al., 2004; Kessler et al., 1996; Regier et al., 1990; Roberts et al., 2007; Swendsen et al., 1998).

Gaining a greater understanding of risk factors for comorbid depression and substance use disorders is critical, not only because of their high prevalence but also because of their negative consequences. Individuals with comorbid mental health and substance use disorders often experience severe illness, disability, and poor treatment outcomes (Burns & Teesson, 2002; Drake et al., 1996; Lehman et al., 1993; Najt et al., 2011).

While comorbid disorders are known to have devastating and pervasive effects in the general population, little is known about their prevalence among African Americans. African Americans experience high levels of social disadvantages, such as poverty, racial/ethnic stigma, and inequitable treatment (Mulia et al., 2008), and the stress that accompanies such disadvantage may be linked to increased risk of mental health and substance use problems among minority populations (Gibbons et al., 2004; Latkin & Curry, 2003; Martin et al., 2003; Mulia et al., 2008; Roxburgh, 2009; Williams et al., 2003). There is evidence that substance use and psychological disorders examined as separate problems follow a chronic and persistent pattern among African Americans (Dawson et al., 2005; Grant et al., 1997; Walsemann et al., 2009) and may be linked to more deleterious consequences compared to other racial groups (Caetano & Clark, 1998; Jackson-Triche et al., 2000; Sloan et al., 2009; Williams et al., 2007). Thus, it is critical to gain a greater understanding of the prevalence and risk factors for comorbidity among African Americans, given the existing level of chronic adversities in this population.

### 1.1. Risk Factors for Substance Use Disorders and Depression: Theoretical Explanations and Empirical Evidence

Theoretically, common factor models have been proposed to explain high rates of comorbidity between depression and substance use disorders in the general population (Caron & Rutter, 1991; Mueser et al., 1998). These models posit that both depression and substance use are rooted in the same risk factors, including a genetic vulnerability to both conditions (Cerdá, Sagdeo, Johnson, & Galea, 2010; Saraceno, Munafó, Heron, Craddock, & van den Bree, 2009; Uhl & Grow, 2004; Volkow, 2004). That is, the specific individual and familial factors that may increase the risk of developing depression may also increase the risk of developing a substance use disorder, hence the significant rates of comorbidity between these conditions. In some ways, common factors models have been proposed as an alternative to the self-medication hypothesis and hypotheses of drug-induced mental health problems; however each of these frameworks has received empirical support (Blume, Schmaling, & Marlatt, 2000; Hayatbakhsh et al., 2007; Marmorstein et al., 2011, van Laar et al., 2007; Weiss, Griffin, & Mirin, 1992), and it is likely that all may be operating to some extent (Swendsen & Merikangas, 2000).

The literature provides strong empirical support for common factor models for major depression and substance use disorders. Prominent among the shared risk factors identified are aggression, conduct problems, delinquent behavior, low socioeconomic status (SES), low school attachment, poor familial relations, family conflict, and parental history of substance use and mental health problems (Costello et al., 2008; Friis et al., 2002; Gilman et

al., 2002; Hawkins et al., 1992; Lieb et al., 2002; Lynskey et al., 2004; Moffitt et al., 2007). However, this evidence is based primarily on studies examining depression and substance use disorder risk factors separately, thus not taking into consideration the substantial overlap between depression and substance use disorders in clinical and community samples (Compton et al., 2007; Grant, 1995; Grant et al., 2004; Kessler et al., 1996; Regier et al., 1990; Roberts et al., 2007; Swendsen et al., 1998).

Recognizing the heterogeneity among individuals with depression and/or substance use disorders, Aseltine, Gore and Colten (1998) argued that risk factors may differ for those with both a substance use disorder with depression compared to those with a substance use disorder or depression without the other. Specifically, they suggest that those with co-occurring problems may have greater stress and less support than those with a single condition, positing that comorbid individuals may represent an independent subtype (Klein & Riso, 1993). Supporting this notion, theorists have stressed the importance of examining subgroups of individuals with similar mental health profiles to better understand etiological influences (Cicchetti & Rogosch, 1996; Swendsen & Merikangas, 2000). This notion of potential qualitative differences between those with a comorbid substance use disorder and depression compared to those with either condition without the other is also supported by evidence suggesting different outcomes of comorbidity compared to single conditions (Lewinsohn, Rohde, & Seeley, 1995).

As a test of this framework, in a rare investigation focusing on the association between social relationships, depressive symptoms, and substance abuse among adolescents, Aseltine and colleagues (1998) found low family support to differentiate between those with comorbid depression and substance abuse in adolescence and those with substance abuse without depression, as well as compared to those with depression without substance abuse. That is, the adolescents with comorbid depression and substance abuse had less supportive relationships with family members than both the depression only and substance abuse only groups. This study also found conflict with family members and with peers to differentiate those with depression without substance abuse from those with substance abuse without depression.

While this early work provides groundwork for increasing our understanding of risk factors for comorbid major depression and substance use disorders, the literature clearly lacks a comprehensive investigation of risk factors for comorbidity, particularly among African Americans and adults. Thus, in this paper, we aim to test whether those with comorbid substance use disorders and depression represent a qualitatively different subtype than those with depression or a substance use disorder on its own. To this end, we utilize data from the Woodlawn Study, an epidemiological prospective study of a community population of urban African Americans, to identify if the same risk factors predict comorbid major depression and substance use disorders, major depression without a substance use disorder, and a substance use disorder without major depression as suggested by common factor models, or if distinct risk factors emerge.

Previous work with the Woodlawn Study has identified predictors of young adult and midlife substance use (Fothergill & Ensminger, 2006; Fothergill et al., 2009) and of major depression by young adulthood (McCord & Ensminger, 1997) separately, finding overlap in risk factors. However, we have yet to identify predictors of comorbidity, and thus it is unclear if the individual and familial risk factors uncovered in previous work (e.g., poor classroom behavior, low SES) differentiate comorbid versus non-comorbid conditions. By using longitudinal data from a high-risk but under-investigated population, we are able to address existing gaps in the literature and extend results of our previous studies. Thus, this

study can increase our understanding of the joint versus separate risk factors associated with major depression, substance use disorders, and their comorbidity.

## 2. Method

### 2.1. Population

The Woodlawn Study is a prospective, longitudinal study consisting of a cohort of 1,242 first graders from Woodlawn, an inner-city community located on Chicago's South Side (52% female, 48% male). The study population consisted of all children who attended first grade in one of Woodlawn's 12 public and private schools in 1966–67. Only 13 families declined to participate; thus there was little selection bias. When the study began, Woodlawn was the fifth poorest community of 76 in Chicago. The population is 99% African Americans and was followed up in adolescence (1976–77, age 16, N=705, along with 939 mothers), young adulthood (age 32–33, N=952), and midlife (age 42–43, N=833). Together 1053 individuals (85% of the original cohort) were interviewed in adulthood and completed the assessments of substance use disorders and major depression.

### 2.2. Measures

Mental disorders diagnoses were based on the *Diagnostic and Statistical Manual for Mental Disorders* Revised Third Edition (DSM III-R, American Psychiatric Association (APA), 1987) for the young adulthood interview and DSM IV (APA, 2000) for midlife.

**2.2.1. Substance Use Disorder**—Adulthood assessments of abuse and dependence were modeled after modules developed for the National Comorbidity Survey (Kessler et al., 1994). The Composite International Diagnostic Interview (CIDI), a structured diagnostic interview, was designed to assess substance abuse and dependence in an interview format according to DSM criteria. Adult interviews assessed abuse and dependence of alcohol, marijuana, cocaine, heroin, analgesics, inhalants, hallucinogens, barbiturates, tranquilizers, stimulants, and sedatives. Those meeting criteria for either abuse or dependence of any substance at either interview were classified as having a lifetime substance use disorder. Age of onset was determined by the self-reported age of first symptom.

**2.2.2. Major Depressive Disorder**—The early adult interview included a module from the Michigan version of the Composite International Diagnostic Interview (CIDI-UM) to diagnose lifetime depression according to DSM-III-R criteria. The midlife interview included the CIDI depression module for generating a lifetime diagnosis of major depressive episode as defined by DSM-IV. Those meeting criteria for major depression at either interview were classified as having lifetime major depression. Age of first depressive episode was also determined.

**2.2.3. Individual Risk Factors**—We examined both behavioral indicators and psychological symptoms. For the behavioral indicators, we created a construct of *low self-control* (Gottfredson & Hirschi, 1990) based on the mean of teachers' overall assessment of first grade conduct, ranging from 0 to 3 (excellent to unsatisfactory), and restless behavior (e.g., fidgets, unable to sit still), ranging from 0 to 3 (adapting to severely maladapting). Adolescents reported their *onset of marijuana use* using the following categories: 1=never used, 2=age 15–16, 3=age 13–14, 4=12 or younger. Utilizing board of education records and self-reports in adulthood, we created a variable indicating whether the participant was a *high school dropout*.

From the adolescents' How I Feel questionnaire, we included two 7-item scales of psychological symptoms – angry/aggressive feelings and depressed feelings (Petersen &

Kellam, 1977). Each scale was calculated by summing across the items that were measured on a 6-point scale (1=not at all to 6=very, very much over the past several weeks). Items indicating *angry/aggressive feelings* ( $\alpha=0.72$ ) were: 1) I feel angry, 2) I feel like I am boiling inside, 3) When I get angry, I stay angry, 4) I lose my temper, 5) If someone insults me, I am likely to hit them, 6) I yell at people, and 7) I get into fights. Items indicating *depressive feelings* ( $\alpha=0.68$ ) were: 1) I feel sad, 2) I cry and don't know why, 3) I feel hopeless, 4) I feel ashamed of myself, 5) I feel guilty, 6) I don't feel worth much, 7) People would be better off without me.

**2.2.4. Familial Risk Factors**—In terms of family indicators, we examined an SES measure, mothers' psychological feelings, families' substance use, and family process indicators. At the first grade interview, mothers reported the *number of years of schooling* they received. Mothers reported the frequency of feeling sad and blue during the 1966 and 1976 assessments; feeling sad and blue fairly or very often at either time point was coded as *frequent depressive feelings*. For the first time, mothers reported on their own and the family's substance use at the adolescent assessment. *Family substance use* was a dichotomous variable representing whether anyone in the family (including the mother herself) used any illegal drug or drank alcohol regularly during the past year.

Adolescents reported on *family conflict* ( $\alpha=.82$ , mean of 5 items) on a 6-point scale, indicating how often they and adults in the family 1) have arguments, 2) say mean things, 3) let out hurt and angry feelings, 4) slam doors in anger, and 5) yell or shout to let off steam. In adolescence, mothers assessed their level of parental supervision surrounding school, friends, and curfew. *Low parental supervision* was defined as leaving school supervision mostly/entirely up to the child, leaving choice of friends up to child, and having no weeknight curfew or curfew after 10pm.

### 2.3. Attrition

Attrition analyses showed few differences between those who completed assessments and those who were missing. Those who completed the adolescent assessment and those missing in adolescence did not differ on such factors as gender, first grade family poverty status, family type, or teachers' ratings of classroom behavior or psychological characteristics. Mothers who were not interviewed at the adolescent assessment were more likely to have been teenage mothers and had greater residential mobility before the child's first grade year.

Further, those who completed at least one adult assessment (N=1053) did not differ significantly from those missing both adult assessments on such variables as mothers' education or their children's early classroom behavior. Individuals who participated in at least one of the adult interviews were less likely to have grown up in poverty and more likely to have graduated high school. Additional details on the study and attrition are presented elsewhere (Crum et al., 2006; Ensminger et al., 2002; Kellam et al., 1982).

### 2.4. Analytic Plan

To reduce the biases associated with attrition, we first employed multiple imputation, which has been shown to produce unbiased estimate and standard errors, to be asymptotically efficient, and to be a better approach than complete cases analysis (Graham, 2009; White, Royston, & Wood, 2009). We imputed 40 datasets as suggested by Graham and colleagues (2007) to maximize study power and maintain the original 1,242 study participants, thus imputing data from all four waves. For regression analyses, these 40 datasets were combined according to Rubin's rule (1987) using StataSE11. Next, based on diagnoses of major depression and of a substance use disorder, we created a four-level categorical variable: substance use disorder with depression, substance use disorder without depression,

depression without a substance use disorder, neither depression nor a substance use disorder. To ensure proper time order between adolescent risk factors and the outcomes for regression analyses, we omitted individuals whose onset occurred before age 17 since their adolescent assessment was conducted at age 16. This led us to exclude 4.9% of the total population (21 individuals with comorbid substance use disorders and depression, five with depression without a substance use disorder, and 35 with a substance use disorder without depression). This small proportion of early onset is consistent with literature showing an older age of onset of mental health disorders among African Americans compared to Whites (Alvanzo et al., 2001; Compton et al., 2000). The final restricted sample for regression analyses was 1,181 (613 women and 568 men). We used multinomial regression with all possible reference groups to identify those risk factors that were statistically significant predictors of major depression and substance use disorders separately and together in unadjusted (Table 2) and fully adjusted models (Table 3). The multivariate model included all risk factors considered, as well as gender. These analyses also held constant age and childhood neighborhood by design.

### 3. Results

As shown in Table 1, there were significant gender differences in the prevalence of depression and substance use disorders ( $p < 0.01$ ). Men were about twice as likely as women to have comorbid substance use disorder and major depression (11.09% vs. 5.69%). For men, it was more common to have a substance use disorder with depression than depression without a substance use disorder (11.09% vs. 4.01%). For women, we found the opposite trend with 11.11% of women meeting lifetime criteria for depression without a substance use disorder compared to 5.69% meeting lifetime criteria for depression with a substance use disorder. Almost 13% of women had a lifetime substance use disorder without depression compared to 26.08% of the men. Because of these sex differences, multivariate models adjusted for sex.

#### 3.1. Bivariate Analyses

Table 2 identifies childhood and adolescent risk factors that distinguished those with comorbid substance use disorders and major depression from those with major depression or a substance use disorder without the other condition from those with neither condition. Means are provided for continuous variables while percentages are given for categorical variables. Compared to those with neither a substance use disorder nor major depression, individuals with a substance use disorder in adulthood (with or without major depression) had statistically significantly lower self-control, began using marijuana at a younger age, and were more likely to drop out of high school. Those with major depression (either with or without a substance use disorder) had more angry/aggressive feelings as adolescents, as well as greater family conflict in adolescence. Depressed individuals without a substance use disorder had more depressive feelings as adolescents and had mothers with fewer years of schooling compared to individuals with neither condition. Neither family history of substance use nor maternal history of depressive feelings distinguished the four groups in bivariate analyses.

#### 3.2. Multivariate Analyses

Table 3 provides the multivariate multinomial regression results from a single regression model that varied the reference group. Even when adjusting for all risk factors considered, we found significant gender differences in the distribution of comorbidity. Men were significantly more likely than women to have a substance use disorder with or without depression, while women were more likely than men to have depression without a substance use disorder. Low self-control in first grade predicted the development of a substance use

disorder without major depression in adulthood compared to those with neither condition. The earlier the onset of marijuana use in adolescence, the greater the likelihood of developing a substance use disorder with or without depression compared to individuals with neither disorder. We also found the earlier the onset of marijuana use, the greater the likelihood of developing a substance use disorder alone compared to depression alone. Additionally, we found that individuals who dropped out of high school were two to two and a half times as likely to develop a substance use disorder with major depression compared to both those who were depressed without a substance use disorder and those unaffected by either disorder. Finally, individuals who had a mother with fewer years of schooling and experienced greater family conflict in adolescence were at increased risk of developing depression without a substance use disorder compared to those with a substance use disorder alone and those with neither condition.

#### 4. Discussion

This study highlights the considerable prevalence of major depression and substance use disorders and their comorbidity within an urban African American community cohort. About 35% of the cohort members (more than 41% of the men and nearly 30% of the women) have one or both of these lifetime disorders; overall, 11% of the men and 6% of the women have comorbid major depression and substance use disorder. While depression rates in this community cohort (15% of men and 17% of women) are similar to those found nationally, 37.1% of men and 18.5% of women were found to have a substance use disorder with or without depression, which is significantly higher than substance use disorder rates reported nationally (Kessler et al., 2005). It is unclear how rates of comorbidity in this community cohort compare to national estimates since comparable data are absent from the literature. Thus, a primary contribution of this study is the provision of an estimate of comorbid depression and substance use disorders in a community cohort of African American men and women.

Despite the well documented comorbidity of major depression and substance use disorders, rarely are risk factors for these two conditions examined together. We find that men and those with earlier onset of marijuana use have increased risk of these comorbid conditions, but also increased risk of substance use disorder without depression compared to those with depression alone or neither condition. Thus, both being male and having an earlier onset of marijuana use seem to be risk factors for adult substance use disorder in general, rather than being specific to comorbidity. These findings are not novel as others (e.g., Anthony & Petronis, 1995; Brook, et al., 2002) have shown that individuals who initiate marijuana use at particularly early ages are especially vulnerable to developing a substance use disorder in adulthood, as well as gender differences in substance use disorders. We extend these findings to those with comorbid substance use disorders and depression.

In examining early risk factors that may differentiate individuals with a substance use disorder with depression from those with a substance use disorder without depression, we found none that were statistically significant at the probability level of 0.05. Thus, there may not be early life risk factors that differentiate these groups. Instead, factors occurring later in the life course may influence whether an individual with a substance use disorder also meets criteria for major depression. We did identify two family factors (family conflict and lack of parental supervision) that are marginally statistically significant in the multivariate model differentiating a comorbid substance use disorder from a substance use disorder without depression, and thus, future research should consider the role of family factors in the development of comorbidity. It may also prove useful to differentiate those who develop a substance use disorder before the onset of depression from individuals who are depressed first and then develop a substance use disorder.

In contrast to the literature suggesting significant overlap in risk factors, we find distinct childhood and adolescent risk factors once we separate comorbid depression and substance use disorders from depression without a substance use disorder and from a substance use disorder without depression. For example, childhood SES, high school dropout, and family conflict distinguish adults with major depression without a substance use disorder from adults with a substance use disorder without depression. Specifically low maternal education in childhood and more family conflict in adolescence uniquely predict adult onset major depression without a substance use disorder and high school dropout uniquely predicts an adult-onset substance use disorder without depression in this population. These findings lend support to the notion of distinct etiologies of major depression and substance use disorders when examining onset in adulthood in contrast to a common factor model (Caron & Rutter, 1991). To the extent that we can generalize from our findings, we tentatively suggest that substance use disorders are more likely to be preceded by earlier behavioral problems, such as those reported by first grade teachers, early marijuana use and dropping out of high school, while family issues such as family conflict and having a mother with low education are more predictive of later depression.

Because we had only retrospective reports on the onset of substance use disorders and major depression, we were unable to determine which disorder occurred first and thus are unable to provide insight into whether substance use disorders increase the risk of later depression or if earlier depression increases the risk of later substance use disorders. However, we did not find evidence that substance use in adolescence was associated with an increased risk of later depression. Nor did we find evidence that depressive feelings in adolescence was associated with an increased the risk of later substance use. Further work is needed to investigate alternative explanations to the common factor models for comorbidity, including self-medication and depression resulting from substance use.

Our finding of low maternal education in childhood as a risk factor for only major depression, and dropping out of high school as a risk factor only for substance use disorders in both unadjusted and adjusted models is interesting since both are measures of socioeconomic well-being but at different points in the life course. Previous research has identified socioeconomic inequalities in both substance use and depression, with individuals of low SES backgrounds having greater risk for these conditions (Lorant et al., 2003; Townsend, Flisher & King, 2007). Dohrenwend and colleagues (1992) put forth evidence that the link between SES and both depression and substance use disorders is a result of social causation – that is the associated adversity of low SES increases the risk for these conditions – and suggests that mechanisms may vary by mental health condition. Consistent with this notion, coming from a family of low SES may increase the risk of depression through increased stress and adversity throughout childhood and adolescence leading to internalizing problems. Further, failing to achieve in school directly limits future social and economic opportunities, especially for those of minority backgrounds, which may increase the likelihood of becoming involved in drug use and drug markets. In fact, we previously reported high school dropout as a consequences of early marijuana use (Green & Ensminger, 2006), and thus may be a mechanism linking early substance use with later substance use disorders. Regardless of the mechanism, our findings continue to highlight the critical need to consider SES in prevention programming and to prevent high school dropout among urban African Americans.

Results also highlight the importance of addressing family conflict during adolescence as individuals with greater family conflict are more likely to develop major depression, controlling for family history, childhood and adolescent behavioral problems and adolescent depressive feelings, among other factors. This is consistent with others who have demonstrated a link between family conflict and depression over time (Gilman et al., 2003;



Sheeber et al., 1997). This also aligns with findings from Aseltine and colleagues (1998), who found in their study of a White middle class sample, that peer conflict during adolescence predicted the development of depression, but not substance use. It may be that conflict leads to poor social relationships and thus a lack of buffers against negative mood (Umberson, et al., 1996). Also individuals with more conflict in their lives may have negative cognitions that may affect the risk of depression over time. More work is needed to identify if and how family conflict during adolescence may potentially increase the risk of major depression in adulthood.

Notwithstanding the study's strengths of prospective design spanning 35 years, use of a standardized diagnostic tool, and focus on urban African Americans, there are a number of limitations to note. First, we do not have comprehensive data on family history of substance use disorders. Mothers only reported during the adolescent interview about their own drug use, as well as family substance use, which did not predict any of the outcomes. Because the Woodlawn cohort was primarily in single-mother families in both childhood (58%) and adolescence (73%) and fathers were not assessed, we lack a comprehensive understanding of paternal history of substance use and depression. We did include an assessment of mother's depressive feelings, which has been shown to be a valid and reliable measure (Brown et al., 1981), including demonstrating predictive validity in terms of negative outcomes in offspring (Green et al., 2012), but we found it did not predict adult depression either with or without a substance use disorder. Previously, we reported that mothers' persistent depressive feelings did relate to earlier onset depression (onset between ages 14 and 32) in daughters (Ensminger et al., 2003). In the current study, we focused on onset between ages 17 and 42 in order to examine adolescent risk factors and properly establish time order. Findings suggest that family history does not relate to later onset depression. Previous work supports the notion that earlier onset depression may be more rooted in family history of psychopathology than is later depression (Jaffee et al., 2002; Kessler & Magee, 1993; Klein et al., 2001; Neuman et al., 1997); however, our lack of genetic data, as well as the a limited number of case with early onset in our community cohort (defined in this study as before age 17) limits our ability to explore the influence. This is an important limitation because individuals with early onset comorbidity have been found to have the most chronic, severe disorders (Rao & Chen, 2009). Thus, it is critical for future research to identify if risk factors for early onset comorbid substance use disorders and depression (i.e., adolescent onset) differ from the risk factors found in this study for later onset comorbidity.

An additional limitation may be that, while we attempted to establish appropriate time order between the onset of disorders and adolescent risk factors, we based onset on retrospective reporting in adulthood, which may be vulnerable to forward telescoping. Thus, while we aimed to focus on adult-onset disorders, our sample may have included a few individuals whose onset was earlier, particularly since both disorders tend to develop gradually over time, and therefore, it is often difficult to pinpoint an exact age of onset. However, since analyses adjusted for both adolescent depressive symptoms and marijuana use onset, we do not expect major effects on findings from some misclassification. Regardless, our results can only suggest factors that may increase the risk of these disorders in adulthood and not evidence of causal associations.

Further, because we focused on a specific and under-investigated urban, minority community, it is unclear how well our results generalize to other populations. Generalizability to other predominantly African American, urban communities also may be limited somewhat by attrition. We also focused on lifetime substance use disorder and major depressive disorder. Thus, it is unclear if the two disorders co-occurred at the same time, and thus, our findings only apply to lifetime comorbidity. Finally, we did not investigate other

types of psychopathology, such as anxiety disorders, which frequently co-occur with substance use disorders and depression. These are all areas for future research.

Despite these limitations, this study makes notable contributions to the literature. It identifies the adulthood prevalence of comorbid substance use disorders and depression in an at-risk community cohort followed from age 6 to 42. Insight into comorbidity among urban Africans Americans is critical because its negative consequences may be further compounded by lower likelihood of receiving necessary substance use and mental health treatment, and greater likelihood of having no access to appropriate treatment services compared to Whites (Alegria et al., 2002; Alvidrez et al., 1999; Garland et al., 2005; Wells et al., 2001).

This study provides evidence of potentially distinct risk factors for a substance use disorder with and without depression and for depression without a substance use disorder. Understanding the separate antecedents for depression and substance use is essential to the successful tailoring of intervention efforts to address these prevalent and disabling conditions. In a universal prevention programming framework, all students would be served well by addressing early classroom behavior, family conflict, early adolescent drug use, and high school dropout, particularly among those of low socioeconomic status, as these risk factors are conceptually interrelated and demonstrated in this study to potentially increase the risk for later depression and substance use disorders.

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### Highlights

- Comorbid depression and substance use disorders are prevalent among urban Blacks.
- Little overlap in risk factors for depression and substance use disorders (SUDs).
- Low SES in childhood and adolescent family conflict predict adult depression.
- Dropping out of high school may increase the risk of adult substance use disorders.
- No variables differentiated comorbid depression and SUDs from each on its own.



**Table 1**

Prevalence of Comorbidity of Substance Use Disorders and Depression among the Woodlawn Study Participants by Gender (N=1181) \*

	Men	Women	Total
N	568	613	1181
Substance Use Disorder with Depression	11.09%	5.69%	8.29%
Substance Use Disorder without Depression	26.08%	12.85%	19.21%
Depression Without a Substance Use Disorder	4.01%	11.11%	7.69%
No Depression or Substance Use Disorder	58.81%	70.35%	64.80%

\* statistically significant gender differences at  $p < 0.01$  based on F-test statistic combining chi-square values across the 40 datasets using the SAS MACRO COMBCHI.

Distribution of Childhood and Adolescent Risk Factors for a Substance Use Disorder (SUD), Depression, and their Comorbidity: Means, Percentages, and Statistical Significance (N=1,181)

**Table 2**

	1 SUD with Depression	2 SUD without Depression	3 Depression without a SUD	4 Neither	Statistical Significance
<u>Individual Risk Factors</u>					
Low Self-Control <sup>a</sup>	1.25	1.35	1.22	1.07	1>4 <sup>*</sup> ; 2>4 <sup>**</sup>
Angry/Aggressive Feelings Scale <sup>b</sup>	20.95	18.99	20.87	17.84	1>4 <sup>**</sup> ; 3>4 <sup>**</sup>
Depressive Feelings Scale <sup>b</sup>	14.80	13.97	15.75	13.82	3>4 <sup>*</sup>
Onset of Marijuana Use <sup>b</sup>	1.37	1.11	0.80	0.83	1>4 <sup>**</sup> ; 2>4 <sup>**</sup> ; 1>3 <sup>**</sup> ; 2>3 <sup>*</sup>
High School Dropout <sup>c</sup>	34.12%	38.82%	25.86%	22.74%	1>4 <sup>*</sup> ; 2>4 <sup>**</sup>
<u>Familial Risk Factors</u>					
Mothers' Years of Schooling <sup>a</sup>	10.69	10.62	10.03	10.70	3<4 <sup>*</sup>
Mothers' Frequent Depressive Feelings <sup>a,b</sup>	25.59%	23.68%	27.21%	25.75%	
Family Substance Use <sup>b</sup>	41.17%	42.60%	35.93%	42.90%	
Family Conflict Scale <sup>b</sup>	4.14	3.57	4.34	3.58	1>2 <sup>*</sup> ; 1>4 <sup>*</sup> ; 3>2 <sup>**</sup> ; 3>4 <sup>**</sup>
Low Parental Supervision <sup>b</sup>	32.02%	19.17%	23.19%	20.41%	

<sup>a</sup> reported in childhood (age 6)

<sup>b</sup> reported in adolescence (age 16)

<sup>c</sup> compiled through retrospective self-reports in adulthood and Board of Education Records

\*\*  $p < 0.01$ .

\*  $p < 0.05$  based on unadjusted multinomial regression with all possible reference groups.

**Table 3**

Multivariate Multinomial Regression Analyses Predicting Comorbid Substance Use Disorders and Major Depressive Disorder from Childhood and Adolescent Risk Factors (N=1181): Odds Ratios (OR) and 95% Confidence Intervals (CI)

	SUD with Depression vs. Neither	SUD without Depression vs. Neither	Depression without a SUD vs. Neither	SUD without Depression vs. Depression without a SUD	SUD with Depression vs. Depression without a SUD	SUD with Depression vs. SUD without Depression
	OR & 95% CI	OR & 95% CI	OR & 95% CI	OR & 95% CI	OR & 95% CI	OR & 95% CI
Male	1.99 (1.15–3.45) <sup>*</sup>	2.00 (1.39–2.89) <sup>**</sup>	0.40 (0.23–0.72) <sup>**</sup>	4.95 (2.58–9.49) <sup>**</sup>	4.92 (2.36–10.26) <sup>**</sup>	0.99 (0.54–1.83)
Low Self-Control <sup>a</sup>	1.07 (0.76–1.51)	1.36 (1.09–1.70) <sup>**</sup>	1.26 (0.90–1.76)	1.08 (0.75–1.57)	0.85 (0.54–1.34)	0.79 (0.54–1.14)
Angry/Aggressive Feelings Scale <sup>b</sup>	1.04 (0.99–1.09) <sup>†</sup>	1.02 (0.99–1.06)	1.04 (0.98–1.09)	0.99 (0.93–1.05)	1.01 (0.95–1.07)	1.02 (0.97–1.07)
Depressive Feelings Scale <sup>b</sup>	1.00 (0.95–1.06)	0.99 (0.95–1.03)	1.02 (0.96–1.07)	0.97 (0.92–1.04)	0.98 (0.92–1.05)	1.01 (0.95–1.07)
Onset of Marijuana Use <sup>b</sup>	1.63 (1.19–2.24) <sup>**</sup>	1.26 (1.00–1.59) <sup>*</sup>	0.98 (0.69–1.38)	1.29 (0.87–1.92)	1.67 (1.12–2.48) <sup>*</sup>	1.29 (0.93–1.80)
High School Dropout <sup>c</sup>	1.31 (0.73–2.35)	2.01 (1.31–3.08) <sup>**</sup>	0.80 (0.41–1.53)	2.53 (1.25–5.11) <sup>*</sup>	1.64 (0.74–3.64)	0.65 (0.35–1.22)
Mothers' Years of Schooling <sup>a</sup>	1.02 (0.91–1.14)	1.00 (0.93–1.09)	0.89 (0.81–0.99) <sup>*</sup>	1.12 (1.00–1.26) <sup>*</sup>	1.14 (0.99–1.32) <sup>†</sup>	1.02 (0.89–1.16)
Mothers' Frequent Depressive Feelings <sup>a,b</sup>	0.92 (0.47–1.80)	0.83 (0.54–1.29)	0.89 (0.48–1.65)	0.93 (0.45–1.92)	1.03 (0.45–2.36)	1.11 (0.53–2.29)
Family Substance Use <sup>b</sup>	0.84 (0.47–1.49)	0.95 (0.66–1.37)	0.68 (0.38–1.23)	1.39 (0.72–2.66)	1.22 (0.57–2.62)	0.88 (0.49–1.60)
Family Conflict Scale <sup>b</sup>	1.22 (0.94–1.58)	0.94 (0.80–1.10)	1.33 (1.02–1.74) <sup>*</sup>	0.70 (0.52–0.96) <sup>*</sup>	0.91 (0.67–1.24)	1.29 (0.99–1.69) <sup>†</sup>
Low Parental Supervision <sup>b</sup>	1.43 (0.73–2.78)	0.71 (0.43–1.17)	1.35 (0.64–2.86)	0.52 (0.22–1.24)	1.06 (0.46–2.43)	2.02 (0.95–4.29) <sup>†</sup>

<sup>a</sup> reported in childhood (age 6)

<sup>b</sup> reported in adolescence (age 16)

<sup>c</sup> compiled through retrospective self-reports in adulthood and Board of Education Records

<sup>\*\*</sup> p<0.01,

<sup>\*</sup> p<0.05,

<sup>†</sup> p<0.10