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Phenotypic and familial associations between childhood maltreatment and cannabis initiation and problems in young adult European-American and African-American women*

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Abstract

Background—Childhood maltreatment is a known risk factor for cannabis initiation and problem use, but the extent to which this association is attributable to shared familial influences is unknown. We estimate the magnitude of associations between childhood maltreatment, timing of cannabis initiation, and cannabis-related problems, in European-American (EA) and African-American (AA) women, and parse the relative influence of additive genetic (A), shared environmental (C), and individual-specific environmental (E) factors on these constructs and their covariation.

Methods—Data were from diagnostic telephone interviews conducted with 3,786 participants (14.6% AA) in a population-based study of female twins. Logistic regression analyses and twin modeling were used to test for associations, and estimate the relative contributions of genetic and environmental influences to childhood maltreatment and cannabis outcomes and their covariation.

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Author Disclosures

Contributors

JDG, CES, and AA conceived of and conducted all analyses and prepared the first and all revised drafts. ACH, KKB and PAFM designed the MOAFTS study and oversaw data collection. ACH consulted on analyses. KKB provided key insights into study of ethnic variation for substance use. ECN, KBW, and VVM provided critical feed-back on ethnic variation for childhood maltreatment. All authors have participated in manuscript preparation and approved the final submission.

Conflicts of Interest

No conflicts to declare.

Results—Maltreatment was significantly associated with increased likelihood of cannabis initiation before age 15 among EAs (OR=6.33) and AAs (OR=3.93), but with increased likelihood of later initiation among EAs only (OR=1.68). Maltreatment was associated with cannabis problems among both groups (EA OR=2.32; AA OR=2.03). Among EA women, the covariation between maltreatment and cannabis outcomes was primarily attributable to familial environment ($r^2=0.67-0.70$); among AAs, only individual-specific environment contributed ($r^2=0.37-0.40$).

Conclusion—Childhood maltreatment is a major contributor to early initiation of cannabis as well as progression to cannabis problems in both AA and EA women. Distinctions by race/ethnicity are not in the relative contribution of genetic factors, but rather in the type of environmental influences that contribute to stages of cannabis involvement.

Keywords

cannabis initiation; cannabis problems; childhood maltreatment; race/ethnicity; twins; heritability

1. Introduction

Childhood maltreatment – experiencing sexual abuse, physical abuse or neglect in childhood – has long been established as a risk factor for early cannabis initiation (Bensley et al., 1999; Harrison et al., 1997; Nelson et al., 2006; Sartor et al., 2015; Werner et al., 2016) and cannabis use disorder (CUD) (Duncan et al., 2008; Kendler et al., 2000; Rogosch et al., 2010). Data from the National Epidemiologic Survey of Alcohol and Related Conditions demonstrate the robustness of these associations: elevated risk for CUD was observed in individuals who reported childhood physical abuse, sexual abuse, or neglect even after adjusting for demographic characteristics and a wide range of psychiatric conditions (Afifi et al., 2012). There is also evidence that the magnitude and stability of the associations between childhood maltreatment and cannabis initiation and the transition to problem use differ between African-Americans (AAs) and European-Americans (EAs) (Sartor et al., 2015). In addition, in a recent study by our group using the current sample to examine specific traumatic experiences in relation to the timing of cannabis initiation, childhood sexual abuse predicted cannabis initiation after adjusting for correlated psychiatric conditions in both AAs and EAs, but physical abuse predicted initiation only in EAs (Werner et al., 2016).

Racial/ethnic differences in the prevalence and patterns of cannabis use onset and CUD have been documented. Although AAs historically have been less likely than their non-Hispanic EA counterparts to report a lifetime history of cannabis use, this difference has gradually decreased (Johnston et al., 2014), with some studies reporting little to no difference (Ewing, 2002; Keyes et al., 2015; Shih et al., 2010). Furthermore, recent research suggests AAs are more likely to report use in the past year (Hasin et al., 2015b). Similarly, while the lifetime prevalence of CUD is comparable across EAs and AAs (Hasin et al., 2015a), the most recent data indicate that CUD is now more common in AAs than EAs (Hasin et al., 2015a; Wu et al., 2016). By contrast, evidence that rates of childhood maltreatment are greater in AAs compared to EAs is unequivocal. Numerous studies have found the prevalence of child abuse or neglect in AAs to be twofold or greater than that among EAs (Lee et al., 2012; Wildeman et al., 2014; Wulczyn, 2009).

Given the escalating rates of cannabis use and CUD among AAs and the greater likelihood of exposure to childhood maltreatment in AAs relative to EAs, studies that focus on racial/ethnic similarities and differences in the nature of the association between cannabis involvement and childhood maltreatment are necessary. One aspect of this research that has been notably overlooked is the extent to which common familial influences might contribute to any observed associations. Childhood maltreatment frequently occurs in the context of familial risk factors, such as parental conflict or separation (Bidarra et al., 2016; Hindley et al., 2006) and parental alcohol problems (McLaughlin et al., 2000; Shin et al., 2009), which are also associated with early initiation of marijuana use (Waldron et al., 2014a; Waldron et al., 2014b) and CUD (Melchior et al., 2011; Schiff et al., 2014). Furthermore, there is evidence that experiences of childhood maltreatment cluster within pairs of identical and fraternal twins that are raised together (Schulz-Heik et al., 2010; Young-Wolff et al., 2011), although the extent to which these familial factors are environmental or genetic has not been explored. Familial factors, both genetic and environmental, play a prominent role in the etiology of cannabis use, while familial environmental factors appear to be less influential for cannabis-related problems. In Verweij et al.'s meta-analysis of twin studies of cannabis use and problem use (2010), estimates of genetic influences on the two outcomes were 40–48% and 51–59%, respectively, whereas estimates of shared environmental influences were 25–39% for initiation but only 15–20% for problem use. Nonetheless, the extent to which associations between cannabis use, CUD and childhood maltreatment can be attributed to common familial factors, either genetic or shared environmental, and to individual-specific experiences remains unknown.

In addition, nearly all prior family and twin studies of cannabis involvement have been conducted in samples of European descent. Two publications utilizing the data analyzed in the present study are amongst the few to document that cannabis use is equally heritable in AA and EA women (Agrawal et al., 2016; Sartor et al., 2009), but that individual-specific environmental influences on cannabis use are more prominent in AAs compared to EAs. Thus, it is possible that the extent to which familial and individual-specific factors influence the correlation between cannabis involvement and childhood maltreatment also differs across racial/ethnic groups.

The current investigation was designed to address two aims, using data from an all-female twin sample aged 18–27 years. The first was to examine the magnitude of phenotypic associations between childhood maltreatment and two stages of cannabis involvement, initiation and development of cannabis-related problems, in AA and EA women. The second, following up on phenotypic findings, was to estimate the relative influence of additive genetic, shared environmental, and individual-specific environmental factors on childhood maltreatment, cannabis outcomes, and their covariation. The latter aim allowed us to estimate the factors contributing to the well-documented association between childhood maltreatment and cannabis involvement.

2. Methods

2.1 Participants

The Missouri Adolescent Female Twin Study (MOAFTS) is a longitudinal study of substance use and related psychopathology in adolescent girls and young women, conducted with same-sex female twins born in Missouri to Missouri-resident parents between July 1, 1975 and June 30, 1985. Twins were identified through birth records and recruited for baseline assessment from 1995 to 1999. The sample was demographically representative of the Missouri population at the time the twins were born, with nearly 15% being AA and the remainder EA (see Heath et al., 1999, 2002; Waldron et al., 2013 for details). Wave 1 interviews were conducted with 3,258 twins (median age=15). Many but not all twin pairs from Wave 1 completed a brief 1-year follow-up (Wave 2, not a source of data for the current study), and a subsample from Wave 1 also completed a comprehensive 3-year retest interview (Wave 3, $n=1,370$, median age=19). Between 2002 and 2005, all twins from the target cohort (excluding those who had withdrawn or whose parents asked that the family not be re-contacted) were contacted for Wave 4 interviews ($n=3,787$, median age=22; 80% of the twins identified from birth records). Wave 5 interviews were conducted between 2005 and 2008 ($n=3,428$ twins; median age=24).

The base sample for present analyses was individuals who completed the Wave 4 interview, since it covered lifetime psychiatric and psychosocial information in young adulthood and had the largest number of participants (one dropped due to incomplete data). Over 95% of Wave 4 participants completed at least one additional interview (Wave 1, 3, or 5) that served as a source of data for coding variables in the current analyses. The sample had a mean age of 21.7 ($SD=2.8$) years at Wave 4, and included 1,038 monozygotic (MZ) twin pairs (911 EA, 127 AA), 734 dizygotic (DZ) twin pairs (606 EA, 128 AA) and 242 twins (199 EA, 43 AA) whose co-twin did not participate. Zygosity was determined using an algorithm based on responses to standard questions regarding similarity (Nichols and Bilbro, 1966). Zygosity for both members of 940 EA and 150 AA twin pairs was confirmed using genotyping, yielding 90.2% and 92.7% accuracy, respectively, which is broadly consistent with reports on other samples of European (Eaves et al., 1989; Reed et al., 2005) and African descent (Whitfield, 2012).

2.2. Procedures

Data were collected via telephone interview using an adaptation of the Semi-Structured Assessment for the Genetics of Alcoholism (Bucholz et al., 1994; Hesselbrock et al., 1999), which assesses *Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV* (American Psychiatric Association, 2000) psychiatric disorders, history of substance use, and related psychosocial constructs. The study was carried out in accordance with the latest version of the Declaration of Helsinki and approved by the Washington University School of Medicine Human Research Protections Office. Prior to participation, informed consent was obtained from all twins 18 years of age or older, as were parental consent and twin assent for twins under age 18.

2.3. Measures

2.3.1. Childhood Maltreatment—Childhood maltreatment, defined as childhood physical abuse, sexual abuse, or neglect occurring before age 16, was queried in the traumatic events section of Waves 1, 3, and 4 interviews. Childhood sexual abuse was also assessed in the health problems and health habits section (at Waves 1, 3, and 4) and the parental discipline and childhood experiences section (at Wave 4). Criteria were met if any qualifying item was reported as occurring before age 16 at any assessment. (See Supplemental Table S1¹ for a list of interview questions and the prevalence of each form of maltreatment).

2.3.2. Timing of Cannabis Initiation—Timing of initiation was examined as a three-level variable based on participants' responses to questions about lifetime cannabis use and age at first use: 0=never, 1= onset age 15, 2= onset < age 15 (asked of everyone at Waves 4 and 5, and those 16+ years old at Waves 1 and 3). Age 15 was chosen to distinguish early- from later-onset users because only 15% of users initiated < age 15, but 15–20% initiated use in each of the subsequent four years (ages 15–18). This definition also satisfied the multivariate normality assumption regarding the distribution of twin and co-twin data (Neale and Maes, 2004) in MZ and DZ twin pairs of both racial/ethnic groups (all $p>0.05$; range: 0.07–0.84). When reported at multiple interviews, age at initiation was derived from the first interview at which cannabis use was endorsed.

2.3.3. Problem Use of Cannabis—Cannabis users were asked if they had ever experienced any of 6 DSM-IV dependence symptoms, excluding withdrawal (Waves 4 and 5) or 4 abuse symptoms (all waves). The distribution was skewed, with 12% and 14% of users endorsing only one and 2+ symptoms respectively. To avoid making normality assumptions, endorsement of any of these 10 symptoms at any assessment was dichotomously defined as problem use. Those who did not report any cannabis use were coded as missing for cannabis problems.

2.4. Data Analysis

Phenotypic analyses testing 1) racial/ethnic differences in prevalence and 2) associations between maltreatment and the cannabis measures within race/ethnicity were conducted using SAS v9 (SAS Institute, 2008). Logistic regression analyses examining the association between childhood maltreatment and cannabis initiation and problem use were conducted separately for EA and AA participants using STATA v11 (StataCorp, 2009), with the “robust” option specified to adjust the standard errors for familial clustering. Trivariate twin models examined the relationship between childhood maltreatment, timing of initiation and cannabis problems. Models were fitted to raw ordinal data in Mx (Neale et al., 2003) using full information maximum likelihood estimation. As detailed elsewhere (Neale and Cardon, 1992), twin models allow for the estimation of additive (A) and non-additive (D) genetic factors, shared environmental factors (C; environmental influences that make twins similar to each other), and individual-specific environmental factors (E; environmental influences not shared by twins and error variance). A is indicated when the MZ twin correlation (r_{MZ})

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> DZ twin correlation (r_{DZ}), D when $r_{DZ} < \frac{1}{2} r_{MZ}$, and C when $r_{DZ} > \frac{1}{2} r_{MZ}$. E is based on the extent to which $r_{MZ} < 1.0$. Thresholds (i.e., prevalences) were adjusted for age at Wave 4 using a median split (≥ 22 vs. < 22 years; to avoid assumptions of linear relationships with age). Initially, EA and AA women were modeled simultaneously with variance components (A, C, E) and prevalences allowed to differ across racial/ethnic groups for all measures (e.g., A_{EA} A_{AA}). Racial/ethnic differences in the variance components were tested by constraining them to be equal (e.g., does $A_{EA} = A_{AA}$), and individual parameters (A, C, E) were constrained to zero to determine the significance of their contribution to the variance in and covariance between cannabis use or problems and childhood maltreatment. Relative model fit was computed using the difference in $-2 \log$ likelihood fit, which represents a χ^2 distribution for the given degrees of freedom.

3. Results

As shown in Table 1, AA women were significantly more likely to report childhood maltreatment (27.3%) than were EAs (15.6%), although age at first maltreatment did not differ by race/ethnicity ($M=8.0$ [$SD=4.0$] years for EAs, 8.3 [$SD=3.9$] years for AAs). Cannabis use was more commonly reported by AAs (57.5% vs 52.5% among EAs), although the percentages of respondents who initiated before age 15 (9.0% vs 7.8%) and who initiated age 15 or later (48.5% vs 44.7%) did not differ by race/ethnicity nor did mean age at first use (AA $M=17.1$ [$SD=2.7$], EA $M=16.8$ [$SD=2.5$]). AA cannabis users were more likely than their EA counterparts to have experienced one or more cannabis problems (35.5% vs 24.5% of users respectively). Nearly all reported ages of onset of maltreatment preceded onset of cannabis initiation. Excluding 15 individuals with cannabis initiation prior to maltreatment (difference in onset age of 1–2 years for 14 individuals, 8 years for one person) did not alter findings.

3.1 Phenotypic Associations Between Maltreatment and Cannabis Outcomes

3.1.1. Childhood Maltreatment and Timing of Cannabis Initiation—There was a moderate and significant correlation between maltreatment and cannabis initiation among EAs ($r = 0.32$ [95% Confidence Interval 0.27, 0.38]) and AAs ($r = 0.19$ [0.07, 0.32]). After controlling for age, EAs who experienced childhood maltreatment were at 6.33 greater odds (95% CI=4.55, 8.81) of reporting cannabis initiation before age 15 and at 1.68 greater odds (95% CI=1.33, 2.14) of cannabis initiation at age 15 or older than EAs without a history of maltreatment. Among AAs, maltreatment was associated with 3.93 greater odds (95% CI=1.97, 7.86) of initiation before age 15, but not increased risk of cannabis initiation at an older age (OR = 0.96; 95% CI=0.62, 1.49).

3.1.2. Childhood Maltreatment and Cannabis Problems—The correlation between childhood maltreatment and cannabis problems (among $N=1699$ EA and $N=318$ AA users) was statistically significant and equally strong in the EAs and AAs (EA $r = 0.28$ [0.20, 0.37]; AA $r = 0.26$ [0.08, 0.44]). After controlling for age, EAs and AAs who experienced childhood maltreatment were at greater odds of reporting one or more cannabis problems than their counterparts without a history of maltreatment (OR = 2.32, [1.77–3.03]; OR = 2.03, [1.23–3.35], respectively).

3.2 Familial Influences On and Overlap Between Childhood Maltreatment and Cannabis Phenotypes

3.2.1 Influences on Individual Phenotypes—Based on the twin-pair correlations (Table 2), a trivariate model with A, C, and E factors was selected as the base model, with variance components and thresholds estimated separately for EAs and AAs. Proportions of variance for the final model are presented in Table 3 and Figures 1a and 1b. Supplemental Table S2² contains models tested and fit indices, and Supplemental Figures S1a and S1b² contain the standardized parameter estimates from the final models. In the trivariate model, there was no evidence for significant genetic influences on childhood maltreatment in either EAs or AAs; remaining estimates of C and E could be equated across racial/ethnic groups (C=73% and E=27% of variance). A influenced cannabis initiation (60%) and problems (46%) to the same degree in EAs and AAs. In EAs, the remainder of the variance was due to C (initiation=18%; problems=23%) and E (initiation=22%, problems=31%), whereas in AAs, only E contributed to non-genetic sources of variance (initiation=40%; problems=54%).

3.2.2. Covariation Between Maltreatment, Cannabis Initiation and Problems—Among EAs, C and E contributed to the association of maltreatment with cannabis initiation ($rC=0.70$; $rE=0.25$; see Table 4 and Figures 1a and 1b), but all covariation between maltreatment and cannabis problems was attributable to C ($rC=0.74$). The association between cannabis initiation and problems was attributable to shared environmental factors that were entirely overlapping with maltreatment ($rC=0.52$) and to genetic overlap ($rA=0.84$, equated across EAs and AAs) that was distinct from maltreatment, which was not itself heritable. In contrast, E explained all of the covariation of maltreatment with the two stages of cannabis involvement among the AAs ($rE=0.36$ – 0.39). After accounting for covariance with maltreatment, there was evidence for residual E that related cannabis initiation to problem use ($rE=-0.55$) as well as the cannabis-specific genetic association that was equated to the EAs ($rA=0.84$).

4. Discussion

Prior studies have shown that exposure to childhood maltreatment is strongly correlated with and predictive of cannabis initiation and progression to problem use. Building upon this prior research, we employed a genetically informative design to estimate the extent to which genetic and environmental factors contribute to this correlation in African-American (AA) and European-American (EA) young women. We extended our understanding of this association in four ways. First, at the phenotypic level, we found childhood maltreatment was associated with greater likelihood of cannabis initiation before age 15 among both EAs and AAs but later initiation only among EAs, and with approximately twofold greater risk of cannabis problems in both racial/ethnic groups. Second, in examining familial influences, we found that whereas childhood maltreatment could be attributed in large part to shared environment (C) in both EAs and AAs, genetic influences (A) accounted for a much greater proportion of variance in cannabis outcomes. Thus, there was no evidence for genetic sources contributing to overlap between maltreatment and cannabis involvement (i.e., gene – environment correlation). Third, we found evidence of racial/ethnic differences in the type of

environmental factors that contribute to both initiation and problem use of cannabis (i.e., C was only important in EAs). Fourth, we found evidence for racial/ethnic differences in the sources of environmental covariation between risk for maltreatment and cannabis outcomes (i.e., rC and rE for EAs, only rE for AAs).

4.1 Phenotypic Associations

Our finding that childhood maltreatment is associated with elevated risk for early initiation of cannabis is consistent with previous research (e.g., Bensley et al., 1999; Harrison et al., 1997; Nelson et al., 2006; Sartor et al., 2015; Werner et al., 2016), including the little research assessing AAs specifically - which, to our knowledge, is limited to prior work by our group that included participants in the current study (Sartor et al., 2015; Werner et al., 2016). The specificity of risk conferred by childhood maltreatment to early initiation in AAs but to more global risk for cannabis initiation at any age in EAs is consistent with our prior studies examining sexual abuse (Sartor et al., 2015; Werner et al., 2016), but contrasts with Werner et al.'s (2016) finding that physical abuse, when examined independently of sexual abuse, was associated with initiation only among EAs.

Our examination of cannabis-related problems indicated that the association with childhood maltreatment is significant and equally strong for EA and AA women. This is consistent with previous research utilizing samples of predominantly European ancestry (e.g., Afifi et al., 2012; Duncan et al., 2008; Kendler et al., 2000; Rogosch et al., 2010) but divergent from the limited work contrasting AAs and EAs, which suggests that the association might be weaker for AAs (Werner et al., 2016) or, in the case of sexual abuse specifically, only significant for onset of problem use beyond age 21 (Sartor et al., 2015). Importantly, unlike these previous studies, the present results are unadjusted for potential confounding factors, e.g., family-level risk factors and co-occurring psychopathology, which may have a greater impact on associations of childhood maltreatment with cannabis outcomes in AAs than EAs.

4.2 Genetic and environmental sources of associations

Individual differences in exposure to childhood maltreatment were attributable to shared (73%) and individual-specific environment, in contrast to some prior studies suggesting the role of genetic influences on exposure to lifetime assaultive trauma in adults (Sartor et al., 2012), but consistent with the idea that a broad index of exposure would be more representative of early familial milieu. While there were no racial/ethnic differences in the contributors to variance in maltreatment, we found evidence of racial/ethnic differences in the factors contributing to cannabis involvement and consequently, in the covariation between maltreatment and cannabis initiation and problems. Specifically, among AAs, we did not find evidence for shared environmental influences on either cannabis initiation or problems, and all covariation with maltreatment was through individual-specific environmental factors, with residual association between stages of cannabis involvement attributable to genetic factors not shared with maltreatment and individual-specific sources partially shared with maltreatment. In contrast, consistent with the prior twin literature on European populations (Verweij et al., 2010), we observed modest shared environmental influences on both cannabis initiation (18%) and problem use (23%) in EAs. Importantly, all of these shared environmental influences overlapped with those related to childhood

maltreatment. The residual covariance between cannabis initiation and progression to problems was due to genetic factors that were unrelated to maltreatment. Taken together, these results indicate that the distinctions by race/ethnicity are not in the relative contribution of genes (i.e., heritability) and environment, but rather in the *type* of environmental influences that contribute to stages of cannabis involvement. While shared environment plays an important role in the association between maltreatment and cannabis outcomes among EAs, only individual-specific factors related maltreatment to cannabis initiation and problems in AAs.

In the absence of prior twin studies of cannabis outcomes in AAs, we can only speculate on the potential sources of this distinction. Considering that the shared environmental influences on cannabis outcomes in EAs overlapped entirely with those that influence maltreatment, it may be that family-level environmental factors that frequently co-occur with maltreatment, such as parental conflict or separation (Bidarra et al., 2016; Hindley et al., 2006) and parental alcohol problems (McLaughlin et al., 2000; Shin et al., 2009), are more closely linked to early and problem use of cannabis in EA than AA girls. This is consistent with findings from our earlier study of sexual abuse and cannabis outcomes, in which the relative contribution of sibling early initiation of cannabis was much greater in EAs than AAs (Sartor et al., 2015).

4.3 Limitations

Our findings should be interpreted in light of some limitations. First, the smaller sample of AA twins may have limited our power to detect racial/ethnic differences between EAs and AAs, and to examine specific factors that might contribute to racial/ethnic differences (e.g., socioeconomic status). Second, we noted a negative individual-specific environmental correlation (Table 4, $rE = -0.55$) between cannabis initiation and problems in AAs, which might plausibly be related to compensatory environmental mechanisms that decelerate progression from early to problem use in AAs, after accounting for common genetic risk. Given the limited exploration of cannabis use in AAs, it is difficult to speculate what these environmental factors may be. Alternatively, the modest association between early cannabis use and cannabis problems in AAs relative to EAs may have resulted in sparseness of data in cross-twin cross-trait correlations, yielding statistical compensation for these estimates. Third, although the majority of the sample was ascertained in the peak years for cannabis initiation and early-to-peak years for problem onset, not all participants had passed through the age of risk for the cannabis outcomes. Fourth, although we attempted to minimize potential retrospective reporting bias by adjusting for participant age in the analyses, retrospective assessments are inherently vulnerable to reporting bias. Fifth, findings may not generalize to other populations, other definitions of maltreatment, or more severe cannabis outcomes (e.g., cannabis use disorder). Relatedly, it is possible that certain forms of maltreatment (e.g., physical vs. sexual abuse) are more closely related to cannabis involvement in EAs vs. AAs (as indicated by Werner et al., 2016). Sixth, our analyses do not address which specific aspects of initiation or problem use differ in their relationship with maltreatment. Our broad definitions of initiation and problems may also have limited a more specific understanding of the effects of maltreatment. Seventh, we did not test whether the heritability of cannabis initiation and problems, and their covariance, was modified by the

presence or absence of maltreatment (i.e., a putative gene $\times$ environment interaction, with maltreatment representing the environmental modifier). Larger sample sizes in the AAs would be required to carefully address this hypothesis. Finally, we collapsed across multiple waves of data. Even though we examine associations with early vs. later initiation, future studies in other samples with systematic longitudinal data, might be particularly informative.

5.0 Conclusions and Future Directions

Our findings have important implications for prevention efforts as well as future investigations of the shared etiology of maltreatment and cannabis involvement. Identifying the sources of liability both to maltreatment and cannabis involvement and the variability across race/ethnicity in their associations will ultimately lead to improvements in interventions with young women who have experienced childhood maltreatment. It will also help mitigate cannabis-associated risks effectively in women of different racial/ethnic backgrounds.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Maltreatment is associated with cannabis initiation before age 15
- Variance in maltreatment is largely attributable to shared environmental factors
- Cannabis initiation and problems are genetically influenced
- Environmental influences on cannabis initiation/problems differ by race/ethnicity
- Sources of covariation between maltreatment and cannabis differ by race/ethnicity

Figure 1.

A=additive genetics, C=shared environment, E=individual-specific environment (including error). Single-headed arrows indicate proportions of variance for a measure; curved double-headed arrows indicate correlations between measures. All paths significant at $p < 0.05$.

a: Final trivariate genetic model examining the proportions of variance for and the genetic and environmental correlations between childhood maltreatment, timing of cannabis initiation, and cannabis problems in a sample of European-American young adult female twins.

b: Final trivariate genetic model examining the proportions of variance for and the genetic and environmental correlations between childhood maltreatment, timing of cannabis initiation, and cannabis problems in a sample of African-American young adult female twins.

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Table 1

Prevalence childhood maltreatment and cannabis use and problems in 3786 European-American and African-American young adult women in the Missouri Adolescent Female Twin Study.

Measure	All (n=3786)	EA (n=3233)	AA (n=553)
Childhood maltreatment ^{* a}	17.3%	15.6%	27.3%
Cannabis initiation			
Before age 15 ^b	8.0%	7.8%	9.0%
Age 15 or older ^c	45.2%	44.7%	48.5%
Never ^{* d}	46.8%	47.5%	42.5%
Any DSM-IV cannabis symptom (of users) ^{* e}	26.3%	24.5%	35.5%

* indicates EA and AA differ at $p < 0.05$

^a ($\chi^2(1) = 44.25, p < 0.001$)

^b ($\chi^2(1) = 0.53, p = 0.47$)

^c ($\chi^2(1) = 2.99, p = 0.08$)

^d ($\chi^2(1) = 4.46, p = 0.03$)

^e N=417 of 1699 EA users; N=113 of 318 AA users, ($\chi^2(1) = 16.70, p < 0.001$)

Twin pair correlations for measures of childhood maltreatment and cannabis outcomes among young adult women in the Missouri Adolescent Female Twin Study

Table 2

	All		EA only		AA only	
	rMZ	rDZ	rMZ	rDZ	rMZ	rDZ
Childhood Maltreatment	0.79 [*] [0.72, 0.85]	0.69 [*] [0.60, 0.78]	0.78 [*] [0.71, 0.86]	0.70 [*] [0.59, 0.80]	0.80 [*] [0.64, 0.96]	0.61 [*] [0.40, 0.82]
Timing of cannabis initiation	0.77 [*] [0.72, 0.81]	0.47 [*] [0.39, 0.55]	0.78 [*] [0.73, 0.82]	0.53 [*] [0.45, 0.61]	0.68 [*] [0.53, 0.82]	0.23 [*] [0.02, 0.44]
Any DSM-IV cannabis symptom (among users)	0.61 [*] [0.49, 0.73]	0.35 [*] [0.17, 0.53]	0.63 [*] [0.50, 0.76]	0.39 [*] [0.20, 0.59]	0.44 [*] [0.07, 0.81]	0.21 [*] [-0.25, 0.67]

^{*} indicates significant at $p < 0.05$.

rMZ=monozygotic twin-pair correlation, rDZ=dizygotic twin-pair correlation, EA=European-American, AA=African-American

Table 3

Proportions of variance from the best-fitting trivariate model assessing genetic and environmental contributions to variability in childhood maltreatment, timing of cannabis initiation, and cannabis use-disorder symptom development among young adult women in the Missouri Adolescent Female Twin Study ^a

EAs	Additive Genetic	Shared Environmental	Individual-Specific Environmental
Childhood maltreatment	---	0.73 [*] [0.67, 0.78]	0.27 [*] [0.22, 0.33]
Timing of cannabis initiation (before age 15, age 15+, never)	0.60 [*] [0.48, 0.71]	0.18 [*] [0.07, 0.30]	0.22 [*] [0.18, 0.26]
Any cannabis use disorder symptom (of cannabis users)	0.46 [*] [0.24, 0.62]	0.23 [*] [0.08, 0.43]	0.31 [0.22, 0.42]
AAs	Additive Genetic	Shared Environmental	Individual-Specific Environmental
Childhood maltreatment	---	0.73 [*] [0.67, 0.78]	0.27 [*] [0.22, 0.33]
Timing of cannabis initiation (before age 15, age 15+, never)	0.60 [*] [0.48, 0.71]	---	0.40 [*] [0.28, 0.52]
Any cannabis use disorder symptom (of cannabis users)	0.46 [*] [0.24, 0.62]	---	0.54 [*] [0.38, 0.76]

^{*} indicates significant at $p < 0.05$

EAs=European-Americans, AAs=African-Americans.

^aFull model $-2 \log\text{-likelihood} = 11351.399$, 60 estimated parameters; final model $\chi^2(19) = 19.572$, $p = 0.42$

Table 4

Genetic and environmental covariation between childhood maltreatment, timing of cannabis initiation, and cannabis use-disorder symptom development among young adult women in the Missouri Adolescent Female Twin Study.

Covariation between:	EAs	AAs
Childhood maltreatment and Timing of cannabis initiation		
rA	---	---
rC	0.70 * [0.50, 1.00]	---
rE	0.25 * [0.07, 0.41]	0.36 * [0.08, 0.62]
Childhood maltreatment and Any cannabis use disorder symptom		
rA	---	---
rC	0.74 * [0.48, 1.00]	---
rE	---	0.39 * [0.05, 0.70]
Timing of cannabis initiation and Any cannabis use disorder symptom		
rA	0.84 * [0.64, 1.00]	0.84 * [0.64, 1.00]
rC	0.52 * [0.30, 1.00]	---
rE	---	-0.55 * [-0.85, -0.07]

* indicates significant at $p < 0.05$.

rA=additive genetic correlation, rC=shared environmental correlation, rE=individual-specific environmental correlation. EAs=European-Americans, AAs=African-Americans.