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Intergenerational Continuity in Cannabis Use: The Role of Parent's Early Onset and Lifetime Disorder on Child's Early Onset

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Abstract

Purpose—Children's early onset of cannabis use was examined as a function of their parent's early onset of cannabis and subsequent incidence of a lifetime cannabis abuse or dependence disorder.

Methods—Prospective, longitudinal data from the Rochester Youth Development Study (RYDS) and the Rochester Intergenerational Study (RIGS) for 442 parent-child dyads (274 father-child, 168 mother-child) were utilized. The children were evenly split by sex. Logistic regression models and a path analysis were estimated to assess the effect of parent's cannabis use on child's onset of cannabis by age 15.

Results—Fathers who began using cannabis by age 15 were more likely to meet the criteria for a lifetime cannabis disorder (O.R. = 5.66, 95% CI = 1.89, 16.90). The offspring of fathers who met the criteria for a disorder had higher odds of early initiation of cannabis use (O.R. = 9.70, 95% CI = 3.00, 31.34). Early onset cannabis use by father was indirectly associated with their child's onset of cannabis use via father's lifetime cannabis disorder. No significant effects for mothers were observed, although analyseswere limited due to the low rate of mothers who met the criteria for a lifetime cannabis disorder.

Conclusions—This study provides evidence of intergenerational continuity in cannabis use among fathers and their children and confirms the need to consider timing of use and intervening mechanisms in the study of continuity in cannabis use across generations.

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No conflicts of interest exist for this work.

Implications and Contribution: Prospective, longitudinal, and intergenerational data were used to examine intergenerational continuity in cannabis use. Intergenerational continuity was observed for fathers. Early onset cannabis use was indirectly associated with a child's early onset of cannabis use via the presence of a lifetime cannabis disorder.

Keywords

Cannabis abuse and dependence; cannabis disorder; early onset; intergenerational continuity

Echoing a political climate calling for further legalization of cannabis, the use of cannabis (for non-medical reasons) is on the rise in the United States.[1] Currently, over 30% of young adults report recent use of cannabis[2] and 40% of high school students report a lifetime prevalence of cannabis use.[3] Moreover, nearly 16% of eighth graders indicate they previously tried cannabis.[1] Despite evidence that cannabis use is becoming increasingly normative, its use by young people can change developmental trajectories towards maladaptation[4] with evidence of short-term (e.g., impulsivity, morbidity, risky sexual behavior, and delinquency) and long-term (e.g., academic underachievement, employment instability, mental illness, psychosocial adjustment, brain development, family functioning) consequences.[4–6]

In this study we focus on early onset of cannabis use, specifically, use that begins by age 15. The timing of onset of cannabis use may be particularly salient to the harmful consequences of use. For example, early onset of cannabis use is linked to subsequent drug abuse and/or dependence.[7–10] In one study,[7] individuals who used cannabis during adolescence were two to four times more likely to display symptoms of cannabis dependence[11] compared to individuals who delayed use until after age 18.

Given the salience of early onset cannabis use, a more thorough understanding of the predictors of the early onset of cannabis is needed. Adopting a life course perspective that stresses the importance of interdependent lives across generations[12], we focus on a generally understudied influence – intergenerational (IG) continuity in cannabis use. This type of continuity is defined by similarity in behavior between parent and child during the same developmental period (e.g., adolescence).[13] We examine the role of a parent's use of cannabis during his/her own adolescence, as well as his/her subsequent abuse or dependence, on his/her child's early onset of cannabis use. Surprisingly little published work is available to quantify the specific direct and indirect effects of a parent's cannabis use during adolescence on offspring's cannabis use, particularly when limited to papers meeting the methodological rigor to study IG hypotheses.[14] One exception is Knight and colleagues[15] who report a direct relationship between frequency of parental use of cannabis during both adolescence and emerging adulthood and frequency of child cannabis use during these same time periods. Kerr, Tiberio, and Capaldi[16] also examined the relationship between frequency of parent cannabis use during adolescence (retrospectively reported for mothers but prospectively reported for fathers) and offspring cannabis use onset during adolescence and found an indirect IG effect via two social context variables – peer marijuana use and peer delinquency. Most recently, Bailey and colleagues[17] reported that frequency of parent's current use of cannabis predicted child's use of cannabis, but frequency of parent's cannabis use during late adolescence and early adulthood (historical use) did not. These three studies provide important knowledge on the topic of IG continuity in cannabis use, but more work is clearly needed.

In this study, we add to the scant literature on this topic by using a prospective, longitudinal, and multigenerational data set. We first examine whether the early onset of cannabis use by an adolescent is a function of his/her parent's early onset of cannabis use (i.e., a direct assessment of IG continuity). We then account for a particularly pernicious consequence of early onset cannabis use – a parent's subsequent cannabis abuse or dependence disorder (based on DSM-IV diagnostic criteria[11]) – and examine whether the presence of a cannabis disorder serves as an intermediate variable between parental early onset of cannabis use and child onset of cannabis use. Specifically, we test four hypotheses:

1. A parent who began using cannabis by age 15 is more likely to meet the criteria for a lifetime cannabis disorder by adulthood.
2. A child is more likely to initiate cannabis use by age 15 if his/her parent initiated cannabis use by age 15.
3. A child is more likely to initiate cannabis use by age 15 if his/her parent met the criteria for a lifetime cannabis disorder.
4. The effect of early onset cannabis use by a parent on his/her child's early onset of cannabis use is mediated by the parent's lifetime incidence of a cannabis disorder.

In any study of IG continuity, it is important to acknowledge the sex of the parent because life events of males and females differentially impact the next generation[18], and parental roles tend to vary for mothers and fathers.[19] Evidence suggests that the antisocial behavior of fathers and mothers differentially affects children with greater continuity in antisocial between fathers and their children compared to mothers and their children.[18,20–22] Therefore, we depart from prior work on IG continuity in cannabis use[15,16,17] and evaluate our hypotheses separately for mothers and fathers in order to determine if sex of the parent conditions IG continuity in cannabis use.

Methods

Sample

The data for this study come from two longitudinal, companion studies. The original study, the Rochester Youth Development Study (RYDS), began in 1988 and the intergenerational extension, the Rochester Intergenerational Study (RIGS), began in 1999. Detailed information about the designs of these studies is presented elsewhere;[23] only a brief summary is provided here.

The original RYDS sample of 1,000 adolescents (referred to as G2; their primary caregiver is referred to as G1) is representative of the 7th and 8th grade public school population of Rochester, NY in 1988. Youth at high risk for antisocial behavior were overrepresented by oversampling males and residence in high-crime areas of the city. RYDS participants completed regular interviews in school or home every six months from 1988-1992 (Phase 1), annually from 1994-1996 (Phase 2), and biannually from 2003 to 2006 (Phase 3). In general, sample retention was good and analyses reveal attrition did not bias the sample.[24]

Beginning in 1999, RIGS selected G2's oldest biological child, referred to as G3, and added new firstborns to the G3 sample in each subsequent year. Both G2 and G3's other primary caregiver completed annual interviews since the inception of RIGS (continuing until G3 turns/turned 18) and G3 completed annual interviews once he/she turned eight. To date, there are prospective, longitudinal data on 529 parent-child dyads. The present analysis utilizes data from 442 parent-child dyads (274 father-child, 168 mother-child); this includes all dyads in which G3 was born prior to 2001 (allowing them to have been at least 15 years old at the last available wave of data collection - 2016). The children were evenly split by sex. Based on G2's report, when G3 was 15 years old, 96% of mothers and 67% of fathers either lived with their child or their child regularly spent the night with them. All data collection procedures were approved by the University at Albany's Institutional Review Board.

Measures

Early onset cannabis use by G2 was measured during Phase 1 of RYDS and by G3 during RIGS. At the first interview for G2, and during the Age 8 interview for G3, respondents reported if they ever used cannabis, and if they had, at what age they started using. In subsequent interviews, respondents reported if they used cannabis since the last interview. Using this information, we created a binary indicator of early onset cannabis use, where the respondent is coded 1 if he/she initiated use of cannabis at or before their age 15 interview, and 0 if they initiated use after the age 15 interview (or not at all). We selected cannabis use onset at or before age 15 because this is in line with previous research identifying problematic outcomes as a result of onset at this age[25] and standards set by the Substance Abuse and Mental Services Association for early experimentation and uptake.[26]

Lifetime cannabis abuse and dependence was measured when G2 was in their late twenties or early thirties (Phase 3) using the Computerized Diagnostic Interview Schedule Version IV (CDIS-IV[27]) during the participant's annual interview. The CDIS-IV is based on the Diagnostic and Statistical Manual, Edition 4, DSM-IV[11] criteria for lifetime substance use, abuse, and dependence. Participants who met the criteria for either lifetime cannabis abuse or dependence (referred to in the results as a disorder) were assigned a 1; those who did not meet the criteria for either abuse or dependence were assigned a 0.

We include a set of control variables that are proposed to be causally prior to G2 early onset of cannabis use and their subsequent cannabis disorder status: *G2 sex*; *G2 race/ethnicity; G2 age* at the start of RYDS; *G1's years of education*; *structure of G2's family* at the start of RYDS (a binary variable indicating respondents who lived with both biological parents); *low income status of G2's family* at the start of RYDS (a binary indicator of poverty level income, receipt of welfare, or unemployment of the primary wage earner). We also controlled for two variables describing the neighborhood where G2 lived at the start of RYDS: 1) the *arrest rate per 100 people* based on Rochester Police Records; 2) *the proportion of residents in the census tract living in poverty* (from U.S. Census Records). Last, we controlled for three variables that were not causally prior to G2 cannabis use but are important when modeling G3 cannabis use: *G3 sex*, *G2 age at G3's birth*, and *the average contact of G2 with G3* from age 10 to 15. The contact variable was created from a two-part

question asked of both G2 and G3. First, the respondent was asked if they lived with the other at the time of the interview. If they did not live with the other, then they were asked how frequently they spent time together. For G2, this frequency measure was captured on a 5-point scale (0=never, 1=once or twice per year, 2=less than once per month, 3=at least once a month, and 4=at least once a week). For G3, this frequency measure was captured on a 4-point scale (0=never, 1=almost never, 2=sometimes, 3=often). We modified this frequency measure by assigning those who lived together a score 1 point higher than the maximum (i.e., 5 for the G2 measure and 4 for the G3 measure). We then created a standardized score at each age. The final contact score was formed by averaging the scores across reporters and years.

Descriptive statistics are presented in Table 1 (for demographic variables) and Table 2 (for cannabis variables). Also in Table 1, we present information to ascertain how the sample used for the current analysis differs from the full RYDS and RIGS samples in terms of the demographic variables.

Analysis

All analyses were conducted in Mplus, Version 7.4.[28] Hypothesis 1 through 3 were assessed using a logistic regression model. Hypothesis 4 was estimated as a path analysis. A weighted least squares means and variances adjusted (WLSMV) probit estimator was used to estimate the mediation model.[29] In order to properly account for missing data, we created 10 multiply imputed datasets in Mplus. All analyses were performed on each of the imputed datasets and the results were pooled using the procedures outlined by Rubin.[30]

Results

Findings for G2 Males

We first considered the relationship between G2 early use of cannabis and subsequent disorder for fathers. As illustrated in Table 2, 25.6% of G2 males used cannabis by age 15, and 6.8% met the criteria for a disorder. As shown in Table 3, a higher proportion of those who used cannabis by age 15 met the criteria for a cannabis disorder in adulthood (17.2%) compared to G2 males who did not use cannabis by age 15 (3.2%). Supporting Hypothesis 1, the association between male G2 early onset cannabis use and male G2 lifetime cannabis disorder was significant, adjusting for G2 background variables; the odds of a disorder were over five times higher among men who initiated use of cannabis by age 15 (O.R. = 5.66, 95% CI = 1.89, 16.90).

Regarding IG continuity in early onset use, early onset cannabis use was more prevalent among G3 participants whose father began using cannabis by age 15 (20.6%) compared to G3 participants whose father delayed cannabis use until after age 15 or not at all (11.1%). However, adjusting for all background variables, a father's early onset cannabis use was not directly associated with G3 early onset cannabis use (O.R. = 1.83, 95% CI = .84, 4.03). On the other hand, early onset cannabis use was more prevalent among G3 participants whose father met the criteria for a lifetime cannabis disorder (50%) compared to G3 participants whose father did not meet criteria for a disorder (10.4%). A father's cannabis disorder was

directly associated with G3 cannabis onset (O.R. = 9.70, 95% CI = 3.00, 31.34) after adjusting for all background variables.

Finally, we tested the path analysis to examine the extent to which IG continuity in early onset use was indirect via a father's lifetime cannabis disorder. The results of our path analysis reveal that male G2 early onset cannabis use was indirectly associated with G3 early onset cannabis use via the father's lifetime cannabis disorder (WLSMV probit estimate for indirect path [product of coefficients method] = .64, 95% CI = .15, 1.13). In this analysis a 95% CI that does not contain 0 is statistically significant ($p<.05$) and is indicative of a significant indirect IG pathway. A father's early onset of cannabis use increased the likelihood that he experienced a cannabis disorder, which subsequently increased the likelihood that G3 initiated use of cannabis at an early age. Therefore, while Hypothesis 2 was not supported, Hypotheses 1, 3 and 4 were supported for G2 males.

Findings for G2 Females

Among G2 females, 33.3% used cannabis by age 15, and 1.9% met the criteria for a lifetime cannabis disorder. Given the small number of G2 females who met the criteria for a disorder (3 females), only Hypothesis 2 was tested. We found no evidence of IG continuity for mothers. Early onset cannabis use was no more prevalent among G3 participants whose mother began using cannabis by age 15 (14.6%) compared to G3 participants whose mother delayed cannabis use to after age 15 or not at all (15.0%). Adjusting for the background covariates, G2 early onset of cannabis was not significantly associated with G3 early onset of cannabis (O.R. = .77, 95% CI = .25, 2.33). In sum, Hypotheses 1, 3 and 4 were not tested given the very small number of G2 mothers who met the criteria for a cannabis disorder, and we found no evidence to support Hypothesis 2.

Discussion

Our research adds to the scientific and public health body of knowledge regarding the “human costs”[31] of drug use as it further demonstrates that the early onset of cannabis use by boys is associated with an increased risk of experiencing a cannabis disorder. Furthermore, we demonstrate that the human costs of cannabis use are not limited to one's own developmental trajectory; a father's history of cannabis use and abuse is associated with his child's eventual early uptake of cannabis use. We find no evidence of IG continuity in early onset use of cannabis for mothers and their children, and we were prevented from considering mothers' more serious cannabis use because so few G2 females in the sample met the criteria for a disorder.

Our study is one of few that considers IG continuity in cannabis use, and, in doing so, we make valuable contributions to the existing literature. It is important however to recognize the limitations of our study. First, the sample size used in this research is relatively small (N=442), and, when separated by G2 sex, the analytic samples become even smaller. The small sample sizes also precluded our ability to consider differential effects as a function of G3 sex and presented problems when considering the role of a G2 cannabis disorder in cannabis continuity among G2 mothers. While our work is informative with respect to IG continuity in cannabis use for fathers, we were unable to fully study IG continuity in

cannabis use among mothers. At this point, we refrain from saying that there is no evidence of IG continuity in cannabis onset among mothers; this would be unwise given the initial sampling strategy of RYDS (i.e., only 27% were female), and the low prevalence of cannabis disorder among this sample of women. However, the lack of a relationship between early onset of cannabis use and a subsequent disorder is interesting in and of itself. It could be a result of antisocial behavior being more serious and exhibiting more stability across the life course among males compared to females[32] or that early onset is more important in the etiology of a cannabis disorder for males than females.[33] It is also important to recognize that a mother's use of other substances (e.g., tobacco and alcohol use) plays an important role in the onset of substance use among her children[34], and it may be that more visible substance use (e.g., tobacco or alcohol) is more relevant to the study of continuity in substance use among mother-child dyads as illegal drug use may be hidden from children. Nevertheless, additional research is needed to speak to these possibilities.

Another limitation arises from the IG nature of the study. Currently, G3s born to parents who delayed parenthood into their late 20s or older are not represented in this research because their firstborns have not yet aged into adolescence (at least not sufficiently so to be included in the analyses presented here). It will be important to revisit these analyses when the full sample of G3s has passed through adolescence.

Last, it is important to note that the Rochester studies represent families who lived in Rochester, NY in the mid-1980s, and the extent to which these findings generalize to other samples is unknown. However, we note that almost all other prospective, longitudinal data sources are limited in the same way with respect to a specific geographic locale. Nonetheless, this is the first study to demonstrate IG continuity among a predominantly minority sample (i.e., 90% of the families were non-White in RIGS compared to 22% in the Oregon Youth Study,[16] 27% in the National Youth Survey Family Study,[15] and 59% in the Seattle Social Development Study Intergenerational Project[17]), suggesting the importance of IG continuity in an ethnically diverse sample.

Notwithstanding the lack of IG continuity in cannabis onset for females, this research has significant implications for policy as it draws attention to the importance of mitigating consequences of early onset of cannabis use in order to limit human costs (i.e., escalation of use to a disorder and the ill consequences associated with abuse) and protect the next generation. Attempts to prevent or delay the onset of cannabis use are now even more imperative to the future well-being of society given the recent legalization of cannabis in some locales, the likely expansion of legalization across the U.S., and the overall increased visibility of cannabis to youth. This research points to a father's use of cannabis, in terms of early onset and a subsequent cannabis disorder, as a factor that puts his child at increased risk for early onset of cannabis use. Therefore, preventing the early onset and escalation of cannabis use and/or mitigating the ill effects of cannabis use will have important implications for the next generation of children.

There are a host of effective universal prevention programs (targeting the general population) aimed at reducing the likelihood of drug use onset, including cannabis, in late childhood and early adolescence such as Positive Action[35] and Life Skills Training.[36]

Moreover, effective selective intervention programs like Project Towards No Drug Abuse[37] can target adolescents who experienced an early onset of cannabis use to prevent subsequent problematic use. In addition, efforts to help fathers who suffered or are suffering from a cannabis disorder recognize that their children are at an increased risk for early onset of cannabis use may also prove fruitful, particularly if fathers can be given the tools to help prevent the use of cannabis by their children. Indeed, there are effective interventions designed to assist parents in this way such as The Family Check-Up[38] and The Strengthening Families Program.[39]

The study presented here is largely descriptive, and sets the stage for important future work. We did not attempt to isolate the specific causal mechanism(s) accounting for the transmission of risk across generations (e.g., heritability, imitation or modeling, transmission of risk factors that promote drug use, etc.), nor did we examine potential moderators of IG continuity other than parental sex. The next step for research is to unpack the mechanisms by which parents' experiences with cannabis influence their children's experiences with cannabis.

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Table 1

Descriptive statistics for background covariates

	Full RYDS Sample			All Known Parents			Full RIGS Sample			RIGS Subsample		
Variable	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**
G2 Male	1000	0.73		727	0.70		529	0.65		442	0.62	
G2 Black	1000	0.68		727	0.69		529	0.72		442	0.74	
G2 Hispanic	1000	0.17		727	0.17		529	0.15		442	0.16	
G2 Age at Start of Study	1000	13.57	0.7 7	727	13.57	0.7 6	529	13.55	0.7 6	442	13.61	0.7 6
Arrest rate (per 100) in G2's Neighborhood	1000	4.19	2.07	727	4.31	2.05	529	4.35	2.04	442	4.46	2.03
Proportion of G2's Neighborhood in Poverty	1000	0.33	0.14	727	0.34	0.14	529	0.34	0.14	442	0.35	0.13
G1 Years of Education	992	11.35	2.15	724	11.26	2.16	527	11.20	2.07	440	11.04	2.04
G1 Low Income Status	901	0.51		678	0.54		499	0.56		415	0.58	
G2 Lived with both Biological Parents	958	0.24		712	0.22		518	0.24		432	0.21	
G2 Age at First Birth	703	21.89	4.9 4	703	21.89	4.9 4	529	21.72	4.7 1	442	20.06	2.8 8
G2 Early Onset	978	0.24		727	0.26		529	0.27		442	0.29	
G2 Cannabis Disorder	678	0.06		574	0.06		490	0.05		411	0.05	

Notes: Full RYDS Sample refers all G2 participants in the RYDS study; All Known Parents refers to all G2 participants in the RYDS study who are known to have had a biological child; Full RIGS Sample refers to all G2s who have taken part in RIGS; RIGS Subsample refers to all G2s in the RIGS study who have a biological child (G3) born prior to 2001.

Table 2

Descriptive statistics for cannabis use variables

	G2 Early Onset			G2 Disorder			G3 Early Onset		
	No	Yes	Missing	No	Yes	Missing	No	Yes	Missing
G2 Males									
N	2 04	70	0	2 32	17	25	2 23	35	16
Percent among observed cases	74.5%	25.6%		93.2%	6.8%		86.4%	13.6%	
G2 Females									
N	112	56	0	159	3	6	138	24	6
Percent among observed cases	66.7%	33.3%		98.2%	1.9%		85.2%	14.8%	

Table 3

Cross tabulations of cannabis use variables

G2 Males								
Panel A	**G2 Disorder**		Panel B	**G3 Early Onset**		Panel C	**G3 Early Onset**	
G2 Early	No	Yes	**G2 Early**	No	Yes	**G2 Disorder**	No	Yes
No	179 (96.8%)	6 (3.2%)	No	169 (89.0%)	21 (11.1%)	No	198 (89.6%)	23 (10.4%)
Yes	53 (82.8%)	11 (17.2%)	Yes	54 (79.4%)	14 (20.6%)	Yes	8 (50.0%)	8 (50.0%)
G2 Females								
Panel D	**G2 Disorder**		Panel E	**G3 Early Onset**		Panel F	**G3 Early Onset**	
G2 Early	No	Yes	**G2 Early**	No	Yes	**G2 Disorder**	No	Yes
No	107 (99.1%)	1 (.9%)	No	91 (85.1%)	16 (15.0%)	No	132 (84.6%)	24 (15.4%)
Yes	52 (96.3%)	2 (3.7%)	Yes	47 (85.5%)	8 (14.6%)	Yes	3 (100%)	0 (0%)

Note: Cells contain the N, with the percentage of the row in parentheses.