

PARENTAL MONITORING AND CANNABIS USE: EPIDEMIOLOGICAL
EVIDENCE FROM TWO PROSPECTIVE COHORT STUDIES

By

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ABSTRACT

PARENTAL MONITORING AND DRUG USE: EPIDEMIOLOGICAL EVIDENCE FROM TWO PROSPECTIVE COHORT STUDIES

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Background: Cannabis is one of the most commonly used psychoactive drugs in the world. The adept level of parental monitoring during childhood and adolescence is one potential intervention target for preventing cannabis use or delaying its onset.

Aims: The overall aim of this dissertation is to estimate the potential short- and long-term impact of parental monitoring on cannabis. The first study (Chapter Three) aims to estimate, prospectively, the influence of parental monitoring assessed at age 11 on cannabis initiation before age 18 years. Next, (Chapter Four) the goal is to estimate the suspected influence of level of parental monitoring assessed at age 11 on level of drug use at age 17 years, while simultaneously testing paths through levels of drug use and affiliation with drug using peers at age 11 years. The final study (Chapter Five) aims to test the prospective association that might link level of parental monitoring with subsequent recently active cannabis smoking, and to examine a potential meditational influence of level of deviant peer affiliation.

Methods: Data for the first two studies are from a longitudinal study of a 1983-1985 birth cohort from southeast Michigan (n=823). Data for the third study come from a prospective cohort of students from an urban public school system in the mid-Atlantic United States entering primary school in 1985 and 1986 (n=2,311). For all three studies drug use was assessed via standardized, self-reported measures and parental monitoring

was assessed via a 10-item standardized child-reported scale. In the first study, Poisson regression with robust error variances was used to estimate the suspected predictive association that links parent monitoring at age 11 and cannabis use up to age 17. The next study used a structural equation modeling (SEM) approach to estimate paths of parental monitoring influence, with levels of affiliation with drug using peers and drug use modeled as latent variables. The final study applied a generalized estimating equations (GEE) logistic regression approach to estimate the association between prior parental monitoring and subsequent recent cannabis use. SEM was used to examine the potential mediating influence of level of deviant peer affiliation.

Results: In the first study, higher parental monitoring at age 11 signaled a reduced risk of cannabis initiation from ages 11 to 17 years (adjusted estimated relative risk = 0.96; 95% confidence interval = 0.94, 0.98). Results from the second study indicated that level of parental monitoring was related to levels of affiliation with drug using peers ($p < 0.05$) and drug use at age 11 ($p < 0.05$) and predicted levels of drug use at age 17 ($p < 0.05$). In the third study, higher levels of prior parental monitoring significantly predicted lower odds of recently active cannabis use (adjusted odds ratio (AOR) = 0.96; 95% confidence interval (CI) = 0.92, 0.99). Level of deviant peer affiliation did not appreciably mediate the association between previous level of parental monitoring and subsequent cannabis use.

Conclusions: These findings from prospective research shed new light and help confirm the theory that parenting and familial characteristics might exert long-lasting influences on a child's risk of initiation and use of illegal drugs. Implications for prevention and future directions for research are discussed.

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TABLE OF CONTENTS

LIST OF TABLES.....	x
LIST OF FIGURES.....	xii
CHAPTER ONE	
STATEMENT OF THE PROBLEM AND SPECIFIC AIMS.....	1
1.1 Statement of the Problem.....	1
1.2 Specific Research Aims.....	5
CHAPTER TWO	
REVIEW OF THE LITERATURE.....	7
2.1 Epidemiology of Cannabis Use	7
2.1.1 Structure of the Review of the Literature on the Epidemiology of Cannabis Use.....	7
2.1.2 Quantity	8
2.1.2.1 Worldwide Cannabis Use.....	8
2.1.2.2 Cannabis Use in the United States.....	9
2.1.3 Location.....	10
2.1.3.1 Individual and Sociodemographic Characteristics.....	10
2.1.3.2 Variation by Place.....	11
2.1.3.3 Time Trends.....	12
2.1.4 Suspected Causes.....	13
2.1.4.1 Family History and Genetics.....	13
2.1.4.2 Parenting Practices.....	15
2.1.4.3 Affiliation with Deviant and Drug Using Peers.....	16
2.1.4.4 Intra-Individual Traits and Preexisting Psychiatric Problems.....	17
2.1.5 Mechanisms.....	18
2.1.5.1 The Gateway Hypothesis and Exposure Opportunity.....	18
2.1.5.2 Cannabis Dependence.....	20
2.1.5.3 Educational Outcomes.....	21
2.1.5.4 Other Psychiatric Consequences.....	22
2.1.6 Prevention and Control.....	23
2.2 Prior Research on Parental Monitoring.....	24
2.2.1 Definition of Parental Monitoring.....	25
2.2.2 Parental Monitoring Conceptualized within a Developmental Model for Antisocial Behavior.....	25
2.2.3 General Conceptual Models Linking Parental Monitoring with Cannabis Use.....	30
2.3 Conclusions.....	33

CHAPTER THREE – MANUSCRIPT ONE	
PARENTAL MONITORING AT AGE 11 AND SUBSEQUENT ONSET OF CANNABIS USE UP TO AGE 17: RESULTS FROM A PROSPECTIVE STUDY.....	34
Abstract.....	34
3.1 Introduction	36
3.2 Methods.....	38
3.2.1 Sample	38
3.2.2 Measures	39
3.2.2.1 Cannabis Initiation, Ages 11-17 Years.....	39
3.2.2.2 Parental Monitoring, Age 11 Years	40
3.2.2.3 Child Covariates.....	40
3.2.2.4 Maternal Covariates.....	41
3.2.3 Statistical Analysis.....	41
3.3 Results.....	42
3.3.1. Comparison of the Original Sample with the Follow-up Sub-samples at Ages 11 and 17 Years.....	42
3.3.2 Parental Monitoring at Age 11 and the Risk of Cannabis Initiation up to Age 17.....	45
3.3.3 Kaplan-Meier Survival Estimates.....	49
3.3.4 Fractional Polynomial Post-estimation Examination of the Parental Monitoring – Cannabis Initiation Association.....	51
3.4 Discussion.....	53
CHAPTER FOUR – MANUSCRIPT TWO	
LONGITUDINAL STUDY OF LEVELS OF ADOLESCENT DRUG USE: AN SEM ANALYSIS OF PARENTAL MONITORING.....	58
Abstract.....	58
4.1 Introduction	59
4.2 Methods.....	61
4.2.1 Data and Sample	61
4.2.2 Measures	62
4.2.2.1 Parental Monitoring	62
4.2.2.2 Drug Use	63
4.2.2.3 Affiliation with Drug Using Peers.....	64
4.2.2.4 Baseline Covariates.....	64
4.2.3 Statistical Analysis.....	64
4.3 Results.....	65
4.3.1. Comparison of the Original Sample with the Subset Used in the Analysis.....	65
4.3.2 Mean Levels of Parental Monitoring and Percentages of Drug Use in the Sample.....	67
4.3.3 Structural Equation Model for Parental Monitoring, Drug Use, and Peer Drug Use at Age 11 and Drug Use at Age 17.....	69
4.4 Discussion.....	73

CHAPTER FIVE – MANUSCRIPT THREE	
LONGITUDINAL PATHS FROM PARENTAL MONITORING TO CANNABIS USE IN CHILDHOOD AND ADOLESCENCE.....	77
Abstract.....	77
5.1 Introduction	79
5.2 Methods.....	81
5.2.1 Study Population and Sample.....	81
5.2.2 Main Measures	83
5.2.2.1 Parental Monitoring, PM	83
5.2.2.2 Recently Active Cannabis Smoking, CAN	83
5.2.2.3 Deviant Peer Affiliation, DPA.....	84
5.2.3 Other Covariates.....	84
5.2.4 Statistical Analysis.....	85
5.3 Results.....	88
5.3.1 Characteristics of the Sample.....	88
5.3.2 Prevalence of CAN and Mean Scores for PM and DPA, 1989- 1994.....	90
5.3.3 Results from the GEE Logistic Regression on the Hypothesized PM- CAN Link.....	92
5.3.4 Examining Clustering at the Individual and Classroom/school Levels Via the ALR.....	95
5.3.5 Testing Hypothesized Mediation through DPA.....	97
5.4 Discussion.....	105
CHAPTER SIX	
FINAL CONCLUSIONS.....	109
6.1 Summary of Findings.....	109
6.2 Limitations.....	110
6.3 Future Directions.....	112
6.4 Conclusions.....	114
APPENDICIES	
Appendix A: Technical Appendix for Chapter Three.....	116
Appendix B: Technical Appendix for Chapter Four.....	120
Appendix C: Technical Appendix for Chapter Five.....	123
Appendix D: Parental Monitoring Scale.....	129
Appendix E: Deviant Peer Affiliation Scale	130
REFERENCES	
References.....	132

LIST OF TABLES

Table 3.1	Description of the initial sample, the subset with follow-up data for ages 11 and 17, and the subset used in the regression analyses. Data come from 823 children sampled from 1983-1985 newborn discharge lists in southeast Michigan and assessed at ages 6, 11, and 17 years.....	44
Table 3.2	Parent monitoring at age 11 and initiation of cannabis use up to age 17, unadjusted models. Data come from 641 children sampled from 1983-1985 newborn discharge lists in southeast Michigan with complete information on all variables.....	47
Table 3.3	Parent monitoring at age 11 and initiation of cannabis use up to age 17, adjusted model. Data come from 641 children sampled from 1983-1985 newborn discharge lists in southeast Michigan with complete information on all variables.....	48
Table 4.1	Description of the initial sample and the subset used in the structural equation model (SEM). Data come from 823 children sampled from 1983-1985 newborn discharge lists in southeast Michigan and assessed at ages 6, 11, and 17 years.....	66
Table 4.2	Mean level of parental monitoring and percentages of drug use in the sample. Data come from 774 children sampled from 1983-1985 newborn discharge lists in southeast Michigan and contributing data to the analyses.....	68
Table 5.1	Characteristics of the sample at each assessment 1989-1994. Data come from 2,311 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.....	89
Table 5.2	Percentages of recent cannabis use and means scores of parental monitoring and deviant peer affiliation for the assessments. Data come from a cohort of 2,311 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.....	91
Table 5.3	Results from the unadjusted GEE linking prior parental monitoring with odds of recent cannabis use. Data come from 1,448 first graders enrolled in a mid-Atlantic public school system and assessed yearly from through 1994.....	93
Table 5.4	Results from the adjusted GEE logistic regression linking prior parental monitoring with odds of recent cannabis use. Data come from 1,448 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.....	94

Table 5.5	Results from the ALR linking prior parental monitoring with odds of recent cannabis use and accounting for clustering. Data from 1,448 participants originally recruited in 1985-1986 and followed-up from 1989-1994.....	96
Table 5.6	Formal tests of mediation for the two main paths of interest that were robust in the SEM. Data come from a cohort of 2,311 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.....	104

LIST OF FIGURES

Figure 1.1	The general conceptual model linking parental monitoring with drug use.....	4
Figure 2.1	The developmental model for antisocial behavior.....	27
Figure 2.2	Re-conceptualization of the developmental model for antisocial behavior.....	29
Figure 2.3	The general conceptual model linking parental monitoring with drug use.....	31
Figure 2.4	The general conceptual model linking parental monitoring with drug use extended to include possible mediation, subgroup variation, and other possible explanatory variables.....	32
Figure 3.1	Kaplan Meier estimates of cumulative incidence of cannabis use from age 11 to 17 years. Data come from 619 children sampled from 1983-1985 newborn discharge lists from two hospitals in southeast Michigan.....	50
Figure 3.2	Estimated probability of cannabis initiation from ages 11 to 17 years by level of parental monitoring. Data come from 641 children sampled from 1983-1985 newborn discharge lists in southeast Michigan with complete information on all variables.....	52
Figure 4.1	Unadjusted SEM predicting levels of drug use at age 17 (Drug 17) from levels of drug use (Drug 11), parental monitoring (PM 11) and affiliation with drug using peers at age 11 (Drug11). Data come from 774 children sampled from 1983-1985 newborn discharge lists in southeast Michigan and contributing data to the analyses.....	70
Figure 4.2	Adjusted SEM predicting levels of drug use at age 17 (Drug 17) from levels of drug use (Drug 11), parental monitoring (PM 11) and affiliation with drug using peers at age 11 (Drug11). Data come from 774 children sampled from 1983-1985 newborn discharge lists in southeast Michigan and contributing to the analyses.....	72
Figure 5.1	SEM estimates linking prior level of parental monitoring (PM) with recent cannabis use (CAN). Data come from 1,302 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.....	98

Figure 5.2	SEM estimates linking prior level of parental monitoring (PM) with level of deviant peer affiliation (DPA). Data come from 1,302 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.....	100
Figure 5.3	SEM estimates testing paths from level of parental monitoring (PM) to level of deviant peer affiliation (DPA) to recent cannabis use (CAN). Data come from 1,302 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.....	102

1. CHAPTER ONE

STATEMENT OF THE PROBLEM AND SPECIFIC AIMS

1.1 Statement of the Problem

Cannabis is one of the most widely used psychoactive drugs in the world (Degenhardt et al., 2008). Cumulative occurrence of cannabis use is especially high in the United States. In 2008, for example, over 40% of individuals aged 12 and older had tried cannabis at least once in their lives, with an estimated 2.2 million of them using for the first time within 12 months of the date of assessment (Substance Abuse and Mental Health Service Administration, 2009). Moreover, estimates from the National Comorbidity Survey have projected that nearly one out of every ten individuals who smoke cannabis eventually develop the clinical syndrome of cannabis dependence (James C. Anthony, Warner, & Kessler, 1994). There is also a body of evidence on psychiatric complications of cannabis smoking, the most disabling of which involves schizophrenia or schizophrenia-like illnesses (Morrison et al., 2009; Tien & Anthony, 1990).

The use of cannabis in childhood and adolescence is of particular public health salience. Compared with cannabis users who initiate during adulthood, early cannabis users have been found to experience excess problems related to their use, namely DSM-IV drug abuse or dependence (Chen, O'Brien, & Anthony, 2005). In addition, early cannabis users have been observed to be more likely to go on to use other drugs (D. B. Kandel, 1984; Yamaguchi & Kandel, 1984a), possibly due to increased opportunities to

try other drugs (Wagner & Anthony, 2002b). Recent epidemiological evidence has also shown a link between early cannabis use and later adverse health and social outcomes. These adverse outcomes include educational outcomes and other psychiatric disturbances (Andreasson, Allebeck, Engstrom, & Rydberg, 1987; Fergusson & Boden, 2008a; M. Lynskey & Hall, 2000).

Therefore, it is important to identify potential factors that might prevent or delay cannabis use. One of the potential factors that has received attention is parental monitoring. Parental monitoring is one facet of parenting, and is defined chiefly by parental supervision, tracking and knowledge of child whereabouts (Dishion & McMahon, 1998). The guiding conceptual framework for the link between parental monitoring and drug use and other antisocial behavior has been presented by Patterson and colleagues (Patterson, DeBaryshe, & Ramsey, 1989) and later refined by Dishion and McMahon (Dishion & McMahon, 1998). The developmental progression for antisocial behavior, sometimes referred to as the “social context model”, posits that parental monitoring reduces drug use in part by reducing affiliation with deviant peers.

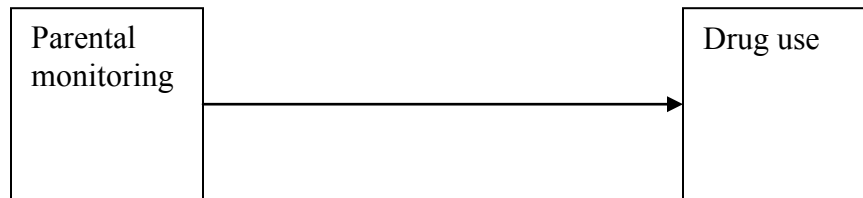
Despite an accumulation of empirical evidence and a well-articulated conceptual framework to link parental monitoring with later drug use initiation in children and adolescents (Dishion & McMahon, 1998), gaps in the literature remain. First, few epidemiologic studies have examined cannabis initiation exclusively in the parental monitoring-drug initiation relationship. Most prior research has grouped all drugs together or has only examined tobacco or alcohol initiation. However, cannabis use in childhood and adolescence is important for the reasons described in the preceding paragraphs. Second, previous studies on parental monitoring and drug use have not been

exhaustive in coverage of potentially important explanatory covariates. Third, it is uncertain whether the association between parental monitoring and drug use is uniform across all subgroups of the population (e.g., across subgroups defined by sex and race/ethnicity). Fourth, prior investigations have been limited in their examination of the link between parental monitoring and subsequent health outcomes to a relatively short time-span, usually no more than two or three years. Fifth, to date, no study on cannabis has tested the main components of the social context model simultaneously within a longitudinal context; that is, tested the paths from earlier parental monitoring to later affiliation with deviant peers to later cannabis use.

This dissertation research will seek to fill in these existing gaps in evidence by examining the role of parental monitoring in cannabis initiation and use. The research is based on longitudinal data from two cohort studies. One study sample consists of a cohort of 823 children born in 1983-1985 in southeast (SE) Michigan. Baseline data were gathered from mothers and children when the children were 6 years of age. Two additional follow-up assessments took place when the cohort members were 11 and 17 years of age. A second study sample is from a cohort study of 2311 youths who were enrolled in first grade in 19 primary schools in an urban public school system in the mid-Atlantic United States during two successive school years in 1985–1986. Follow-up assessments were conducted every year until 1994.

Data from these sources will allow for the estimation of the hypothesized relationships that link parental monitoring with later occurrence of cannabis smoking and other drug use, within the framework of the general conceptual model depicted in Figure 1.1.

Figure 1.1 The general conceptual model linking parental monitoring with drug use



1.2 Specific Research Aims

Three studies in the form of manuscripts will be conducted for the dissertation research, with the following specific aims, each of which involves estimation of specific relationships or paths within the overarching framework of the general conceptual model.

Specific Aim 1: Using data from the SE Michigan cohort, to estimate prospectively the suspected causal association that links parental monitoring in pre-adolescence (age 11) with the cumulative incidence of cannabis use initiation up to late adolescence (age 17), the age interval when cannabis initiation is most likely to occur.

Sub Aim 1a: To test whether the relationship varies between blacks and whites, and/or males and females, or whether a single regression slope estimate serves well to summarize this association.

Sub Aim 1b: To study the time to first cannabis use in relation to level of parental monitoring.

Specific Aim 2: Using data from the SE Michigan cohort, to estimate the influence of level of parental monitoring assessed at age 11 on levels of drug use at age 17, while simultaneously examining potential paths through levels of drug use and deviant peer affiliation at age 11.

Specific Aim 3: Using data from the Mid-Atlantic cohort study, to estimate the suspected influence of earlier levels of parental monitoring on later cannabis use over a five year interval.

Sub Aim 3a: To test whether the association is consistent over the five year interval or whether it varies as a function of age.

Specific Aim 4: Using data from the Mid-Atlantic cohort study, to simultaneously test the suspected paths linking earlier levels of monitoring to subsequent levels of affiliation with deviant peers to later cannabis use.

All of these aims involve specification of a statistical model that is appropriate for estimation.

2. CHAPTER TWO

REVIEW OF THE LITERATURE

2.1 Epidemiology of Cannabis Use

2.1.1 Structure of the Review of the Literature on the Epidemiology of Cannabis Use

The present review of the literature on the epidemiology of cannabis use is organized in relation to the five main rubrics of epidemiology, as suggested by (J. C. Anthony & Van Etten, 1998). Accordingly, the sequence of sub-headings of this section is as follows: (1) *Quantity*, with respect to the global burden of disease associated with cannabis use; (2) *Location*, with respect to subgroups of the population that are differentially affected by cannabis use, and the location of affected subgroups as defined in relation to characteristics of person, place, and time; (3) *Suspected Causes*, with respect to characteristics that have come to be accepted as possible causal determinants of cannabis use; (4) *Mechanisms*, with respect to linkages of states and processes that lead up to cannabis use and that influence its consequences or aftermath (e.g., secondary comorbidities and disabilities or impairments if the condition is not treated effectively); and (5) *Prevention and Control*, with respect to mass action interventions that might be used to reduce incidence, duration, or suffering associated with cannabis use. The review includes the significance of the cannabis use in terms of health and social outcomes and has a particular emphasis on early cannabis use (i.e., use in childhood and adolescence).

2.1.2 Quantity

2.1.2.1 Worldwide Cannabis Use

Globally, cannabis is one of the most commonly used psychoactive drugs. According to estimates from the United Nations, between 129 and 191 million people used cannabis in 2008; equivalent to 2.9% to 4.3% of the entire global population aged 15 to 64 years (UNODC, 2010). In 2008, Oceania, which includes Australia and New Zealand, and the Americas had the highest annual prevalence; 9.3% to 14.8% and 6.3% to 6.6% of their populations aged 15 to 64, respectively (UNODC, 2010). Furthermore, within these regions with a high prevalence of cannabis use, there was substantial variation within region. For example, within the Americas, North America had a higher percentage of past year cannabis use (~10%) compared with South America (~3%) (UNODC, 2010).

Estimates of the cumulative incidence or cumulative occurrence (sometimes called “lifetime prevalence”) of cannabis use have been published recently by the World Mental Health (WMH) consortium (Degenhardt et al., 2008). Cumulative incidence varied widely among the study sites, ranging from less than 1% in the People’s Republic of China to 42.4% in the United States (US) (Degenhardt et al., 2008). European nations had relatively moderate estimates of cumulative incidence (ranging from 6.4% to 19.0%); the Middle East and Africa had relatively low percentages (ranging from 2.7% to 8.4%) (Degenhardt et al., 2008).

2.1.2.2 Cannabis Use in the United States

Turning to evidence from the US, findings from the 2008 National Survey on Drug Use and Health (NSDUH) indicate that an estimated 41% of individuals aged 12 and older have used cannabis at least once in their lives (Substance Abuse and Mental Health Service Administration, 2009). This estimate is consistent with the one obtained by the WMH. According to the NSDUH, an estimated 15.2% of the US household population were current (past-month) cannabis users (Substance Abuse and Mental Health Service Administration, 2009).

The NSDUH also provides estimates for annual incidence, which might have less of the biases associated with estimating cumulative occurrence of use (e.g., older adults having to recall cannabis use earlier in life). In the 2008 NSDUH, an estimated 2.2 million individuals started to use cannabis (Substance Abuse and Mental Health Service Administration, 2009). This number is equivalent to about 0.9% of the total US household population, and 1.5% of those at risk for initiation (i.e., among individuals who have never tried cannabis). The number of recent initiates obtained in 2008, i.e., around 2 million individuals, was similar to estimates obtained for the five preceding years of the NSDUH (Substance Abuse and Mental Health Service Administration, 2009). Further, more than 60% of the new initiates in 2008 were under the age of 18 years when they first tried cannabis (Substance Abuse and Mental Health Service Administration, 2009), indicating that cannabis initiation is most likely to occur at younger ages.

US population-based school surveys, such as Monitoring the Future (MTF), provide additional evidence with respect to the rubric of quantity. In the 2008 MTF survey, an estimated 15% of eighth-graders, 30% of tenth-graders, and 43% of twelfth-graders had ever smoked cannabis (Johnston, O'Malley, Bachman, & Schulenberg, 2009). Taken together, these estimates illustrate that cannabis use in the US is relatively common and usually begins before adulthood.

2.1.3 Location

2.1.3.1 Individual and Sociodemographic Characteristics

There are several sociodemographic characteristics associated with cannabis use, and cannabis initiation, in particular. As previously described in the section on quantity, age is one of the most noteworthy characteristics associated with cannabis use and initiation. Moreover, as reported by the 2008 NSDUH, the mean age at first cannabis use was 17.8 years among recent initiates aged 12 to 49 years (Substance Abuse and Mental Health Service Administration, 2009). In addition, it has been noted that almost two-thirds of new initiates are under the age of 18 years (Substance Abuse and Mental Health Service Administration, 2009). Few individuals initiate cannabis use after the age of 26 years (Substance Abuse and Mental Health Service Administration, 2009).

With respect to sex, it has been commonly observed that males have an excess risk of cannabis initiation (Substance Abuse and Mental Health Service Administration, 2009). These differences might be traced back to exposure opportunity; that is, males

have been found to be more likely to be offered a chance to try cannabis than females, but given this chance to try both sexes are equally likely to go on to use (Van Etten & Anthony, 1999). There is evidence, however, that male-female differences with respect to initiation might be less substantial in recent years (Substance Abuse and Mental Health Service Administration, 2009). Recent evidence from the NSDUH shows virtually no male-female differences with respect to initiation (Substance Abuse and Mental Health Service Administration, 2009).

Differences with respect to race/ethnicity have been observed in cannabis initiation. However, it is often difficult to disentangle these observed associations from social disadvantage. For example, rates of annual cannabis incidence are much higher for American Indians/Alaskan Natives (J. C. Gfroerer, Wu, & Penne, 2002; Substance Abuse and Mental Health Service Administration, 2009); however, American Indians/Alaskan Natives also tend to have lower levels of socioeconomic status.

2.1.3.2 Variation by Place

As presented in the rubric of quantity, there is substantial variation in cannabis use by region and country of the world. It should be noted that it is often difficult to compare cannabis consumption patterns, mainly due to the variation in resources available to collect data. As noted in the UN report, many countries around the world lack adequate resources to conduct large-scale epidemiologic studies of drug use (UNODC, 2010). Therefore, reliable estimates of cannabis use are scarce in these countries. Nonetheless, it is generally observed that countries with relatively higher

levels of per capita income have higher percentages of recent (past-year) and cumulative (lifetime) use (Degenhardt et al., 2008; UNODC, 2010). Highest percentages of cannabis use have been consistently noted for the US, New Zealand, and Australia (UNODC, 2010).

Studies have also shown variation in recent cannabis use at the microsocial level. For example, studies in the US and New Zealand have found that recent cannabis use tends to cluster within neighborhoods (Bobashev & Anthony, 1998; Wells, Degenhardt, Bohnert, Anthony, & Scott, 2009). The estimates are of similar magnitude for both countries (pair-wise odds ratios between 1.3 and 1.6), and are also similar to the amount of clustering seen in villages of the developing world during diarrheal disease outbreaks (Bobashev & Anthony, 1998; Wells et al., 2009).

2.1.3.3 Time Trends

As described previously in the rubric of quantity, the number of annual cannabis initiates among US households has remained relatively stable over the past five years for which there is data, an estimated 2 million individuals aged 12 years and older (Substance Abuse and Mental Health Service Administration, 2009). A report by Gfroerer and colleagues (2002) provides more information on time trends with respect to annual incidence in the US since 1965 (i.e., reliable statistics before 1965 are relatively scarce within the US) (J. C. Gfroerer et al., 2002). The authors found that the rate of annual cannabis initiation rose from less than 5 incident users per 1000 person-years for individuals aged 12 years and older during the late 1960s and early 1970s to a peak in

1976-1977 of more than 20 incident users per 1000 person-years (J. C. Gfroerer et al., 2002). Since that peak, cannabis initiation declined to 8.5 per 1000 person-years in 1990, and then increased steadily again to almost 17 per 1000 person-years in 1996 (J. C. Gfroerer et al., 2002). After 1996, rates declined to 13.6 per 1000 in 1999 (J. C. Gfroerer et al., 2002). In the MTF, the prevalence of recent (past-year) use among twelfth graders peaked in 1978 and 1979 and decreased during the 1980s until 1992, when it doubled from 1992 to 1997 from an estimated 22% to 39% (Johnston et al., 2009). Since 1997, annual prevalence has declined and leveled to an estimated 32% (Johnston et al., 2009).

The general trends described above were observed for both males and females; however, rates for males were higher. The mean age at first use decreased from about 19 years in the early 1970s to 17 years in the 1990s (J. C. Gfroerer et al., 2002). Recent data from the NSDUH found that the average age of new initiates was just under 18 years (Substance Abuse and Mental Health Service Administration, 2009). Similarly, evidence from the cross-sectional WMH surveys found that use was especially common in younger cohorts (Degenhardt et al., 2008).

2.1.4 Suspected Causes

2.1.4.1 Family History and Genetics

Familial studies have presented evidence for the shared influences of cannabis use. For example, parent-child correlations of cannabis use have been estimated to range from 0.3 to 0.5 (Brook, Whiteman, Gordon, & Brook, 1985; J. Gfroerer, 1987).

Likewise, correlations between older brothers and younger siblings have been observed to be of similar magnitude (Brook, Whiteman, Gordon, & Brenden, 1983). In other epidemiologic research along these lines, the odds of lifetime cannabis use disorders were substantially elevated in siblings, adult children, and spouses of probands with cannabis use disorders (odds ratios ranging from 3.6 to 6.9) (Merikangas et al., 2009). These findings are consistent with previous findings reported by the authors, and of similar magnitude (Merikangas et al., 1998).

Twin studies have been able to provide further clues about genetic, shared environmental, and non-shared environmental sources of variance. Kendler and colleagues have estimated the heritability, i.e., the genetic influence, of cannabis use using male and female population-based twin registries; the estimates ranged from 0.4 for females to 0.3 for males (Kendler, Karkowski, Neale, & Prescott, 2000; Kendler & Prescott, 1998). In these studies, shared and non-shared environmental influences were roughly equivalent for males (~0.3) but differed for females (0.35 for shared and 0.25 for non-shared) (Kendler et al., 2000; Kendler & Prescott, 1998). Other studies have shown similar results with respect to females; however, male estimates differed somewhat (e.g., Lynskey and colleagues observed 0.67 for genetic influences in males) (M. T. Lynskey et al., 2002; Miles et al., 2001).

Although twin studies are able to differentiate between sources of variation due to genetics and shared and non-shared environments, they do not have the ability to explain what the actual causal agents are with respect to each influence. Currently, there has been minimal progress made in identifying the actual genes related to cannabis use; however, there are some exceptions that will need to be replicated in other samples

(Johnson et al., 2008; Stallings et al., 2005; Stallings et al., 2003; Uhl, Liu, Walther, Hess, & Naiman, 2001). In addition, future studies are needed to elucidate possible genetic and environmental interplay in relation to cannabis use.

2.1.4.2 Parenting Practices

A number of studies have been conducted with respect to the potential impact of parent-child relationships and parenting practices on drug use, including cannabis use. A large body of evidence has linked lower levels of parental monitoring, defined as tracking, supervision, and knowledge of child whereabouts, with increased risk for drug use and initiation (Chilcoat & Anthony, 1996; Chilcoat, Dishion, & Anthony, 1995; DiClemente et al., 2001). Nevertheless, the association has been less well studied in the context of cannabis initiation and use and the consistency of the findings from the few studies that have examined the specific link between parental monitoring and cannabis use is mixed. For example, a cross-sectional study of adolescent females found that teens with lower parent monitoring had an estimated two-fold increased odds of cannabis use (DiClemente et al., 2001). Other longitudinal studies have reported similar results; namely, that low parental monitoring predicted adolescent cannabis use (Hayatbakhsh et al., 2008; Martins, Storr, Alexandre, & Chilcoat, 2008). With respect to initiation, in a longitudinal study of Seattle youths followed from the ages of 10 to 18 years, Kosterman and colleagues (2000) found that parents' proactive family management, a variable assessing parents' monitoring, rules, discipline and reward practices, was associated with reduced risk of cannabis initiation (Kosterman, Hawkins, Guo, Catalano, & Abbott,

2000). A latent class analysis of cannabis use patterns in a sample of black middle-school students also found that lower parental monitoring was associated with increased odds of membership in a latent class characterized by cannabis use and problems in sixth grade (Reboussin, Hubbard, & Ialongo, 2007). However, the estimated relationship in that study was attenuated and non-significant by seventh and eighth grade. It is important to note that that study focused upon early cannabis involvement and did not cover the grades or ages when cannabis use is most likely to occur. Similarly, using data from the National Survey of Parents and Youth, Tang and Orwin (2009) found that parental monitoring signaled lower odds of cannabis initiation at ages 12 and 13 years, but not for ages 14 through 16 years (Tang & Orwin, 2009). In other work on parent-child bonding, authors have suggested that bonding might protect youths and reduce the risk for cannabis use (Brook, Richter, & Whiteman, 2000).

2.1.4.3 Affiliation with Deviant and Drug Using Peers

Affiliation with drug using peers is one of the strongest and most consistent predictors of drug initiation in childhood and adolescence (Guo, Hill, Hawkins, Catalano, & Abbott, 2002; van den Bree & Pickworth, 2005). For example, a longitudinal study of adolescents in Germany found that higher levels of affiliation with drug using peers predicted initiation and regular cannabis use (Hofler et al., 1999; von Sydow, Lieb, Pfister, Hofler, & Wittchen, 2002). These results are similar to findings from longitudinal studies in the US and New Zealand (Fergusson, Swain-Campbell, & Horwood, 2002; van den Bree & Pickworth, 2005). In addition, there is some evidence

that peers might be more influential in earlier adolescence than later adolescence and adulthood (Fergusson, Swain-Campbell et al., 2002; Monahan, Steinberg, & Cauffman, 2009); although this finding is not universal (Guo et al., 2002). Moreover, there is a plausible hypothesized mechanism that links affiliation with drug using peers with increased opportunities to use cannabis (Lloyd & Anthony, 2003).

2.1.4.4 Intra-Individual Traits and Preexisting Psychiatric Problems

Related personality traits, namely, aggression, delinquency, novelty-seeking, and risk-taking have been consistently linked with early drug use (Brook, Whiteman, Cohen, & Tanaka, 1992; Molina & Pelham, 2003; Rios-Bedoya, Wilcox, Piazza, & Anthony, 2008; Rosenberg & Anthony, 2001a; van den Bree & Pickworth, 2005). For example, Rios-Bedoya et al. (2008) found that a measurement of risk taking was associated with onset of cannabis use by young adulthood (Rios-Bedoya et al., 2008). Similarly, studies have reported an association between conduct and/or attention problems and early drug use, including cannabis use (Fergusson, Horwood, & Ridder, 2007; Molina & Pelham, 2003). In a longitudinal study, Molina and Pelham (2003) found that early adolescent conduct and attention problems predicted later drug use (Molina & Pelham, 2003). Another study found a synergistic impact of conduct and attention problems on later drug use (Flory, Milich, Lynam, Leukefeld, & Clayton, 2003). It is not known, however, if drug use is an extension of these related traits and earlier behavior problems or a consequence of them.

2.1.5 Mechanisms

2.1.5.1 The Gateway Hypothesis and Exposure Opportunity

A series of reports by Kandel and colleagues have found that cannabis use is often preceded by tobacco and alcohol use (D. Kandel, 1975; D. Kandel & Faust, 1975; D. B. Kandel, Yamaguchi, & Chen, 1992; Yamaguchi & Kandel, 1984a, 1984b). Studies have also shown that regular cannabis users are more likely to go on to use other drugs, such as cocaine and heroin (D. Kandel, 1975; D. Kandel & Faust, 1975; D. Kandel & Yamaguchi, 1993). In addition, early cannabis users (i.e., individuals who use before the age of 18) are more likely to go on to use other drugs (Yamaguchi & Kandel, 1984a, 1984b). These observations led to the development of a “gateway” hypothesis, which posits that early tobacco and alcohol use leads to cannabis use, which in turn leads to other drug use, such as cocaine and heroin (D. B. Kandel & Yamaguchi, 1985). It should be noted, however, that there is considerable debate surrounding the gateway theory (J. C. Anthony, 2002; Morral, McCaffrey, & Paddock, 2002; Morral, McCafrey, & Paddock, 2002).

The gateway hypothesis is a useful model with respect to relating stages of drug use; however, it is descriptive in nature and does not explain the underlying mechanisms responsible for the transition processes. Therefore, several research groups have tested mechanisms that go beyond the explanatory nature of the gateway process. For example, in a large national dataset Wagner and Anthony (2002) drew upon the concept of “exposure opportunity,” i.e., the fact that drug initiation cannot occur unless there is an

opportunity to try it, to examine whether individuals who used tobacco and alcohol had increased opportunities to use cannabis and whether they were more likely to use cannabis once an opportunity had occurred (Wagner & Anthony, 2002b). The authors also examined whether cannabis smokers were more likely to use cocaine given an opportunity to try cocaine (Wagner & Anthony, 2002b). The study found that, compared with non-users, tobacco and alcohol users were much more likely to have an opportunity to try cannabis and to use cannabis once an exposure opportunity had occurred (Wagner & Anthony, 2002b). Cannabis users were also much more likely to use cocaine once an opportunity had arisen (Wagner & Anthony, 2002b). In addition, the authors concluded that the observed sequences could not be explained by drug-seeking behavior because of the time constraints they had placed on the data (Wagner & Anthony, 2002b). In another test of the association between cannabis use and other subsequent drug use, Fergusson and Horwood (2000) found that affiliation with drug using peers predicted an increased risk of drug use; however, it did not explain completely the association between cannabis use and other drug use (Fergusson & Horwood, 2000). In twin research along these lines, Gillespie et al. (2009) found that cannabis availability, was the most important factor for initiation of cannabis smoking (Gillespie, Neale, & Kendler, 2009).

Using a discordant twin design, Lynskey et al. (2003) has probed other aspects of the gateway hypothesis; namely, the possibility that genetic influences explain the gateway phenomenon (M. T. Lynskey et al., 2003). The study found that twins who had used cannabis before the age of 17 years were more likely to use other drugs subsequently, compared with their co-twins (M. T. Lynskey et al., 2003). The finding points to the potential importance of other influences, rather than genetic factors, since

twins should be no different with respect to other drug use if there was a common genetic factor (M. T. Lynskey et al., 2003). Other longitudinal and population-based studies have adjusted for an array of familial and personal factors, including behaviors and attitudes, and continued to find an association linking prior cannabis use with other subsequent drug use (Fergusson & Horwood, 2000). Despite the evidence, the possibility remains that the association between early cannabis use and other drug use is spurious, arising from a common underlying individual susceptibility.

2.1.5.2 Cannabis Dependence

It is noteworthy that the majority of individuals who initiate cannabis use never go on to experience the cannabis dependence syndrome (James C. Anthony et al., 1994). Nonetheless, some users do develop the clinical syndrome of cannabis dependence, which is likely to be the most common adverse outcome related to cannabis use. The chief features of cannabis dependence are characterized by disturbances of the mental life (e.g., recurrent thoughts about use), disturbances of behavior (e.g., repetitions of cannabis-related behavior), and neuroadaptation related to use (e.g., feeling tolerance) (J. C. Anthony, 2006).

Using data from the National Comorbidity Survey (NCS), Anthony et al. (1994) have estimated that one out of every 9 to 11 individuals (~10%) who smoke cannabis eventually develop the clinical syndrome of cannabis dependence (James C. Anthony et al., 1994). Wagner and Anthony (2002) have also shown that the estimated risk for becoming dependent upon cannabis peaks in the second or third year after onset; then,

from the fifth year and later drops toward the null (Wagner & Anthony, 2002a). In a study of the emergence of clinical features among cases of dependence, loss of control and continuation of use despite harm were the most common initial features (Rosenberg & Anthony, 2001b).

Cannabis smokers who initiate before adulthood might experience a larger burden of cannabis dependence. Early onset cannabis smokers have been shown to develop problems related to their use, i.e., clinical features of dependence, more quickly than later-onset cannabis users, and are more likely to develop cannabis dependence within 24 months after onset of use, even when elapsed time from onset is taken into account (Chen et al., 2005). Specifically, the risk might be one in six among those who initiate before the age of 18 as compared to the overall estimate of one in ten (J. C. Anthony, 2006). Nonetheless, the causal implications of these findings remain unclear. It is possible that early-onset cannabis use causes greater risk of becoming dependent; however, individuals prone to developing dependence might also be more prone to early onset use.

2.1.5.3 Educational Outcomes

A number of studies have found associations linking cannabis use with poor school performance and reduced educational attainment. A literature review by Lynskey and Hall (2000) summarized the influence of cannabis use in adolescence on educational outcomes (M. Lynskey & Hall, 2000). The authors found evidence for the hypothesis that cannabis use contributes to poor achievement (M. Lynskey & Hall, 2000). They also suggest the possibility that poor school performance predicts cannabis use, citing

additional evidence (M. Lynskey & Hall, 2000). A third possibility is that an underlying common causal factor influences both cannabis use and poor educational outcomes (e.g., externalizing problems); however, empirical examinations of this idea have shown that even after adjustment for other covariates, cannabis use continues to be associated with reduced educational attainment (M. Lynskey & Hall, 2000).

2.1.5.4 Other Psychiatric Consequences

A body of evidence links cannabis use, especially early use, with other subsequent psychiatric complications and co-morbidities. A highly contentious topic is the potential causal relationship between cannabis use and psychotic symptoms and disorders. For example, studies on a Swedish male cohort have reported a dose-response relationship linking the number of times cannabis was used before the age of 18 years and an excess risk of schizophrenia, even after adjustment for an array of potential confounding variables such as IQ (Andreasson et al., 1987). These findings have been subsequently replicated by other studies in other parts of the world (Henquet et al., 2005; van Os et al., 2002). Self-medication, i.e., cannabis is used with the intention to reduce symptoms of schizophrenia, has generally not been supported in the literature (Henquet et al., 2005; van Os et al., 2002). Alternative explanations for the association, namely the possibility that the association is an artifact of an unmeasured confounder or an underlying susceptibility for both cannabis use and psychoses, have not been completely ruled out.

A number of studies have also examined the causal relationship between cannabis use and depression. In comparison to the research on cannabis and schizophrenia,

weaker evidence exists for a modest association (O.R. <2.0) between cannabis use and depression in epidemiologic cross-sectional and longitudinal studies (Chen, Wagner, & Anthony, 2002; Fergusson, Horwood, & Swain-Campbell, 2002). However, authors of the studies have not been convinced that they have completely controlled for potential confounders.

There have been associations linking cannabis use and other psychiatric symptoms and disorders, as well. For example, in a longitudinal study in New Zealand, Fergusson and Boden (2008) have reported on the increased risk for adult-onset attention deficit hyperactivity disorder (ADHD) symptoms in early cannabis users (Fergusson & Boden, 2008a). However, replication of this finding is warranted. In all of the studies of psychiatric co-morbidities subsequent to cannabis use a common limitation is the possibility of uncontrolled background variables or underlying susceptibilities; however, conducting a study with random assignment to early cannabis use is not possible due to obvious ethical considerations. Nonetheless, in future research on cannabis cessation there is the possibility of randomly assigning treatment and observing the occurrence of secondary endpoints, such as other psychiatric co-morbidities, among the intervention groups.

2.1.6 Prevention and Control

Epidemiological evidence is scarce with respect to prevention and control of drug use and/ or cannabis use. A school-based randomized trial on the effectiveness of Drug Abuse Resistance Education (DARE) showed no school differences with respect to drug

use at either five-year or ten-year assessment (Clayton, Cattarello, & Johnstone, 1996; Lynam et al., 1999). Spoth et al. (2009) have recently reported on a promising school-based intervention aimed at reducing opportunities to use drugs and drug use (Spoth, Gyll, & Shin, 2009). The authors found that the intervention, the Iowa Strengthening Families Program, was associated with reduced exposure to drug use (odds ratios ranging from 1.2 to 2.4) and reduced twelfth grade drug use (odds ratios ranging from 2.9 to 6.4) compared with the control condition (Spoth et al., 2009). Other promising school-based interventions have been reported, highlighting the need for “booster” follow-up interventions to improve efficacy (Botvin, Baker, Dusenbury, Botvin, & Diaz, 1995). Mass media and advertising campaigns aimed at reducing drug use have been less well studied. Nonetheless, a study by Block et al. (2002) found reduced cannabis use among individuals able to recall anti-drug advertising (Block, Morwitz, Putsis, & Sen, 2002). Future research is needed on the effectiveness of population-based intervention and prevention efforts in all segments of the population.

2.2 Prior Research on Parental Monitoring

The next sections of the dissertation present more information on a plausible prevention and intervention target and suspected cause of drug use, i.e., parental monitoring. The definition of parental monitoring and conceptual models linking parental monitoring with drug use are described in more detail.

2.2.1 Definition of Parental Monitoring

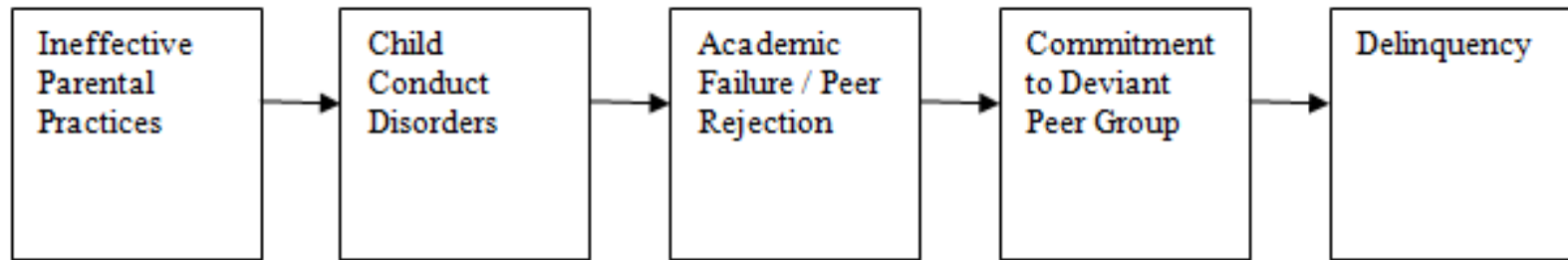
Dishion and McMahon (1998) have suggested that parental monitoring, which includes tracking, supervision, and knowledge of child whereabouts, is one component of three interrelated dimensions of parenting (Dishion & McMahon, 1998). The other two dimensions encompass motivation (e.g., norms, values, and goals) and behavior management (e.g., setting limits and negotiating) (Dishion & McMahon, 1998). Parental monitoring occurs in the home, school, and community; that is, in all of the environments of the child (Dishion & McMahon, 1998). It is also a salient part of the child's development from infancy to young adulthood and should be contextually and culturally appropriate (Dishion & McMahon, 1998).

2.2.2 Parental Monitoring Conceptualized within a Developmental Model for Antisocial Behavior

Patterson et al. (1989) have provided a conceptual framework, the developmental model for antisocial behavior, which others have called the “social context model”, for understanding the relationship between parental monitoring and antisocial behavior, including drug use (Patterson et al., 1989). The model is sketched in Figure 2.1 and posits that in early childhood ineffective parental practices, including monitoring, lead to child conduct disorders (Patterson et al., 1989). In turn, the conduct disorders precipitate academic failure and peer rejection in middle childhood (Patterson et al., 1989). In late childhood and early adolescence, rejection and academic failure prompt commitment to

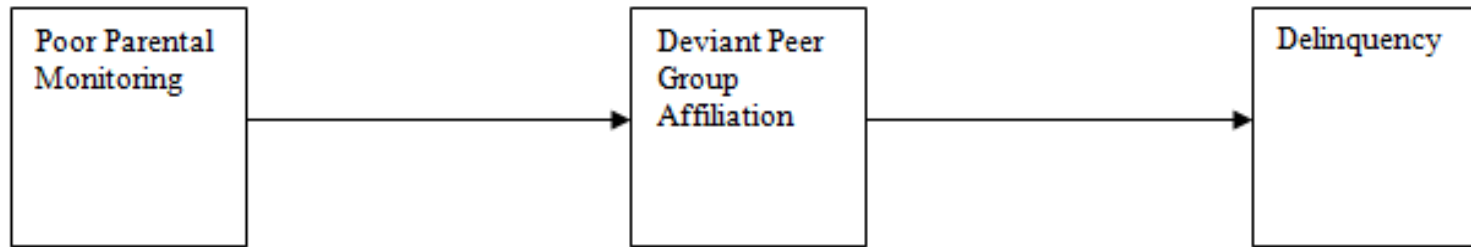
deviant peer groups and delinquency, including drug use (Patterson et al., 1989). The model weaves together empirical evidence from previous findings (Patterson et al., 1989).

Figure 2.1 The developmental model for antisocial behavior (Patterson et al. 1989)



Recent epidemiological studies have tested components of this model. For example, Lloyd and Anthony (2003) found that higher levels of parental monitoring were associated with lower levels of affiliation with deviant peers across late childhood and early adolescence (Lloyd & Anthony, 2003). Other longitudinal studies have observed associations linking higher parental monitoring with reduced drug use initiation (Chilcoat & Anthony, 1996; Chilcoat et al., 1995). However, recent evidence is not consistent and does not support the link between parental monitoring and poor school performance. For example, Coley and Hoffman (1996) found that lower levels of monitoring were associated with higher math achievement scores in third and fourth graders from two parent families (Coley & Hoffman, 1996). In another study of sixth graders, highest grade point averages were associated with more moderate levels of parent monitoring (Kurdek, Fine, & Sinclair, 1995). Therefore, some have re-conceptualized the antisocial model, which is displayed in Figure 2.2 (Dishion & McMahon, 1998; Lloyd & Anthony, 2003). The model in Figure 2.2 is more similar to recent conceptual models described by Dishion and McMahon (Dishion & McMahon, 1998). Drawing upon the evidence, this model links parental monitoring to subsequent affiliation with deviant peers to subsequent delinquency (e.g., drug use).

Figure 2.2 Re-conceptualization of the developmental model for antisocial behavior



2.2.3 General Conceptual Models Linking Parental Monitoring with Cannabis Use

The general conceptual model for this dissertation is presented in Figure 2.3. In this model, there is a direct path from parental monitoring to cannabis use. This model can be elaborated to test for vectors of possible mediators (e.g. deviant peer affiliation), vectors of potential markers of subgroup variation or effect-modifiers (e.g. sex and race/ethnicity), and vectors of other potential explanatory variables. Figure 2.4 depicts this extension of the general conceptual model.

Figure 2.3 The general conceptual model linking parental monitoring with drug use

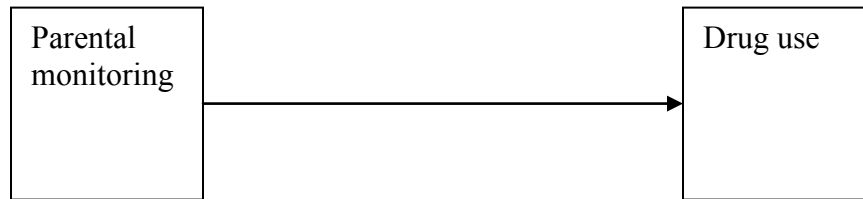
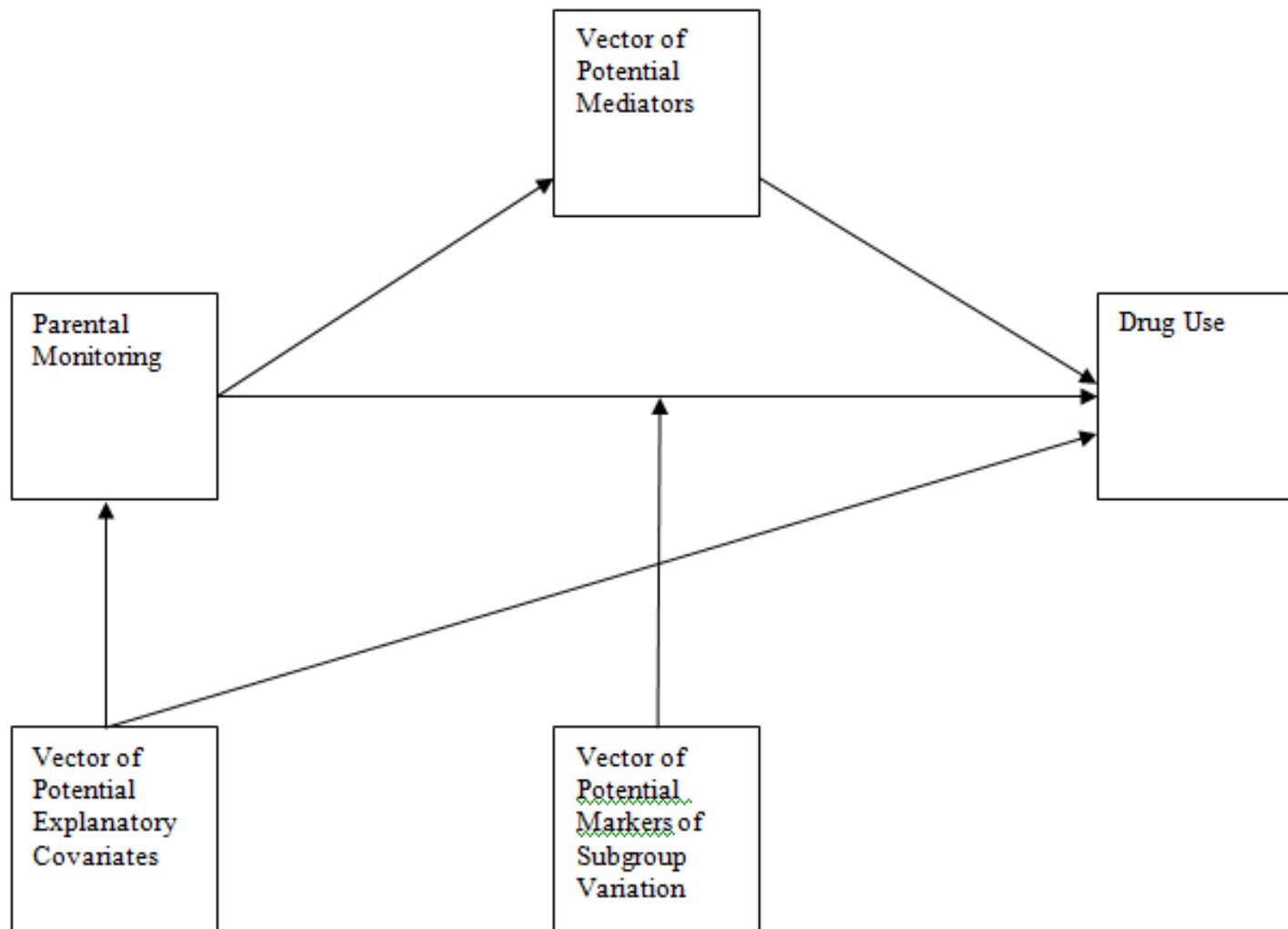


Figure 2.4 The general conceptual model linking parental monitoring with drug use extended to include possible mediation, subgroup variation, and other possible explanatory variables



2.3 Conclusions

Cannabis use, especially use before the age of 18 years, is a serious public health problem. In the US, the treatment burden (in total number of people) associated with cannabis use is second only to alcohol use (Substance Abuse and Mental Health Service Administration, 2009). Moreover, early cannabis users have been found to experience excess problems related to their use, namely DSM-IV drug abuse or dependence, and have been shown to be more likely to go on to use other drugs (Chen et al., 2005; D. Kandel, 1975). Recent epidemiological evidence has also suggested a link between early cannabis use and later adverse health and social outcomes (Brook, Adams, Balka, & Johnson, 2002). These adverse outcomes include poor educational outcomes and other psychiatric disturbances, which are associated with decreased productivity and higher health care costs (Brook et al., 2002; Chen et al., 2005; Fergusson & Boden, 2008a; M. Lynskey & Hall, 2000; Tien & Anthony, 1990; Wagner & Anthony, 2002a, 2002b). Hence, there is a substantial need for the identification of possible targets for prevention and early intervention of cannabis use. This dissertation research will use data from two longitudinal studies to probe the long-term influence of a suspected causal agent and potential target for cannabis prevention, parental monitoring.

3. CHAPTER THREE - MANUSCRIPT ONE

PARENTAL MONITORING AT AGE 11 AND SUBSEQUENT ONSET OF CANNABIS USE UP TO AGE 17: RESULTS FROM A PROSPECTIVE STUDY

ABSTRACT

Background: Early-onset cannabis smoking is a risk marker for subsequent adverse psychiatric outcomes. Delay or prevention of early-onset drug use might be achieved via parenting interventions such as programs to increase parents' effective monitoring and supervision of their children. The aim of this study is to estimate, prospectively, the influence of parental monitoring assessed at age 11 on the initiation of cannabis use before age 18 years.

Methods: Data are from a longitudinal study of 823 low birth weight and normal birth weight children randomly selected from 1983-1985 newborn discharge lists of two major hospitals in southeast Michigan, one serving inner-city mothers and the other, suburban mothers. Parental monitoring was assessed at age 11 via 10 items, and the parental monitoring – cannabis prediction was estimated for the 641 children who were assessed at baseline, at age 11 years, and at age 17 years. Poisson regression with robust error variances was used to estimate the predictive association that links levels of parental monitoring at age 11 with the risk of cannabis use up to age 17, adjusting for other important covariates.

Results: Higher levels of parental monitoring at age 11 were associated with a reduced risk of cannabis initiation from ages 11 to 17 years (adjusted estimated relative risk = 0.96; 95% confidence interval = 0.94, 0.98).

Conclusions: This prospective investigation found that higher levels of parental monitoring predicted a reduced occurrence of cannabis initiation from ages 11 to 17 years. Consistent with evidence reported elsewhere, these findings from prospective research help confirm a theory that parenting and familial characteristics might exert long-lasting influences on a child's risk of starting to use illegal drugs.

3.1 Introduction

In the United States in 2008 more than two million individuals aged 12 years and older smoked cannabis for the first time (Substance Abuse and Mental Health Service Administration, 2009). Most of these recent initiates were adolescents younger than 18 years of age. This group of early-onset cannabis users (i.e., those who started to use before the age of 18) is of particular public health salience for several reasons. Early onset cannabis smokers are more likely to develop cannabis dependence within 24 months after onset of use, even when elapsed time from onset is taken into account (Chen et al., 2005). It is important to note, however, that the causal implications of these findings remain unclear. The possibility of a common underlying susceptibility for both early cannabis use and cannabis dependence has not been completely ruled out. Early cannabis users are also more likely to go on to use other drugs (D. B. Kandel, 1984; Yamaguchi & Kandel, 1984a), possibly due to increased opportunities to try these other drugs (Wagner & Anthony, 2002b). In addition, there is some indication that cannabis users might be more likely to experience other subsequent psychiatric problems (Fergusson & Boden, 2008a, 2008b; Tien & Anthony, 1990).

Given these findings, early adolescence might be a critical developmental interval with respect to preventing or delaying onset of cannabis use. Parental monitoring (i.e., awareness, tracking, and supervision of children's activities) is a specific facet of parenting influence that may lend itself to preventive approaches. A series of studies have estimated associations linking higher levels of parental monitoring with lower odds of drug initiation (Chilcoat & Anthony, 1996; Chilcoat et al., 1995; DiClemente et al., 2001). Several potential mechanisms have been posited. One suggestion is that children

with higher levels of parental monitoring are less likely to associate with deviant peers, thereby reducing their exposure to drugs (Lloyd & Anthony, 2003). In twin research along these lines, Gillespie et al. (2009) found that cannabis availability, which the authors noted might be determined by aspects of parent-child relationships like monitoring, was the most important factor for initiation of cannabis smoking (Gillespie et al., 2009).

Against this background of empirical evidence and a well-reasoned theoretical framework to link parental monitoring with later drug use initiation in children and adolescents (Dishion & McMahon, 1998), several gaps in the literature remain. First, few epidemiologic studies have specifically examined cannabis smoking in the past research on the parental monitoring-drug initiation relationship. Most prior research has grouped all drugs together or has only examined tobacco or alcohol initiation. However, as described above, early-onset cannabis smokers represent a potentially important risk subgroup. Second, previous studies on parent monitoring and cannabis initiation have not always considered potentially important covariates, namely, maternal smoking and peer influences. Third, it is uncertain if the association between parental monitoring and drug use is uniform across certain subgroups of the population, specifically across subgroups defined by sex and race/ethnicity. For example, a previous study found that higher levels of parental monitoring were associated with lower odds of tobacco smoking initiation only among white adolescents (Bohnert, Rios-Bedoya, & Breslau, 2009). Fourth, previous studies have had relatively short follow-up periods, usually no more than two years, and have not covered the ages when cannabis initiation is most likely to occur.

Here, in this new research, the focus is on early-onset cannabis smoking, and the epidemiological evidence is from a cohort of urban and suburban children from a large Midwest metropolitan area. The aim is to estimate prospectively the relationship that links parental monitoring in pre-adolescence (age 11 years) with the cumulative incidence of cannabis use up to late adolescence (age 17 years). The present investigation overcomes the gaps in the literature in three specific ways. First, the late-childhood and adolescent intervals that are covered are the times when cannabis initiation is most likely to occur and where problems related to use appear most frequently (i.e., the clinical features of cannabis dependence and the clinical syndrome of cannabis dependence). Second, tests are conducted to examine whether the association between monitoring and onset of cannabis use varies between blacks and whites, and for males versus females. Third, the investigation takes into account important covariates, including peer smoking and drinking, as well as maternal smoking.

3.2 Methods

3.2.1 Sample

Data are from a longitudinal study on the neuropsychiatric sequelae of low birth weight (LBW) and normal birth weight (NBW) children. Detailed information on the sample is available elsewhere (Breslau et al., 1996), and is briefly summarized here. LBW and NBW children were randomly selected from 1983-1985 newborn discharge lists of two hospitals in southeast Michigan, one serving an inner-city community and the

other serving a suburban community. Forty-seven children with severe neurologic impairments were excluded from the initial sample. Of the 1095 children eligible for the study, 823 (75.2%) participated in the initial assessment from 1990 to 1992, when they were six years of age. Follow-up assessments were conducted at 11 years (n=717; 87.1%) and 17 years of age (n=713; 86.6%). Six hundred and fifty-seven children completed both age 11 and age 17 follow-up assessments. The institutional review boards of the participating institutions approved the study.

3.2.2 Measures

3.2.2.1 Cannabis Initiation, Ages 11-17 Years

The cumulative incidence of cannabis use up to age 17 was assessed during the age 17 assessment via the following dichotomous (coded *Yes (1)* or *No (0)*) question about cannabis use: “Have you ever, even once, used marijuana or hashish?” Data from the age 11 assessment were used to identify children who had initiated cannabis smoking by age 11 and were therefore no longer at risk for initiation during the 11 to 17 years age-span. In addition, for those respondents who had initiated cannabis by age 17, a follow-up question queried the age of their first cannabis use.

3.2.2.2 Parental Monitoring, Age 11 Years

Level of parental monitoring was assessed by child self-report at age 11 via a standardized 10-item scale (the full scale is attached in Appendix D) (Capaldi & Patterson, 1989; Chilcoat et al., 1995). The items encompass child supervision and tracking of activities outside the school environment (e.g., whether an adult was present within 1 hour of the child arriving home from school, how often the child talked with the parents about plans for the coming day, and whether the child knew how to contact the parents if they were not at home after school). On seven of the items, children responded on a five-point scale ranging from *All of the time (1)* to *Never (5)*; responses were coded either *Clear (1)* or *Unclear (2)* on two items; and on a single item the responses were coded *Yes (1)* or *No (2)*. A parental monitoring score was constructed by reversing the coding and summing the scores on the 10 items. Possible scores ranged from a low of 10 to a high of 41.

3.2.2.3 Child Covariates

Tobacco smoking and alcohol use at age 11 were assessed via standardized child-reported drug questions at age 11 years. Peer smoking and alcohol use was assessed by child self-report at age 11 years via two *Yes* or *No* questions: “Do you have any friends around your age who ever smoke tobacco cigarettes?” and “Do you have any friends around your age who ever drink alcohol?” Community, Sex, and Race were as assessed at baseline. Birth weight was obtained from hospital records.

3.2.2.4 Maternal Covariates

Maternal smoking was assessed at baseline when children were 6 years of age. Mothers were classified as smokers if they had ever smoked daily for one month or more up to the time of the interview. Maternal education and maternal marital status were also assessed at baseline.

3.2.3 Statistical Analysis

Using a method described by Zou (2004), unadjusted and adjusted Poisson regressions with robust error variances were conducted to estimate the predictive association linking parental monitoring at age 11 years with the initiation of cannabis use from the ages of 11 to 17 years (Zou, 2004). This estimate has been shown to yield more precise estimates of relative risk than the odds ratio derived in traditional logistic regression, especially when the outcome of interest occurs in greater than 10% of the sample (i.e., when estimated odds ratios may not approximate relative risks) (Zou, 2004). Male-female subgroup variation in the parental monitoring-cannabis initiation association was evaluated via product terms, as was subgroup variation associated with race. None were detected at the alpha level of 0.05.

For a subsidiary survival analysis, a dichotomous variable was constructed from the original parental monitoring scale using the median as the cutoff (*low parental monitoring* (<37) and *high parental monitoring* (≥ 37)). Next, Kaplan-Meier curves were derived for the above- and below-median monitoring groups in order to inspect the

failure rates of first cannabis use, year by year from age 11 to 17 years. Specifically, the approach involved specifying the elapsed time from the age at the age 11 assessment until the age of first cannabis use. Adolescents who never used cannabis contributed person-years up to the time of their age at the age 17 interview. A total of 619 children with age of onset information contributed data to the Kaplan-Meier analysis. A log-rank test was conducted to formally test whether the two survival curves differed from one another ($\alpha = 0.05$).

Finally, in a post-estimation exploratory step, the method of plotting fractional polynomials, as described by Royston and Altman (1994), was employed to probe into the issue of possible non-linearity in the parental monitoring – cannabis initiation association (Royston & Altman, 1994). All analyses were conducted using Stata 11 (StataCorp, 2009).

3.3 Results

3.3.1. Comparison of the Original Sample with the Follow-up Sub-samples at Ages 11 and 17 Years

Six hundred fifty seven of the initial 823 children participated in both waves of follow-up assessment. For focus on cannabis onset after level of parental monitoring was assessed, individuals who had used cannabis before age 11 years were excluded ($n=7$). Nine children had missing information on one or more of the covariates of interest. Therefore, the resulting sample size for the analysis was $n=641$. As shown in Table 3.1,

the initial sample differs negligibly from the subset followed up at age 11 and 17 years and from the subset used in the regression analyses, after exclusions noted above.

Table 3.1 Description of the initial sample, the subset with follow-up data for ages 11 and 17, and the subset used in the regression analyses. Data come from 823 children sampled from 1983-1985 newborn discharge lists in southeast Michigan and assessed at ages 6, 11, and 17 years.

	Initial sample (n=823) %	Sample with follow-up data for ages 11 and 17 (n=657) %	Sample used in the regression analyses (n=641) %
Urban	50.2	52.4	51.8
Low birth weight	57.5	56.9	56.8
Black	42.9	46.3	45.4
Male	48.6	46.6	46.2
Mothers' education			
< High school	16.9	16.0	15.6
High school	27.5	26.2	26.5
Some college	37.3	38.5	38.4
College	18.3	19.3	19.5
Single mother	32.8	32.6	32.4

3.3.2 Parental Monitoring at Age 11 and the Risk of Cannabis Initiation up to Age 17

An estimated 35% of the 641 individuals started smoking cannabis during the follow-up period, from ages 11 to 17 years. The third column in Table 3.2 depicts the cannabis initiation rate during follow-up (%) for each of the covariates of interest. Table 3.2 also depicts estimates from the unadjusted Poisson regressions linking the covariates of interest with the cumulative incidence of cannabis use from age 11 up to age 17 years; Table 3.3 depicts the adjusted regressions. With respect to parental monitoring, an increase of one point on the parental monitoring scale signaled an estimated five percent decrease in the likelihood of initiating cannabis smoking by age 17 years (estimated relative risk, ERR, = 0.95; 95% confidence interval, CI, = 0.93, 0.97). A slightly attenuated but statistically robust association remained after adjustment for other important covariates (adjusted ERR, AERR, = 0.96; 95% CI = 0.94, 0.98). No subgroup variation for sex or race was detected at the alpha level of 0.05 (i.e., product-terms for sex and race with parental monitoring were not statistically significant).

Other findings of interest are presented in the adjusted table (Table 3.3). Compared with white adolescents, black adolescents were less likely to initiate cannabis use from age 11 to age 17 years (AERR = 0.64; 95% CI = 0.48, 0.87). Males were more likely to initiate cannabis use during follow-up (AERR = 1.25; 95% CI = 1.01, 1.56). Smoking tobacco by age 11 was associated with an increased risk of cannabis initiation from the ages of 11 to 17 years (AERR = 1.31; 95% CI = 1.02, 1.69). Having a friend who smoked at age 11 was also robustly associated with initiating cannabis during follow-up (AERR = 1.63; 95% CI = 1.31, 2.03). With respect to maternal covariates,

having a single mother at baseline and having a mother who smoked tobacco at baseline were associated with an increased risk of cannabis initiation during follow-up (AERR = 1.63; 95% CI = 1.27, 2.09, and AERR = 1.54; 95% CI = 1.25, 1.91, respectively).

Table 3.2 Parent monitoring at age 11 and initiation of cannabis use up to age 17, unadjusted models. Data come from 641 children sampled from 1983-1985 newborn discharge lists in southeast Michigan with complete information on all variables.

	n	% cannabis initiation 11 to 17 years	Estimated Relative Risk	95% Confidence Interval	p value
Parent Monitoring	641	34.6	0.95	(0.93, 0.97)	<0.001
Birth weight					
Low	364	33.8	0.95	(0.76, 1.17)	0.607
Normal	277	35.7	Ref		
Community					
Urban	332	34.3	0.98	(0.79, 1.22)	0.870
Suburban	309	35.0	Ref		
Race					
Black	291	32.3	0.88	(0.71, 1.10)	0.261
White	350	36.6	Ref		
Sex					
Male	296	41.6	1.45	(1.17, 1.79)	0.001
Female	345	28.7	Ref		
Smoked Tobacco by age 11					
Yes	64	62.5	1.98	(1.58, 2.48)	<0.001
No	577	31.5	Ref		
Drank alcohol by age 11					
Yes	64	53.1	1.63	(1.26, 2.11)	0.001
No	577	32.6	Ref		
Had a friend who smoked tobacco at age 11					
Yes	180	52.8	1.92	(1.56, 2.35)	<0.001
No	461	27.5	Ref		
Had a friend who drank alcohol at age 11					
Yes	516	49.6	1.60	(1.29, 1.99)	<0.001
No	125	31.0	Ref		
Maternal Education					
< High School	100	37.0	Ref		
High School	170	37.1	1.00	(0.73, 1.38)	0.992
Some College	246	33.7	0.91	(0.67, 1.24)	0.560
College	125	31.2	0.84	(0.59, 1.21)	0.360
Maternal Marital Status					
Single	208	42.3	1.37	(1.11, 1.69)	0.004
Married	433	30.9	Ref		
Maternal tobacco smoking at baseline					
Smoker	234	45.7	1.62	(1.31, 1.99)	<0.001
Non-smoker	407	28.3	Ref		

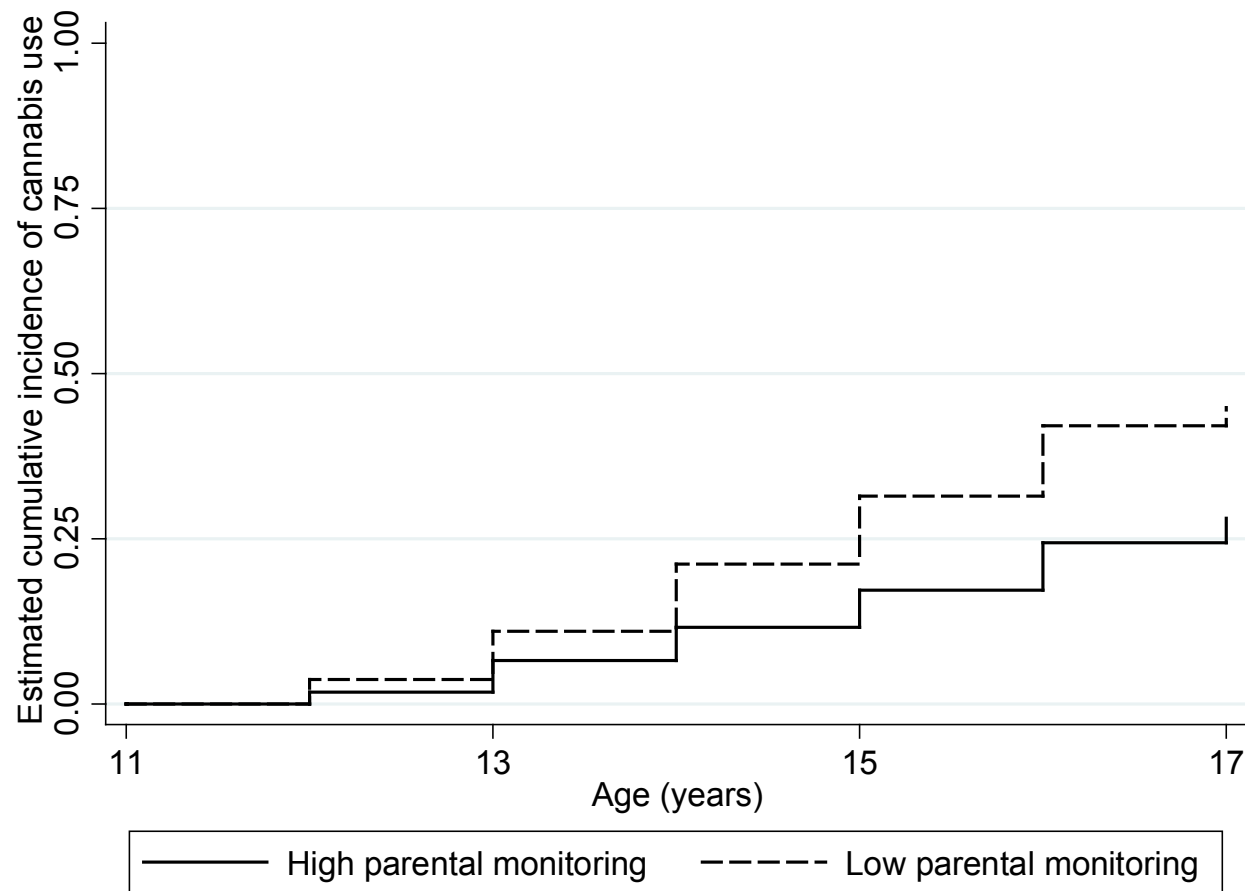
Table 3.3 Parent monitoring at age 11 and initiation of cannabis use up to age 17, adjusted model. Data come from 641 children sampled from 1983-1985 newborn discharge lists in southeast Michigan with complete information on all variables.

	Adjusted Estimated Relative Risk	95% Confidence Interval	p value
Parent Monitoring	0.96	0.94, 0.98	<0.001
Birth weight			
Low	0.85	(0.69, 1.04)	0.113
Normal	Ref		
Community			
Urban	1.01	(0.75, 1.37)	0.925
Suburban	Ref		
Race			
Black	0.64	(0.48, 0.87)	0.004
White	Ref		
Sex			
Male	1.25	(1.01, 1.56)	0.042
Female	Ref		
Smoked Tobacco by age 11			
Yes	1.31	(1.02, 1.69)	0.035
No	Ref		
Drank alcohol by age 11			
Yes	0.98	(0.73, 1.33)	0.899
No	Ref		
Had a friend who smoked tobacco at age 11			
Yes	1.63	(1.31, 2.03)	<0.001
No	Ref		
Had a friend who drank alcohol at age 11			
Yes	1.25	(0.99, 1.57)	0.061
No	Ref		
Maternal Education			
< High School	Ref		
High School	1.17	(0.85, 1.59)	0.338
Some College	1.06	(0.79, 1.44)	0.687
College	1.33	(0.91, 1.95)	0.144
Maternal Marital Status			
Single	1.63	(1.27, 2.09)	<0.001
Married	Ref		
Maternal tobacco smoking at baseline			
Smoker	1.54	(1.25, 1.91)	<0.001
Non-smoker	Ref		

3.3.3 Kaplan-Meier Survival Estimates

Figure 3.1 depicts estimates from the Kaplan-Meier analysis. The estimated cumulative incidence of cannabis use by age is displayed for the above- and below-median monitoring groups. As shown in the figure, the failure estimates for the lower parental monitoring group were consistently higher than those for the higher parental monitoring group. That is, by the end of each age interval adolescents in the lower parental monitoring group were more likely to have initiated cannabis smoking. By age 17, an estimated 45% of the individuals in the lower monitoring group had initiated cannabis use, compared with an estimated 28% in the higher monitoring group. The log-rank test revealed that the curves for the two parental monitoring groups were significantly different from one another ($p < 0.001$).

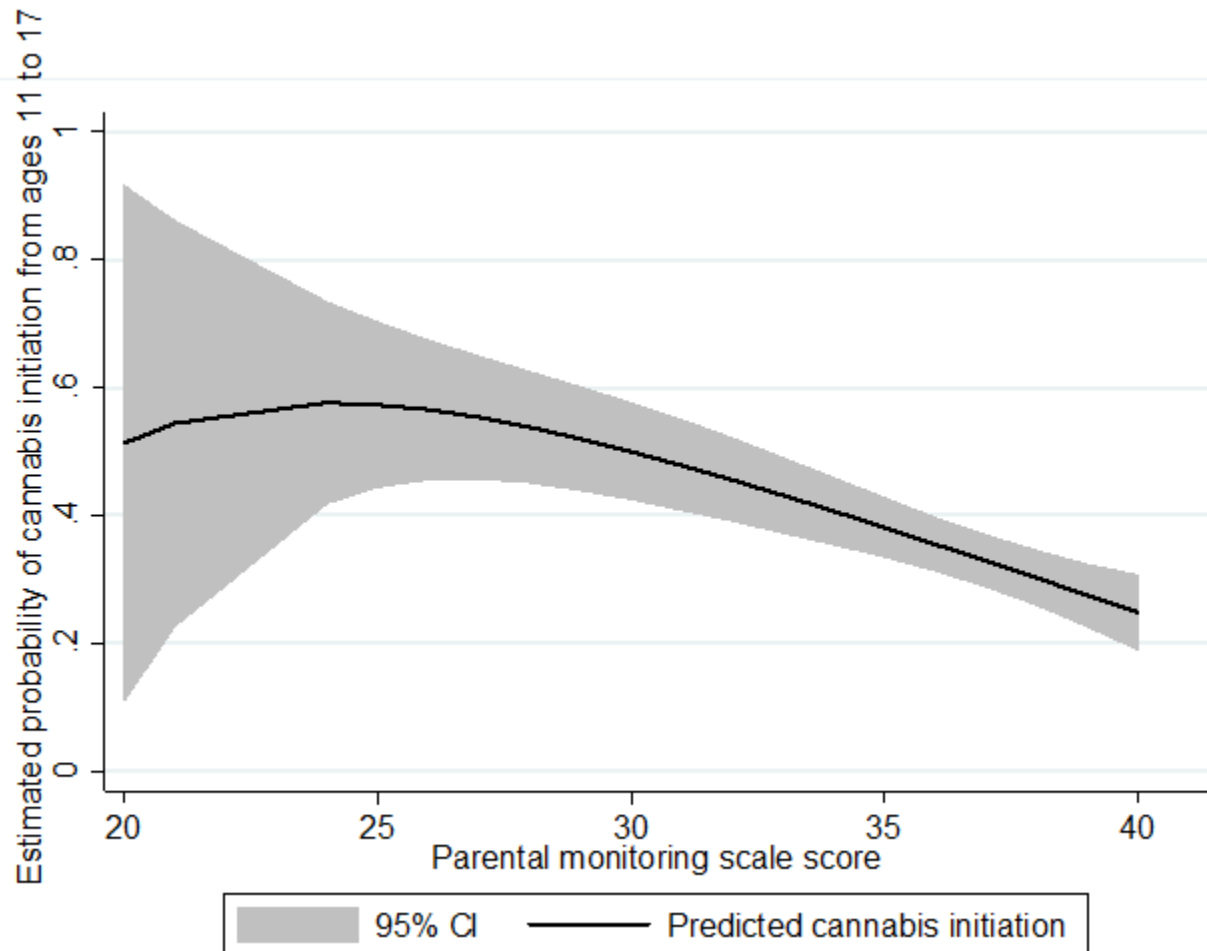
Figure 3.1 Kaplan Meier estimates of cumulative incidence of cannabis use from age 11 to 17 years. Data come from 619 children sampled from 1983-1985 newborn discharge lists from two hospitals in southeast Michigan.



3.3.4 Fractional Polynomial Post-estimation Examination of the Parental Monitoring – Cannabis Initiation Association

In post-estimation analyses, the method of plotting fractional polynomials was used to probe into the issue of non-linearity in the estimated association that links early parental monitoring with later cannabis initiation. Figure 3.2 depicts the fractional polynomial plot of the estimated probability of cannabis initiation from 11 to 17 years with 95% confidence intervals across levels of parental monitoring. With the exception of the very low end of the monitoring distribution, where sparse data created wide confidence bounds, there is a consistent linear decline in the probability of cannabis initiation for increasing scores on the parental monitoring scale.

Figure 3.2 Estimated probability of cannabis initiation from ages 11 to 17 years by level of parental monitoring. Data come from 641 children sampled from 1983-1985 newborn discharge lists in southeast Michigan with complete information on all variables.



3.4 Discussion

In this longitudinal study of a hypothesized predictive relationship linking parental monitoring levels at age 11 years with the cumulative incidence of cannabis use up to age 17, a primary finding was that children with higher levels of parental monitoring were less likely to initiate cannabis use across the age-span from 11 to 17 years. Specifically, for every one unit increase on the parent monitoring scale, there was an estimated four percent reduction in the likelihood of cannabis initiation, with statistical adjustment for other covariates. Children in the lower parental monitoring group at age 11 were more likely than children in the higher monitoring group to initiate cannabis use at each age interval from 11 to 17 years. Estimates from the fractional polynomial analysis suggest no attenuation of the relationship at the highest levels of parental monitoring. That is, parents at the highest parental monitoring level at age 11 years did not seem to induce a negative reaction and “acting out” in response to these higher parental monitoring levels.

Several strengths and selected limitations should be considered before a more detailed discussion of the findings. A major strength is the epidemiological frame for the original study sample. This was not a sample of delinquent or drug-involved youths; nor was it a sample of high-risk youths as often has been the case in research on family factors and parenting in relation to adolescent-onset drug use. Furthermore, the assessments of levels of parental monitoring were taken more than five years before the assessment of the cannabis initiation outcomes. This strength of the research design reduces a threat to validity of the study estimates in the form of inadvertent reciprocity

(i.e., the possibility that a child's expression of curiosity or interest in cannabis smoking might cause parents to increase their monitoring and supervision levels). It also reduces the threat of "shared methods covariation," as might be induced when the assessments of monitoring and outcomes are completed after a short-span follow-up (e.g., one year apart).

There are also several potential limitations to note. First, although the findings indicate a robust and stable association between early monitoring and the risk of cannabis initiation later on, an important question remains. Does early parental monitoring have a lasting influence regardless of its continuity into late adolescence? The apparent long term benefits of early parent monitoring might reflect stability of parenting behavior over time, as children mature, rather than an investment that pays off later on by deterring adolescents from involvement with cannabis. Future studies might be more illuminating if they included re-assessments of parental monitoring during follow-up intervals to test this question. Second, it is possible that sample attrition influenced the findings.

However, there were no differences on sample characteristics between the initial and follow-up samples to support this assertion. Third, due to the observational nature of this study and the assumption that no other potential explanatory variables were omitted, causal interpretations are not warranted at this time. Replications and studies that test the outcomes of interventions directed toward enhancing parental monitoring are needed.

The findings from the present study, focused on cannabis initiation in adolescence, are consistent with a large body of evidence linking lower levels of parental monitoring with increased odds of drug use (Chilcoat & Anthony, 1996; Chilcoat et al., 1995; DiClemente et al., 2001). With respect to the specific hypothesized link between

parental monitoring and cannabis use, the findings from the present investigation generally converge with the findings of the few published studies; however, there are some differences. For example, the findings from the present investigation are consistent with a cross-sectional study of adolescent females that found that teens with lower parent monitoring had an estimated two-fold increased odds of cannabis use (DiClemente et al., 2001). Similarly, in a longitudinal study of Seattle youths followed from the ages of 10 to 18 years, Kosterman and colleagues (2000) found that parents' proactive family management, a variable assessing parents' monitoring, rules, discipline and reward practices, was associated with reduced risk of cannabis initiation (Kosterman et al., 2000). A latent class analysis of cannabis use patterns in a sample of predominantly black middle-school students found that lower parental monitoring was associated with increased odds of membership in a latent class characterized by cannabis use and problems in sixth grade (Reboussin et al., 2007). However, this relationship was attenuated and non-significant by seventh and eighth grade. It is noteworthy that Reboussin and colleagues (2007) examined early cannabis involvement and did not cover the grades or ages when cannabis use is most likely to occur during adolescence. Similarly, using data from the National Survey of Parents and Youth, Tang and Orwin (2009) found parental monitoring signaled lower odds of cannabis initiation at ages 12 and 13 years, but not for ages 14 through 16 years (Tang & Orwin, 2009). Both studies suggested that other factors, namely peers, might have become more influential than parents in later years. In contrast, in the present study both parental monitoring and affiliation with tobacco using peers were important predictors of cannabis initiation, and

that the influence of parental monitoring on cannabis initiation was stable over the ages from 11 to 17 years.

In a previous report on this follow-up study the influence of parental monitoring on tobacco initiation varied with respect to race/ethnicity (Bohnert et al., 2009). Lower levels of parental monitoring were linked with higher odds of tobacco initiation only among white adolescents. In the present investigation, subgroup differences with respect to race/ethnicity on the parental monitoring - cannabis initiation relationship were not detected. The reasons for these differences between the two investigations are unclear, but reduced statistical power to detect sub-group variation might be at play. In both studies black adolescents had lower levels drug initiation; this finding is consistent with a number of empirical studies and results from national school-based surveys (Guo et al., 2002; Johnston et al., 2009).

Affiliation with drug using peers is one of the strongest and most consistent predictors of drug initiation in childhood and adolescence (Guo et al., 2002; van den Bree & Pickworth, 2005). Some have suggested that peers might be more influential than parents or more influential at older ages than parents with respect to cannabis smoking (Guo et al., 2002; Reboussin et al., 2007; Tang & Orwin, 2009; van den Bree & Pickworth, 2005). In the present study, early affiliation with peers who used tobacco was related to cannabis initiation. However, the inclusion of affiliation with tobacco and alcohol using peers as covariates did not appreciably dampen the association of parental monitoring with cannabis initiation in the adjusted model. Both were important predictors of cannabis initiation.

Consistent with evidence from prior studies, the present findings, from prospective research, help confirm one conceptual hypothesis, i.e., that parenting and familial characteristics might exert long-lasting influences on a child's risk of starting to use illegal drugs. The present study focused on the interval from late childhood to the end of adolescence, covering ages when cannabis initiation in the United States is most likely to occur. Trials aimed at strengthening parenting practices are underway and some of them show promise (Spoth et al., 2009). Future prevention research should continue to evaluate the effect of interventions aimed at promoting parent monitoring. Additional longitudinal studies will be necessary to help elucidate the possible paths from parental monitoring to drug use.

4. CHAPTER FOUR - MANUSCRIPT TWO

LONGITUDINAL STUDY OF LEVELS OF ADOLESCENT DRUG USE: AN SEM ANALYSIS OF PARENTAL MONITORING

ABSTRACT

Aims: To estimate the influence of level of parental monitoring (PM) assessed at age 11 on level of drug use at age 17, simultaneously testing paths through levels of drug use and affiliation with drug using peers (DUP) at age 11.

Setting: Data are from children born in 1983-1985, sampled and recruited in southeast Michigan.

Participants: Of the original cohort (n=823) children, 774 (87%) contributed data to the present study.

Measurements: Standardized interviews assessed drug use at ages 11 and 17, and PM and DUP at age 11. A structural equation model (SEM) was used to test paths of PM with DUP and drug use at ages 11 and 17.

Findings: According to the pre-specified SEM, the estimated level of drug use at age 11, as well as DUP, depended upon PM level, which also predicted drug use at age 17. DUP at age 11 did not predict drug use at age 17.

Conclusions: Level of PM may influence levels of drug use over a longer time span than previously believed (six years). This evidence helps substantiate the theory that parenting characteristics might exert long-lasting influences on a child's use of drugs.

4.1 Introduction

Alcohol, tobacco, and cannabis are three of the most widely used psychoactive drugs in the world (Degenhardt et al., 2008). In the United States, experimentation and use of these drugs is common, often occurring before the age of 18 years (Substance Abuse and Mental Health Service Administration, 2009). Drug use before adulthood, however, is associated with adverse psychiatric and social outcomes. For example, early-onset drug users might be more likely to experience problems related to their use, namely, DSM-IV drug abuse or dependence, compared with those who start using later in life (Chen et al., 2005; Grant et al., 2006; Winters & Lee, 2008). This association is not entirely explained by having greater elapsed time from onset of first drug use (Chen et al., 2005). Nonetheless, early-onset use might not be causally related to later drug problems, i.e., individuals prone to early use might also be more liable to experience drug problems. There is also evidence that links early-onset use with an excess risk for other later psychiatric disturbances (de Graaf et al.; Fergusson & Boden, 2008a; Tien & Anthony, 1990) and to poorer school performance and lower educational attainment (M. Lynskey & Hall, 2000); although the causal significance of these associations remains uncertain.

It follows that prevention or delay of the onset and use of alcohol, tobacco, and cannabis has salience in public health work. Most of the public health work along these lines has a focus on school and/or family. For example, Botvin and colleagues have conducted large-scale randomized trials in school-based settings (Botvin et al., 2000). In contrast, interventions developed on the basis of the social learning theory as applied to the family often are directed toward high risk family environments (e.g., (Kumpfer &

Alvarado, 2003)) or enhancement of positive parenting practices (e.g., (Dishion & Kavanagh, 2000)), such as the surveillance and monitoring conducted by parents during the childhood and adolescent years (i.e., parental monitoring (PM)). Implicit in these school and family oriented interventions is an assumed importance of affiliation with drug using or otherwise rule-breaking peers (i.e., deviant peers).

An important issue that has surfaced in the school intervention research involves the equivalent of “booster shots” in public health vaccination initiatives. Namely, it seems that many early primary prevention intervention effects fade as children mature through the school years. This fading of early intervention effects has motivated a line of research on “booster” drug prevention programs (Botvin et al., 1995). The issue of intervention program “fade” and the need for “booster” programs has been less well studied in the context of family centered studies, perhaps based upon an assumption that the effects of PM and other family-level parenting practices are inherently transient, and less important as the child progresses into middle and late adolescence.

An alternative to the “transient effects” assumption about PM and other positive parenting effects is a “persistent effects” hypothesis. A test of the “persistent effects” hypothesis requires: (a) evidence of an inverse association that links levels of PM with levels of drug use during the childhood years, and (b) evidence of an inverse association that links early PM levels with later levels of drug use (e.g., use in middle to late adolescence). In most of the prior research on PM, the assumption of “transient effects” has been made (e.g., Lloyd and Anthony, 2003). Here, the focus of inquiry is to test these alternative specifications for “transient” versus “persistent” effects of PM, as a guide toward future enhancement of parenting programs to prevent youthful drug use.

Clearly, if the “transient effect” assumption holds, then booster sessions for PM might be required. Of course, the idea of “booster” programs to help parents maintain adept levels of PM has some complex facets, in addition to logistical problems faced when interventionists have attempted to bring parents in for group prevention programs or to go out to parents in their homes.

To a lesser extent, these same kinds of processes might be taking place in the context of peer influences on drug use. Evidence is conflicting with respect to whether peer influence is a relatively short-lived phenomenon in childhood and adolescence (i.e., transient)(e.g., (Steinberg & Monahan, 2007)) or more persistent (e.g., (Guo et al., 2002)).

Hence, the aim of the present study is to use a representative cohort to evaluate the potential persistent influence of parental monitoring assessed at age 11 on levels of drug use at age 17, while simultaneously examining potential paths through levels of drug use and affiliation with drug using peers at age 11 years.

4.2 Methods

4.2.1 Data and Sample

Longitudinal data come from a 1983-1985 birth cohort of children from southeast Michigan. Complete information on the population, sampling procedures, and assessments is available elsewhere (Breslau et al., 1996), and is briefly summarized here. Low birth weight and normal birth weight children were randomly selected from 1983-

1985 newborn discharge lists of two hospitals in southeast Michigan, one serving a disadvantaged urban community and the other serving a middle-class suburban community. At the time of recruitment, 47 children with severe neurologic impairment were excluded from the initial sample. A total of 823 (75.2%) out of the 1095 children eligible for the study participated in the initial assessment from 1990 to 1992, when they were six years of age. Follow-up assessments were conducted when the children were 11 (n=717; 87.1%) and 17 years of age (n=713; 86.6%). Children were assessed via standardized face-to-face interviews. Of the original cohort of 823 children, 774 (94.0%) contributed data to the present investigation. The institutional review boards of the participating institutions approved the study.

4.2.2 Measures

4.2.2.1 Parental Monitoring

Parental monitoring (PM 11) was elicited from the children when they were 11 years of age using a 10-item scale (Appendix D) (Capaldi & Patterson, 1989; Chilcoat et al., 1995). The scale encompasses information on parental supervision and tracking of the child. A summary score was generated by reversing the coding and summing the scores. Scores ranged from a low of 10 to a high of 41, with higher scores indicating higher levels of monitoring.

4.2.2.2 Drug Use

Level of drug use at age 17 (Drug 17) was constructed as a continuous latent variable from the following three dichotomous (yes/no) child-reported questions on alcohol, tobacco cigarette smoking, and cannabis smoking, respectively:

- “Have you ever, even once, had a drink of any type of alcoholic beverage?
Do not include sips from another person’s drink.”
- “Have you ever smoked a cigarette, even one or two puffs?”
- “Have you ever, even once, used marijuana or hashish?”

Level of drug use at age 11 (Drug 11) was constructed as a continuous latent variable from the following child-reported age of onset questions which were coded into dichotomous used/never used variables for alcohol, tobacco cigarette smoking and cannabis smoking, respectively:

- “How old were you the first time you drank beer, wine, wine coolers, liquor, or any other drink with alcohol in it?”
- “How old were you when you first smoked a tobacco cigarette, even just a puff?”
- “How old were you the first time you smoked (marijuana/reefer)?”

4.2.2.3 Affiliation with Drug Using Peers

Level of affiliation with drug using peers at age 11 (DUP 11) was constructed as a continuous latent variable from the following three dichotomous (yes/no) child-reported questions on peer alcohol, tobacco cigarette smoking, and cannabis smoking, respectively:

- “Do you have any friends around your age who ever drink alcohol?”
- “Do you have any friends around your age who ever smoke tobacco cigarettes?”
- “Do you have any friends around your age who ever smoke (marijuana/reefer)?”

4.2.2.4 Baseline Covariates

The two stratification variables, birth weight and community, as well as sex, and maternal education were measured at baseline.

4.2.3 Statistical Analysis

Structural equation modeling (SEM) was used to simultaneously test paths of PM11 with DUP 11 and Drug 11 and 17. The final model was adjusted for the two stratification variables, birth weight and community, as well as, sex and level of maternal

education. All models were fit in Mplus 6.0 using the weighted least squares means and variance adjusted estimator (Muthén & Muthén, 1998-2010). The latent variable SEM approach provides several advantages over traditional regression techniques. First, it allows for simultaneous testing of multiple regression paths of complex relationships. Second, common sources of variation between outcome variables are taken into account and the path estimates are unencumbered by assumptions about error. Third, Mplus handles missing data using multiple imputation methods to use all available data (Muthén & Muthén, 1998-2010). Model fit was assessed via the Comparative Fit Index (CFI) (where good model fit is denoted by a score >0.90) (Bentler, 1990), the Tucker-Lewis Index (TLI) (where good model fit is denoted by a score >0.90) (Bentler & Bonett, 1980), and the Root Mean Square Error of Approximation (RMSEA) (where good model fit is denoted by a score <0.05) (Bentler, 1990).

4.3 Results

4.3.1 Comparison of the Original Sample with the Subset Used in the Analysis

Forty-nine individuals were excluded from the analytic sample because they did not have data on any of the outcomes in the structural equations model. Table 4.1 presents a comparison of the original sample at age 6 years ($n=823$) and the analytic sample used in the SEM ($n=774$). The two samples did not differ on any key sample characteristics ($p>0.05$).

Table 4.1 Description of the initial sample and the subset used in the structural equation model (SEM). Data come from 823 children sampled from 1983-1985 newborn discharge lists in southeast Michigan and assessed at ages 6, 11, and 17 years.

	Initial sample (n=823) %	Sample used in the SEM (n=774) %
Low birth weight	57.5	57.2
Urban	50.2	50.9
Male	48.6	48.1
Maternal Education		
Less Than High School	16.9	16.7
High School Graduate	27.5	26.9
Some College	37.3	37.6
College Graduate	18.4	18.9

4.3.2 Mean Levels of Parental Monitoring and Percentages of Drug Use in the Sample

As shown in Table 4.2, the mean score for parental monitoring was 36.1 (standard deviation = 4.0). With respect to affiliation with drug using peers, almost 30% of the children had a friend who smoked tobacco cigarettes at age 11 years, the highest percentage of affiliation among the three drugs (i.e., percentages of affiliation with alcohol and cannabis using peers were lower). Table 2 also depicts percentages of drug use at ages 11 and 17 years. The proportion of children using drugs at age 11 was relatively low. The percentage with a history of alcohol use was about 10%; nearly identical to the percentage for having a history of smoking tobacco cigarettes. Few children had smoked cannabis by age 11 years ($n=7$; 1%). By age 17, the prevalence of drug use was higher; an estimated 59% had a history of alcohol use; 45% had a history of tobacco cigarette smoking; 35% had a history of cannabis use.

Table 4.2 Mean level of parental monitoring and percentages of drug use in the sample. Data come from 774 children sampled from 1983-1985 newborn discharge lists in southeast Michigan and contributing data to the analyses.

	Age 11	Age 17
Parental Monitoring (Mean (SD))	36.1 (4.0)	---
Peer Alcohol Use (%)	19.6	---
Peer Tobacco Cigarette Smoking (%)	28.0	---
Peer Cannabis Smoking (%)	19.6	---
Ever Alcohol Use (%)	10.1	58.9
Ever Tobacco Cigarette Smoking (%)	10.6	44.7
Ever Cannabis Smoking (%)	1.0	34.7

4.3.3 Structural Equation Model for Parental Monitoring, Drug Use, and Peer Drug Use at Age 11 and Drug Use at Age 17

Figure 4.1 presents the SEM with standardized path estimates for the 774 cohort members, adjusted only for the stratification variables. All model fit indices were in the good to excellent range (CFI = 0.976; TLI = 0.968; RMSEA = 0.037). As shown in Figure 1, level of parental monitoring (PM 11), level of drug use (Drug 11), and level of affiliation with drug using peers (DUP 11) were all correlated at age 11 ($p < 0.05$). PM 11 was inversely related to both. Higher levels of PM 11 were associated with lower levels of Drug 11 and DUP 11. Drug 11 and DUP 11 were correlated ($p < 0.05$). Namely, higher levels of Drug 11 were associated with higher levels of DUP 11. PM 11 predicted levels of drug use at age 17 (Drug 17) ($p = 0.01$). The negative estimate indicates that higher levels of PM 11 were associated with lower levels of Drug 17. In addition, higher levels of Drug 11 were predictive of higher levels of drug use at age 17 years ($p < 0.04$). In contrast, the association between level of DUP 11 and level of Drug 17 was not statistically significant by conventional standards ($p < 0.30$).

Figure 4.1 Unadjusted SEM predicting levels of drug use at age 17 (Drug 17) from levels of drug use (Drug 11), parental monitoring (PM 11) and affiliation with drug using peers at age 11 (Drug11). Data come from 774 children sampled from 1983-1985 newborn discharge lists in southeast Michigan and contributing data to the analyses. (*p<0.05)

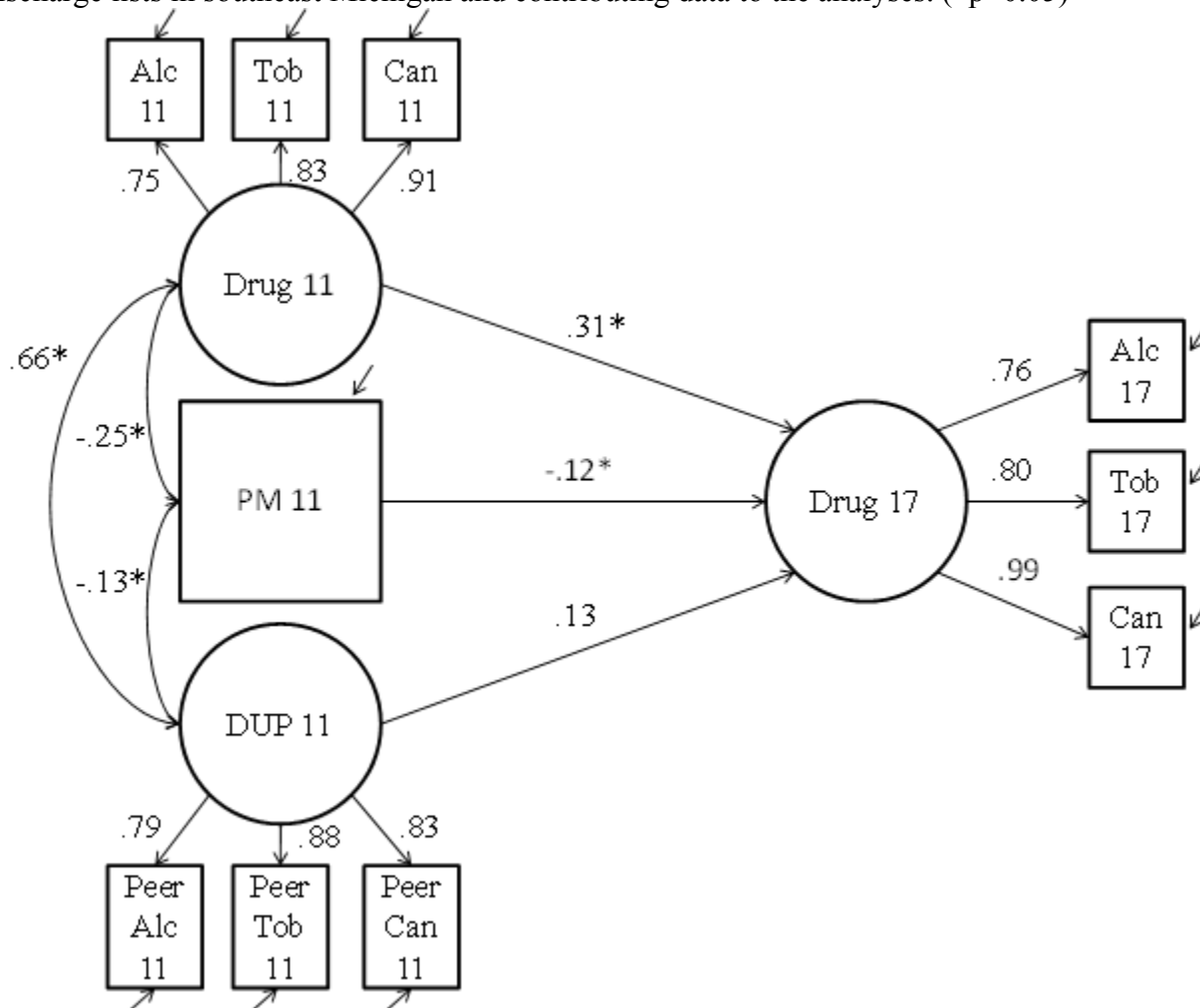
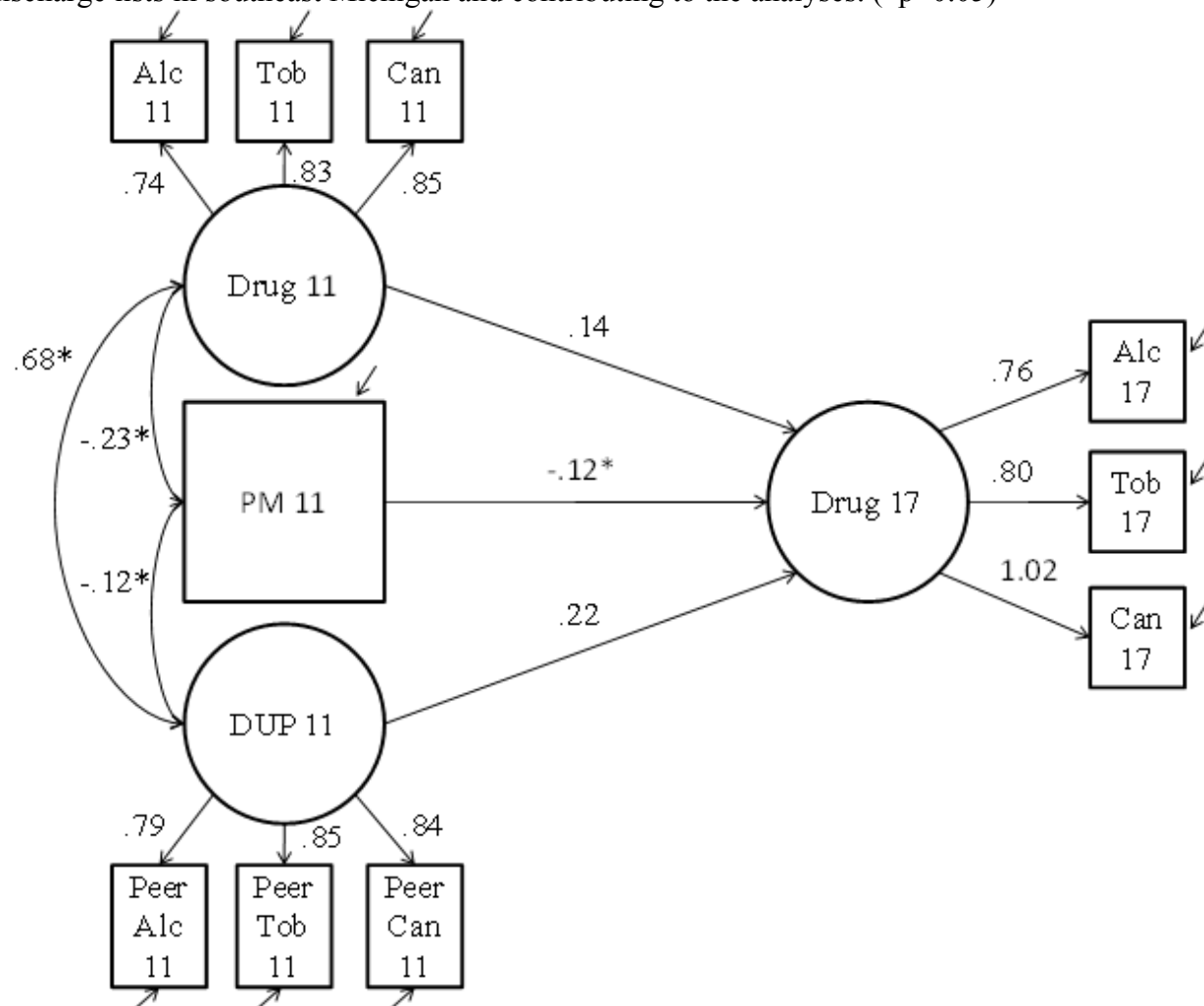


Figure 4.2 displays results of the SEM after adjustment for sex and maternal education. Model fit indices remained in the good to excellent range ($\chi^2 = 0.948$; TLI = 0.935; RMSEA = 0.040). Notably, only level of PM 11 was robustly associated with level of Drug 17 in the adjusted model ($p=0.01$). The association between level of Drug 11 and level of Drug 17 was no longer statistically significant after adjustment for sex ($p=0.31$).

Figure 4.2 Adjusted SEM predicting levels of drug use at age 17 (Drug 17) from levels of drug use (Drug 11), parental monitoring (PM 11) and affiliation with drug using peers at age 11 (Drug11). Data come from 774 children sampled from 1983-1985 newborn discharge lists in southeast Michigan and contributing to the analyses. (*p<0.05)



4.4 Discussion

Using a well-characterized 1983-1985 birth cohort, an estimate was derived for a suspected causal influence of level of parental monitoring assessed at age 11 on level of drug use at age 17, simultaneously probing other potential paths through levels of drug use and affiliation with drug using peers at age 11 years. The findings from this study can be summarized succinctly. First, at age 11 years, parental monitoring, level of affiliation with drug using peers, and level of drug use were all robustly correlated with one another. Second, parental monitoring at age 11 robustly predicted level of drug use at age 17 years. Namely, higher levels of monitoring predicted lower levels of drug use. Third, level of affiliation with drug using peers at age 11 did not predict level of drug use at age 17. These findings lend considerable support to the “persistence effects” hypothesis of parental monitoring on drug use; parental monitoring was related to drug use at childhood and at the end of adolescence.

Before a more detailed discussion of the findings, several limitations should be considered. Because parental monitoring and the questions used to construct the latent variables (level of drug use at age 11 and level of affiliation with drug using peers) were all assessed at age 11, the nature and direction of relationships between these correlated variables is uncertain. For example, it might be the case, as some have suggested (Lloyd & Anthony, 2003; Patterson et al., 1989), that parental monitoring reduces contact with drug using peers, in turn lowering a child’s chance to use drugs. Unfortunately, these data cannot disentangle the sequential processes of this relationship. In addition, because parental monitoring was assessed at a single time point (age 11 years), it is unknown as to

whether it was stable or changed throughout the follow-up interval. Nonetheless, parental monitoring at this one time point robustly predicted levels of drug use at age 17 years. Another limitation is that data on affiliation with drug using peers at ages other than 11 years are unavailable in this sample. To the extent that affiliation with friends who used drugs might have changed as the children matured, and whether and how these changes in friends might have influenced later drug use, cannot be assessed in this study. An additional limitation on causal inference is due to the observational nature of the data.

There is a large body of evidence documenting the role of parents and peers on child and adolescent drug use. With respect to parenting practices, parental monitoring is one key element that has been shown to be associated with child drug use. Specifically, findings from previous research have consistently documented that higher monitoring is associated with a lower risk of alcohol, tobacco, and other drug use in childhood and adolescence (Chilcoat & Anthony, 1996; Chilcoat et al., 1995; DiClemente et al., 2001). Using an SEM to simultaneously examine multiple influences of drug use in later adolescence, the finding that higher levels of parental monitoring at age 11 predicted lower levels of drug use at age 17 support and strengthen findings from prior research. Moreover, this finding complements other similar findings from investigations examining this association with common regression techniques, such as logistic regression. It also provides evidence consistent with a durable influence of parental monitoring through the ages when alcohol, tobacco, and cannabis use are most likely to occur. Collectively, the findings lend support for the persistence hypothesis. This potential long-term influence of parental monitoring could be due to two distinct possibilities which are not necessarily mutually-exclusive: 1) children that are highly monitored at earlier ages might internalize

or carry with them the benefits of adept monitoring throughout development; 2) parental monitoring is a persistent parental characteristic.

Previous studies have also reliably found that children who have drug using friends or affiliate with deviant or drug using peers are more likely to use alcohol, tobacco, and other drugs (Bauman & Ennett, 1996; Guo et al., 2002; van den Bree & Pickworth, 2005). With respect to adolescent drug use, some have suggested that peers might be more influential and more stable of an influence during adolescence (Guo et al., 2002; van den Bree & Pickworth, 2005). In contrast, there are findings that resistance to deviant peers is elevated in older adolescents. For example, Steinberg and Monahan (2007) reported that resistance to peers increased in a linear fashion between ages 14 and 18 (Steinberg & Monahan, 2007). The findings from the present investigation support this latter observation; i.e., the association between level of affiliation with drug using peers and level of drug use in childhood, and no association between level of affiliation with drug using peers in childhood and level of drug use in adolescence. There is also the possibility that the influence of the age 11 peers “faded” over time. This “fade” might be related to changes in peer group composition (i.e., peers at age 11 might not be the same peers at age 17). Similarly, peers at age 16 or 17 might be more influential with respect to drug use at age 17. Unfortunately, data on drug using peers at age 17 were unavailable.

Findings from the present study have potential implications for future research. Assessing parental monitoring, peer, and drug use pathways with more fine-grained longitudinal data (e.g., even more frequent than year-by-year follow-up assessments) would allow for the testing of hypothesized pathways from monitoring to affiliation with

drug using peers to later drug use. It would also make it possible for an examination of stability and change in parental monitoring and its potential influence on drug use. In addition, with respect to prevention efforts, booster sessions might not be needed if adept parental monitoring can be established in the childhood years. Nonetheless, this idea is speculative. The observed prediction will need to be replicated and tested in trials of preventions aimed at improving parental monitoring.

5. CHAPTER FIVE - MANUSCRIPT THREE

LONGITUDINAL PATHS FROM PARENTAL MONITORING TO CANNABIS USE IN CHILDHOOD AND ADOLESCENCE

ABSTRACT

Aims: To test the prospective association between parental monitoring (PM) and subsequent recently active cannabis smoking, and to examine the hypothesized mediational influence of deviant peer affiliation (DPA).

Design: A prospective longitudinal study completed within the context of a group-randomized trial in a sample of grade school students.

Setting: A mid-Atlantic United States urban public school system.

Participants: 2,311 children, with assessments yearly from 1989-1994.

Measurements: Logistic regression models with generalized estimating equations (GEE) were used to regress recently active cannabis smoking on the prior year's assessment of PM level, adjusting for other covariates. Structural equation modeling (SEM) of the paths was used to examine the potential mediating influence of DPA.

Findings: Higher levels of PM predicted lower odds of being a recently active cannabis smoker even after statistical adjustment for other covariates (adjusted odds ratio, AOR = 0.96; 95% confidence interval, CI = 0.92, 0.99). No appreciable mediation by level of DPA was detected for the paths linking previous level of PM with later occurrence of cannabis smoking.

Conclusions: The results from the present investigation help to shed new light on the potential protective influence of parenting practices on later drug use. The findings suggest that PM might help to prevent cannabis use throughout late childhood and early adolescence. In addition, the predictive association between PM and cannabis use was not appreciably mediated by level of DPA, in contradiction of a commonly held yet rarely tested hypothesis.

5.1 Introduction

Cannabis use in childhood and adolescence is a public health concern. Although alternative explanations have not been completely ruled out, mounting evidence links early-onset cannabis use with increased risk for later adverse health and social outcomes, including drug problems such as drug dependence and other psychiatric illnesses, as well as educational achievement (Chen et al., 2005; Fergusson & Boden, 2008a; M. Lynskey & Hall, 2000; Tien & Anthony, 1990; Wagner & Anthony, 2002a, 2002b). Therefore, in order to identify potential preventive targets and inform intervention development, it is important to gain a better understanding of the hypothesized paths that might lead to more early-onset cannabis use.

Patterson et al. (1989) have proposed one comprehensive model, a developmental progression for antisocial behavior, sometimes referred to as the social context model, to explain a mechanism leading to child and adolescent antisocial behavior, including early drug use (Patterson et al., 1989). The model, slightly refined by others based on empirical findings, posits a mechanism in which poor parental monitoring (PM), defined chiefly by tracking and knowledge of child whereabouts, leads to affiliation with deviant peers, in turn leading to subsequent antisocial behavior such as drug use. Several longitudinal studies have examined components of this model with respect to drug use. For example, Lloyd and Anthony (2003) found that higher levels of PM were associated with lower levels of affiliation with deviant peers across late childhood and early adolescence (Lloyd & Anthony, 2003). Other longitudinal studies have observed associations linking higher parental monitoring with reduced drug initiation (Chilcoat & Anthony, 1996; Chilcoat et al., 1995). No epidemiologic study to date, however, has

tested the main components of the hypothesized model simultaneously within a longitudinal context; i.e., simultaneously testing the hypothesized paths from PM to affiliation with deviant peers to cannabis use.

Other salient gaps in the literature remain in the hypothesized influence of PM on drug use. Notably, few studies have examined the specific suspected predictive association between PM and cannabis use. However, as previously stated, cannabis use in childhood and adolescence might be especially deleterious. Prior research has also focused predominantly on drug initiation and has not considered whether PM is associated with persistence of drug use or solely with initiation. Moreover, it is unknown whether PM continues to exert an influence over time, regardless of whether a child has initiated drug use in the past. In addition, previous investigations have been limited in their examination of the link between PM and drug use to a relatively short time span, usually no more than two or three years.

The present study uses a longitudinal study design and a well-characterized, epidemiologically-credible sample of children from a school system in the mid-Atlantic United States to help fill the existing gaps in the literature by testing the following aims. First, the goal is to estimate the suspected influence of earlier levels of PM on later cannabis use with data collected yearly over a six year study interval. Second, a test is conducted to determine whether the association is consistent over the study interval or whether it varies over time as children age. Third, direct paths from earlier levels of PM to later cannabis smoking and indirect paths from earlier levels of monitoring to subsequent levels of affiliation with deviant peers to later cannabis smoking are derived

and tested. That is, mediation by deviant peer affiliation (DPA) is tested in the hypothesized predictive relation between PM and cannabis use.

5.2 Methods

5.2.1 Study Population and Sample

The present study builds on an epidemiology and prevention research program initiated by Professors Sheppard Kellam, James C. Anthony, and their colleagues at the Prevention Research Center of the Johns Hopkins University Bloomberg School of Public Health. Detailed information on the program, research design and methods are available elsewhere (Kellam & Anthony, 1998; Kellam et al., 1991) and are briefly summarized here. The protocol for the research was reviewed and approved by the review board for protection of human subjects in research at the Johns Hopkins University Bloomberg School of Public Health. The data analysis protocol was also reviewed and approved by the review board at Michigan State University.

In brief, data come from a prospective longitudinal study completed within the context of a group-randomized trial of two interventions: a Good Behavior Game and a Mastery of Learning curriculum (Kellam, Brown et al., 2008; Kellam, Reid, & Balster, 2008; Kellam et al., 1991). The study population was designated to include all first graders entering 19 public elementary schools of a single urban school system located in the mid-Atlantic United States during two successive school years (cohort 1 entering in 1985 and cohort 2 entering in 1986), with some schools designated as intervention

schools and matched schools designated as external controls. Sub-sampling did not take place; efforts were made to recruit all incoming first-graders ($n=2,311$; cohort 1 = 1,196, cohort 2 = 1,115). Specifically, there were external school controls as well as within-school classroom controls. At the time of entry into first grade, children in each intervention school were randomly assigned to either an intervention classroom or an internal standard-setting classroom (control). Children entering external control schools were assigned at random to these first grade classrooms.

The 19 participating schools were located within the catchment area of five pre-identified urban areas of the school system. The composition of these areas encompassed very poor to middle class families who were mainly non-Hispanic black and white. Moreover, the 19 schools and city neighborhoods where the children were growing up were conceptualized as an ecological niche. Therefore, follow-up assessments were focused on the first-graders who remained in the same school system.

From 1985 through 1994, children were assessed via regular standardized face-to-face interviews with teachers. Starting at third grade (Spring of 1989), and continuing through Spring of 1994, there were standardized face-to-face interviews with the children themselves, which covered a variety of health and behavioral outcomes, including drug use. The present investigation focuses on the 1989-1994 child assessments, when the children matured from middle-late childhood through early adolescence.

The number of students interviewed varied each year by the maximum number of assessments allowed by the school during weeks in April-June, after school achievement testing. In the Spring of 1994, the investigators decided to focus resources on the second cohort of students who remained in the school system, and they only interviewed cohort

one student when cohort two students were not available. This explains the smaller numbers of interviewed participants in 1994.

5.2.2 Main Measures

The following main interview measurements were taken each Spring from 1989 to 1994.

5.2.2.1 Parental Monitoring, PM

Level of PM was assessed via a child-reported 10-item scale. As described elsewhere, the items were drawn from the Oregon Social Learning Center Parent Monitoring Scale and adapted for age-appropriateness (Capaldi & Patterson, 1989; Chilcoat et al., 1995; Lloyd & Anthony, 2003). The items in the scale encompass parental supervision, tracking and knowledge of child whereabouts (e.g., “How often, before you go out, do you tell your parents when you will be back?”). The complete scale is included in Appendix D. Possible scores ranged from a low of 10 to a high of 41.

5.2.2.2 Recently Active Cannabis Smoking, CAN

Recently active (past-year) cannabis smoking (CAN) was assessed via a single child-reported item: “When was the last time you smoked (marijuana)?”, with the local term for marijuana inserted within the parentheses, as described elsewhere (Wilcox,

Storr, Benoit, & Anthony, 2005). Children were coded in relation to a dichotomous variable; those who had smoked cannabis since Spring of the previous year (1) and those who had not (0).

5.2.2.3 Deviant Peer Affiliation, DPA

Level of DPA was assessed via a five-item scale. As previously described, the items were drawn from the Oregon Social Learning Center Peer Behavior Scale and adapted for age-appropriateness (Capaldi & Patterson, 1989; Chilcoat et al., 1995; Lloyd & Anthony, 2003). The five items assess friendships with children who participate in deviant behaviors such as cheating on tests and hitting others. The scale is included in Appendix E. Possible scores ranged from a low of five to a high of 25.

5.2.3 Other Covariates

Covariate values for sex, racial/ethnic minority status, and free/subsidized school lunch status at first grade were drawn from a centralized school database at baseline, as described elsewhere (Kellam & Anthony, 1998). Early aggression was measured upon entry into primary school via the standardized teacher interview assessment, the Teacher Observation of Classroom Adaptation-Revised (TOCA-R) (Lloyd & Anthony, 2003). Tobacco smoking in the year prior to CAN was evaluated each year from 1989 to 1993 via the following child-reported assessment question: “When was the last time you smoked tobacco?” For each year, a dichotomous variable was created to indicate those

who had smoked tobacco since the previous Spring (1) and those who had not (0).

Alcohol use in the year prior to CAN was evaluated each year from 1989 to 1993 in the same manner; i.e., a dichotomous variable constructed from the assessment question: “When was the last time you drank beer, wine or any other alcohol drink?” Cohort, intervention group, and classroom/school were all design variables determined at baseline. A dummy-coded indicator variable was created for each year of outcome assessment.

5.2.4 Statistical Analysis

First, in order to estimate the time-series relationship linking prior level of PM with the log-odds of CAN across the five outcome time points (i.e., 1990-1994), a logistic regression approach, applying generalized estimating equations (GEE) was used (Zeger, Liang, & Albert, 1988). Specifically, the log-odds of CAN was modeled as a function of prior level of PM (i.e., the data were arrayed so that each row was time-lagged; the log-odds of CAN for time point t was regressed on level of PM for time point $t-1$). Next, the model was elaborated to include potentially confounding covariates. These covariates included: sex, racial/ethnic minority status, free/subsidized school lunch status at first grade, teacher-rated aggression in first grade, prior recent tobacco and alcohol use (i.e., time-lagged, $t-1$), cohort, intervention group, and year of outcome assessment. A few children ($n=11$) tried cannabis before the time of initial PM assessment; a variable to indicate this history of cannabis smoking was also introduced as a covariate. Product-terms between the covariates and PM were also evaluated, but none qualified for entry at

the alpha level of 0.05. The GEE produces population-averaged estimates, taking into account interdependencies of repeated observations for the same subject over time (Zeger et al., 1988). For the present analysis, 1,448 individuals had available data for these GEE analyses, which were performed using Stata 11 (StataCorp, 2009).

Second, alternating logistic regression (ALR) was used to check the estimates obtained from the adjusted GEE model and examine the potential influence of clustering at the classroom/school level (Bobashev & Anthony, 2000). ALR is similar to GEE in that it takes into account interdependencies of correlated data and produces population averaged estimates. Unlike GEE, however, ALR produces a directly interpretable estimate of the amount of clustering in the data in the form of the pair-wise odds ratio (PWOR). Furthermore, ALR is capable of handling two levels of clustering; here, the individual and the classroom/school. The ALR model was fit using SAS 9.1 (SAS Institute, Cary, NC).

Third, structural equation modeling (SEM)/path analysis was used for simultaneous examination of prospective paths from level of PM to level of DPA and onward to CAN. Following the steps outlined by Baron and Kenny (1986), three separate models were estimated to examine mediation (Baron & Kenny, 1986). In the first model, paths from level of PM at each time t to CAN at each time $t+2$ were estimated to test for direct effects. The model also simultaneously adjusted for prior and subsequent assessments of PM and CAN. In the second step, paths from level of PM at each time t to level of DPA at each time $t+1$ were modeled to test for the predictive association linking prior PM, the initial variable, with subsequent DPA, the hypothesized mediator. In the second model, statistical adjustment was made for prior and subsequent

assessments of level of PM and level of DPA. In the comprehensive third model, effects were estimated for the paths from level of PM at time t to level of DPA at time $t+1$ to CAN at time $t+2$, as well as for the direct path from level of PM at t to CAN at $t+2$. The third model also simultaneously adjusted for prior and subsequent assessments of PM, DPA, and CAN. For all three models, SEM was implemented under maximum likelihood estimation with robust standard errors (MLR) using Monte Carlo integration with 1000 integration points in Mplus 6.0 (Muthén & Muthén, 1998-2010). The MLR SEM approach in Mplus uses multiple imputation methods that allow for the inclusion of subjects with incomplete data (Muthén & Muthén, 1998-2010). The SEM analyses were conducted for 1,302 individuals with available data. Mplus produced linear regression slopes for the meditational path when level of DPA was modeled as the outcome (i.e., Gaussian) and logistic regression slopes in the form of log-odds with corresponding odds ratios when CAN was the outcome (i.e., binary).

Fourth, for an additional check of mediation, main predictive paths that were statistically robust in all three models (i.e., where the paths were robust from PM to recent cannabis use, PM to DPA, and PM to DPA to recent cannabis use) were tested for mediation using the `binary_mediation` program with the `bootstrap` command with 500 replications in Stata 11 (StataCorp, 2009; UCLA). In accordance with Kenny's approach for testing mediation with dichotomous outcomes, the program standardizes the coefficients before using the product of coefficients method to compute indirect effects (Kenny, 2009; UCLA). This allows for the direct comparison of coefficients from one model to another in computing the direct and indirect effects (Kenny, 2009; UCLA). It should be noted, however, that some of the complexities of the data that are accounted for

in the SEM analyses (e.g., repeated measurements) are lost in the traditional regression framework of the binary_mediation program.

5.3 Results

5.3.1 Characteristics of the Sample

Table 5.1 provides a description of key study sample characteristics at each year of assessment. As shown in the table, at each time point there was a balance of approximately 50% males and 50% females. A majority of the sample was from a racial/ethnic minority subgroup (most of whom were African-American). As was previously stated in the methods, most of the adolescents assessed in 1994 were from cohort 2. The percentages of participants from each intervention group were similar each year (~60% standard; ~20% Good Behavior Game; ~20% Mastery Learning).

Table 5.1 Characteristics of the sample at each assessment 1989-1994. Data come from 2,311 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.

	Total Sample n=2,311		1989 n=1,530		1990 n=1,233		1991 n=1,543		1992 n=1,416		1993 n=1,251		1994 n=816	
Mean age range in years			8-9		9-10		10-11		11-12		12-13		13-14	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Sex														
Male	1,151	49.8	770	50.3	595	48.3	758	49.1	694	49.0	603	48.2	399	48.9
Female	1,160	50.2	760	49.7	638	51.7	785	50.9	722	51.0	648	51.8	417	51.1
Racial/ethnic minority status														
Nonminority	761	32.9	398	26.0	286	23.2	352	22.8	288	20.3	240	19.2	153	18.8
Minority	1,550	67.1	1,132	74.0	947	76.8	1,191	77.2	1,228	79.7	1,011	80.8	663	81.2
Cohort														
1	1,196	51.8	784	51.2	639	51.8	762	49.4	705	49.8	632	50.5	269	33.0
2	1,115	48.2	746	48.8	594	48.2	781	50.6	711	50.2	619	49.5	547	67.0
Intervention group														
Standard	1,339	57.9	883	57.7	688	55.8	892	57.8	816	57.6	712	56.9	477	58.5
Good behavior game	452	19.6	319	20.9	262	21.2	324	21.0	303	21.4	270	21.6	159	19.5
Mastery learning	520	22.5	328	21.4	283	23.0	327	21.2	297	21.0	269	21.5	180	22.0

5.3.2 Prevalence of CAN and Mean Scores for PM and DPA, 1989-1994

Table 5.2 depicts the prevalence (percentage) of CAN, the mean level of PM, and the mean level of DPA for each year of assessment. CAN prevalence proportions increased over the six years of assessment. Values were low for the first four years (~1%) and higher for the final two years of assessment (4.3% and 13.4%, respectively). The mean level of PM was relatively stable over the six assessment years, approximately 33 with a standard deviation of 5. The mean level of DPA was also relatively stable over time, ranging from 9.9 to 11.1.

Table 5.2 Percentages of recent cannabis use and means scores of parental monitoring and deviant peer affiliation for the assessments. Data come from a cohort of 2,311 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.

Assessment year	Recent (past-year) cannabis use			Parental monitoring			Deviant peer affiliation		
	n	%	SE	n	Mean	SD	n	Mean	SD
1989	1,506	0.7	0.2	1,435	32.2	5.2	1,513	11.1	3.9
1990	1,231	0.6	0.2	973	33.4	5.4	1,223	9.9	3.8
1991	1,541	1.2	0.3	1,361	33.2	5.0	1,536	10.4	3.9
1992	1,416	1.3	0.3	1,317	32.9	5.1	1,410	10.6	3.9
1993	1,247	4.3	0.6	1,163	32.8	5.1	1,251	10.9	3.9
1994	813	13.4	1.2	695	33.2	5.3	813	10.9	4.1

*The cannabis use value in 1989 is cumulative to the Spring of 1989; other estimates are prevalence proportions reflecting use since the prior Spring.

5.3.3 Results from the GEE Logistic Regression on the Hypothesized PM-CAN Link

Tables 5.3 and 5.4 present results from the crude and adjusted GEE logistic regression models, respectively. In the crude model, higher levels of PM predicted lower odds of CAN (odds ratio, OR = 0.93; 95% confidence interval, CI = 0.90, 0.96). Specifically, a one point increase on the PM scale signaled a seven percent decrease in the odds of CAN. After adjustment for covariates, the estimated effect of PM on CAN was slightly attenuated; however, it remained statistically robust (adjusted OR, AOR = 0.96; 95% CI = 0.92, 0.99). Because no statistical interactions were detected between PM and any of the other covariates, the null hypothesis that the common odds ratio for PM was uniform across subgroups could not be rejected. Notably, the predictive association between level of PM and CAN did not vary by year of assessment.

The covariate-adjusted GEE logistic regression yielded other findings of interest (Table 5.4). Males, children with higher levels of aggression in first grade, and prior tobacco smoking and alcohol use (i.e., one year prior to CAN) predicted increased odds of CAN. Cohort 2 children had lower odds of CAN than their older counterparts in cohort 1. Compared with the first year of assessment, there was an excess occurrence of CAN in 1993 and 1994.

Table 5.3 Results from the unadjusted GEE logistic regression linking prior parental monitoring with odds of recent cannabis use. Data come from 1,448 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.

	Odds Ratio	95% Confidence Interval	p value
Parental monitoring in the prior year	0.93	(0.90, 0.96)	<0.001
Sex			
Female	Ref	-	-
Male	2.52	(1.67, 3.80)	<0.001
Race/ethnic minority status			
Nonminority	Ref	-	-
Minority	1.29	(0.78, 2.16)	0.32
Eligible for subsidized lunch in first grade			
No	Ref	-	-
Yes	1.06	(0.71, 1.57)	0.785
First grade aggression	1.35	(1.16, 1.58)	<0.001
Cannabis use at 1989 assessment			
No	Ref	-	-
Yes	5.26	(1.59, 17.47)	0.007
Cigarette smoking in the prior year			
No	Ref	-	-
Yes	9.65	(6.27, 14.84)	<0.001
Alcohol use in the prior year			
No	Ref	-	-
Yes	4.62	(3.31, 6.46)	<0.001
Cohort			
1	Ref	-	-
2	0.74	(0.51, 1.07)	0.113
Year of assessment			
1990	Ref	-	-
1991	1.50	(0.59, 3.76)	0.393
1992	1.83	(0.76, 4.41)	0.179
1993	6.14	(2.96, 12.72)	<0.001
1994	23.72	(11.27, 49.92)	<0.001

Note: The analysis also adjusted for intervention group. Data are suppressed because another investigator is responsible for reporting on this variable.

Table 5.4 Results from the adjusted GEE logistic regression linking prior parental monitoring with odds of recent cannabis use. Data come from 1,448 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.

	Adjusted Odds Ratio	95% Confidence Interval	p value
Parental monitoring in the prior year	0.96	(0.92, 0.99)	0.026
Sex			
Female	-	-	-
Male	2.26	(1.45, 3.50)	<0.001
Race/ethnic minority status			
Nonminority	-	-	-
Minority	1.41	(0.82, 2.42)	0.211
Eligible for subsidized lunch in first grade			
No	-	-	-
Yes	1.05	(0.68, 1.62)	0.832
First grade aggression	1.28	(1.06, 1.55)	0.011
Cannabis use at 1989 assessment			
No	-	-	-
Yes	6.15	(1.99, 18.98)	0.002
Cigarette smoking in the prior year			
No	-	-	-
Yes	4.52	(2.48, 8.26)	<0.001
Alcohol use in the prior year			
No	-	-	-
Yes	2.07	(1.37, 3.15)	0.001
Cohort			
1	-	-	-
2	0.49	(0.33, 0.73)	0.001
Year of assessment			
1990	-	-	-
1991	1.07	(0.36, 3.22)	0.9
1992	1.39	(0.52, 3.73)	0.507
1993	4.63	(1.97, 10.90)	<0.001
1994	23.83	(10.04, 56.54)	<0.001

Note: The analysis also adjusted for intervention group.

Data are suppressed because another investigator is responsible for reporting on this variable.

5.3.4 Examining Clustering at the Individual and Classroom/school Levels Via the ALR

Because children were sampled with respect to classroom/school, the extent to which clustering would have an impact on the findings from the GEE logistic regression was evaluated using ALR. Findings from the adjusted ALR are shown in Table 5.5. The findings from ALR were nearly identical to the findings from the adjusted GEE logistic regression. The estimated influence of parental monitoring on recent cannabis use was the same ($OR = 0.96$; $95\% CI = 0.92, 0.99$). The pair-wise odds ratios (PWOR) depict the amount of clustering on the individual and classroom/school levels. As shown in the table, there was clustering on the individual level ($PWOR = 3.18$; $95\% CI = 1.42, 7.11$). Clustering at the classroom/school level was attenuated after individual-level clustering and other covariates were taken into account ($PWOR = 0.90$; $95\% CI = 0.77, 1.06$).

Table 5.5 Results from the ALR linking prior parental monitoring with odds of recent cannabis use and accounting for clustering. Data come from 1,448 participants originally recruited in 1985-1986 and followed-up from 1989-1994.

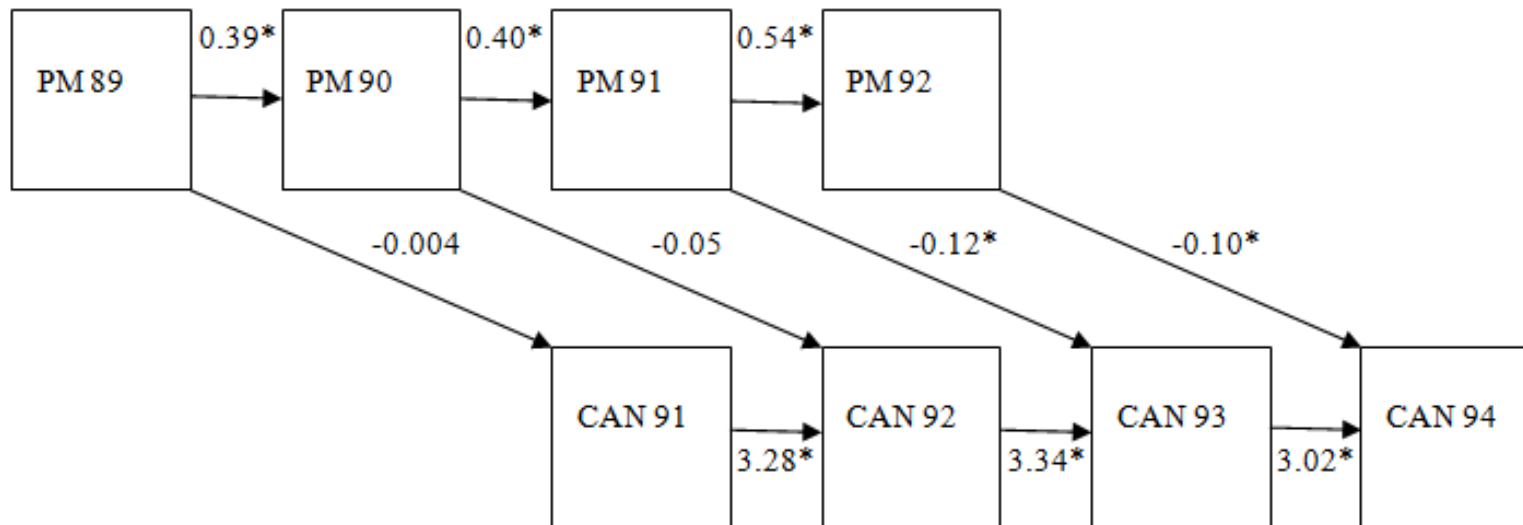
	Adjusted Odds	95% Confidence	
	Ratio	Interval	p value
Parental Monitoring	0.96	(0.92, 0.99)	0.015
Sex			
Female	Ref	-	-
Male	2.30	(1.55, 3.42)	<0.001
Race/ethnic minority status			
Nonminority	Ref	-	-
Minority	1.34	(0.79, 2.29)	0.278
Eligible for subsidized lunch in first grade			
No	Ref	-	-
Yes	1.07	(0.69, 1.68)	0.759
First grade aggression	1.27	(1.04, 1.56)	0.02
Cannabis use at 1989 assessment			
No	Ref	-	-
Yes	6.02	(1.93, 18.73)	0.002
Cigarette smoking in prior year			
No	Ref	-	-
Yes	4.22	(2.36, 7.63)	<0.001
Alcohol use in prior year			
No	Ref	-	-
Yes	2.04	(1.40, 2.97)	<0.001
Cohort			
1	Ref	-	-
2	0.49	(0.33, 0.71)	<0.001
Year of assessment			
1990	Ref	-	-
1991	1.15	(0.40, 3.27)	0.796
1992	1.45	(0.53, 3.95)	0.467
1993	4.86	(1.96, 12.05)	0.001
1994	26.31	(1.55, 11.09)	<0.001
	Pair-wise		
Clustering	Odds Ratio	95% CI	p value
Individual	3.18	(1.42, 7.11)	0.005
CSS	0.90	(0.77, 1.06)	0.227

Note: Intervention group was also adjusted for but data are suppressed because another investigator is responsible for reporting on this variable.

5.3.5 Testing Hypothesized Mediation through DPA

Figure 5.1 depicts the SEM linking prior level of PM with subsequent CAN (two years later). Specifically, the figure depicts the estimated lagged influence of level of PM on CAN (i.e., the probability of CAN at each time $t+2$ regressed on level of PM at time t) taking into account the other paths in the model. Paths from level of PM to CAN indicated that higher monitoring was associated with lower odds of CAN at each assessment; however, only the final two paths with CAN in 1993 (CAN93) and 1994 (CAN94) as the outcomes were robust (for CAN93: coefficient = -0.12, OR = 0.88, $p < 0.05$; for CAN94: coefficient = -0.10, OR = 0.90, $p < 0.05$).

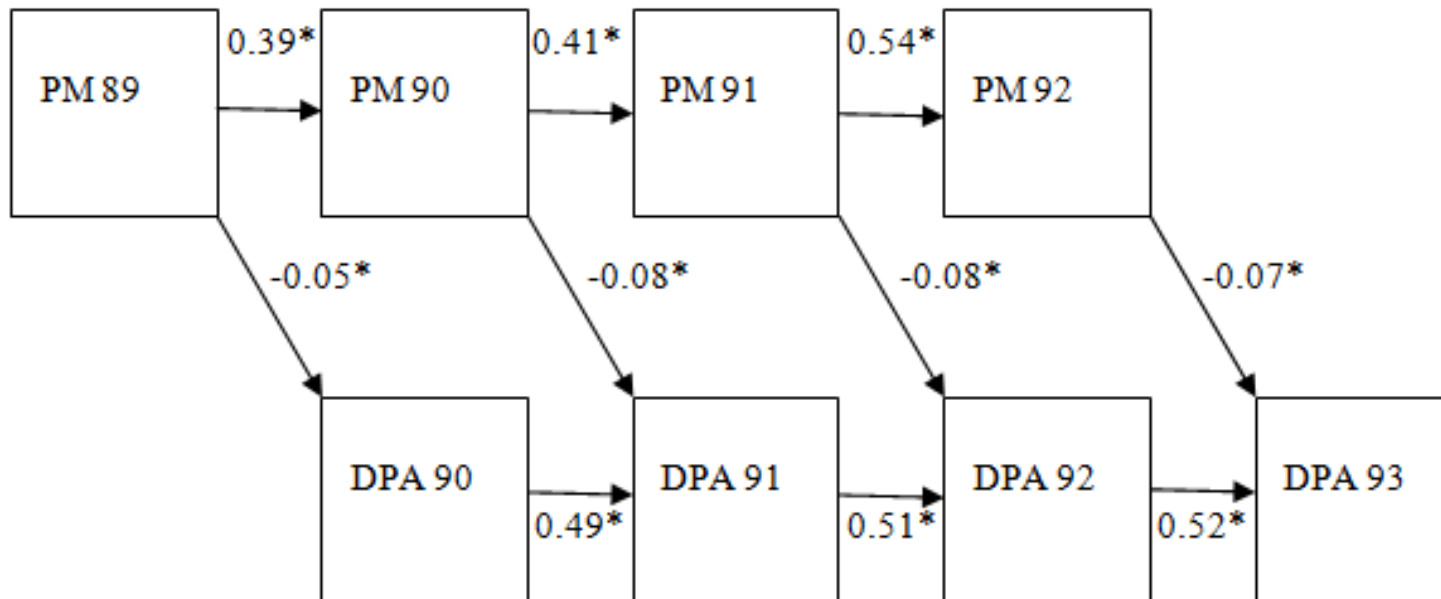
Figure 5.1 SEM estimates linking prior level of parental monitoring (PM) with recent cannabis use (CAN). Data come from 1,302 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.



* $p < 0.05$

Figure 5.2 depicts the SEM evaluation of hypothesized paths from prior level of PM to subsequent level of DPA. The figure shows a robust predictive association for each path linking prior level of PM with subsequent level of DPA (coefficients ranging from -0.05 to -0.08; all $p < 0.05$). For each path, higher levels of PM were associated with lower levels of DPA.

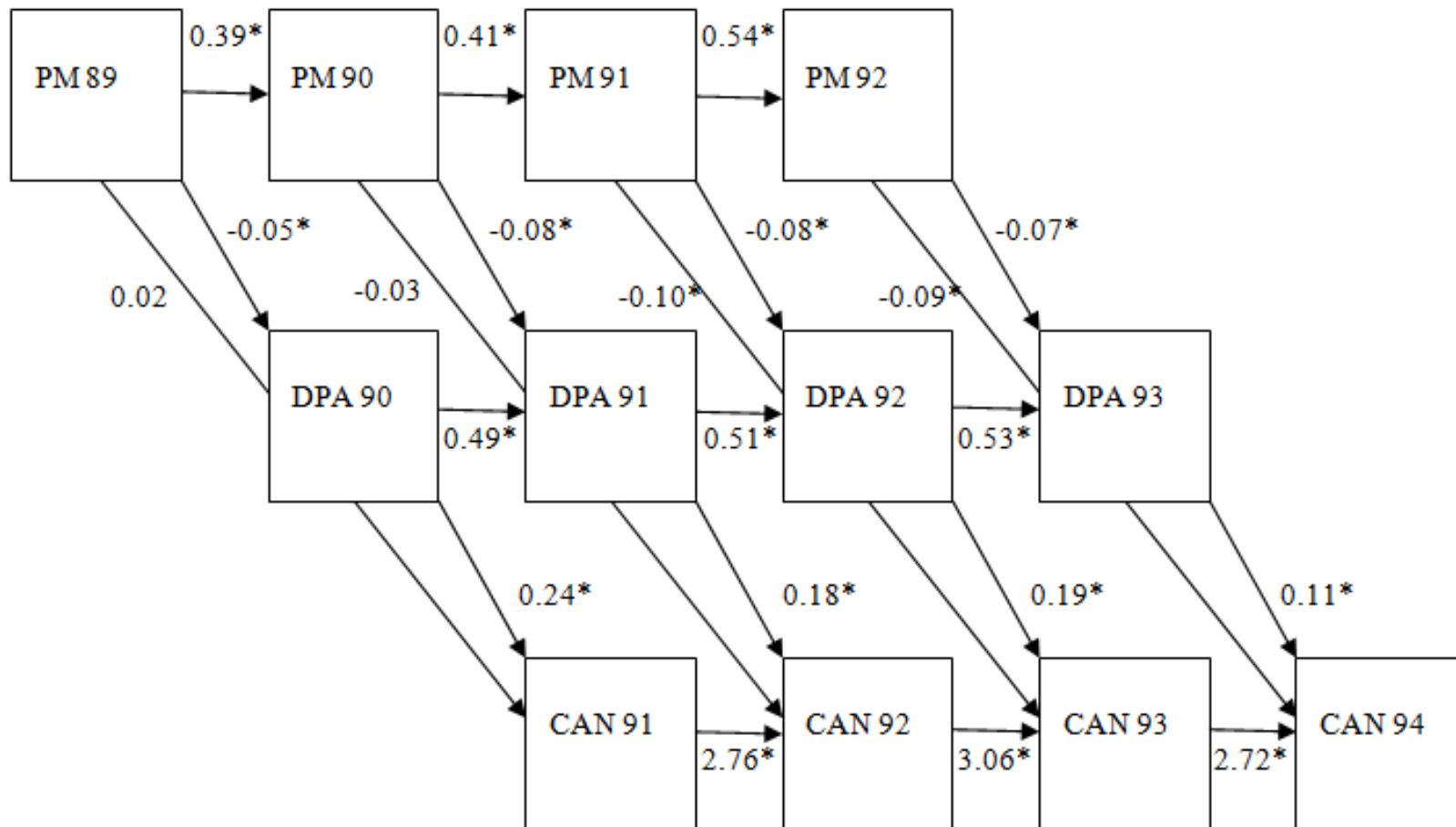
Figure 5.2 SEM estimates linking prior level of parental monitoring (PM) with level of deviant peer affiliation (DPA). Data come from 1,302 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.



* $p < 0.05$

Figure 5.3 depicts the model extended to examine potential mediation via level of DPA (i.e., paths from level of PM at t to level of DPA at $t+1$ to CAN at $t+2$). For each sequence of the main paths of interest, level of PM was associated with level of DPA ($p < 0.05$), which was associated with subsequent CAN ($p < 0.05$). Direct predictive paths from level of PM to CAN remained inverse and robust for the final two paths (i.e., the paths with CAN93 and CAN94 as the outcomes). Taken together with the findings from the two previous figures, the results from Figure 3 provide evidence for slight mediation by level of DPA in the association between level of PM and later CAN for the two final main regression paths of interest (i.e., with CAN93 and CAN94 as the outcomes). Nonetheless, the mediation is not appreciable. This is evidenced by the lack of sizeable attenuation in the main direct predictive regression paths, from PM in 1991 (PM91) to CAN93 and from PM in 1992 (PM92) to CAN94, between Figure 1 and Figure 3. For example, the coefficient and corresponding OR for the path from PM92 to CAN94 was -0.10 and 0.90, respectively, in Figure 1, which was not appreciably distorted with the addition of the suspected mediator, DPA in 1993, in Figure 3 (coefficient = -0.09, OR = 0.92).

Figure 5.3 SEM estimates testing paths from level of parental monitoring (PM) to level of deviant peer affiliation (DPA) to recent cannabis use (CAN). Data come from 1,302 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.



* $p < 0.05$

Additional tests for mediation were then estimated for the two main paths that met criteria for mediation in the SEMs (i.e., the path from PM91 to DPA92 to CAN93 and the path from PM92 to DPA93 to CAN94). The total estimated indirect and direct effects are shown in Table 5.6 for each main path. As shown in the table, none of the confidence intervals entrap the null; therefore, all of the estimated effects are statistically robust. The table also shows the estimated proportion of total effect mediated for each path. Both are minimal, 0.31 for the path with CAN93 as the outcome and 0.17 for the path with CAN94 as the outcome, indicating no appreciable mediation via level of DPA. That is, level of DPA explained a very small proportion of the predictive association between level of PM and recent cannabis use.

Table 5.6 Formal tests of mediation for the two main paths of interest that were robust in the SEM. Data come from a cohort of 2,311 first graders enrolled in a mid-Atlantic public school system and assessed yearly from 1989 through 1994.

Estimated path from PM91 to DPA92 to CAN93		
	Estimate	Bias Corrected 95% Confidence Interval
Indirect effect	-0.07	(-0.11, -0.05)
Total indirect effect	-0.07	(-0.11, -0.05)
Direct effect	-0.16	(-0.29, -0.002)
Total effect	-0.23	(-0.35, -0.08)
Estimated proportion of total effect mediated = 0.31		
Estimated path from PM92 to DPA93 to CAN94		
	Estimate	Bias Corrected 95% Confidence Interval
Indirect effect	-0.05	(-0.08, -0.02)
Total indirect effect	-0.05	(-0.08, -0.02)
Direct effect	-0.23	(-0.34, -0.11)
Total effect	-0.27	(-0.39, -0.16)
Estimated proportion of total effect mediated = 0.17		

5.4 Discussion

The findings from the present prospective study strengthen the evidence base for the potential preventive influence of adept PM on later drug involvement in important and novel ways. First, a robust predictive association linking higher levels of PM with decreased odds of recent cannabis use was found. Specifically, every one point increase on the monitoring scale signaled a four percent decrease in the odds of recent cannabis use, even after statistical adjustment for other covariates. Second, variation by year of assessment was not detected, indicating that the association between level of PM and later cannabis use was relatively stable as the children aged. Third, in a test of the main components of the model of developmental progression for antisocial behavior, or social context theory, no appreciable mediation of the PM-CAN relationship by level DPA was detected.

The findings from the present investigation are consistent with other longitudinal studies that have observed a lower risk of drug use with increased levels of PM (Chilcoat & Anthony, 1996; Chilcoat et al., 1995). Nonetheless, most of the prior evidence has been limited to illegal drug initiation in general, and has not focused on cannabis smoking in specific. The findings from the present investigation are consistent with the few studies that have examined the relationship between PM and cannabis smoking in a longitudinal context. For example, a prior prospective study conducted in Australia found that poor PM was one of four predictors of adolescent drug use, including cannabis (Hayatbakhsh et al., 2008). Similarly, in a population-based sample of adolescents in the United States, the National Survey of Parents and Youth (NSPY), low PM was associated with cannabis use (Martins et al., 2008). The results from the present study strengthen

and extend the findings from this prior work, offering the advantage of testing the relationship between prior levels of PM and subsequent occurrence of CAN over multiple yearly assessments. The results from the present investigation suggest that PM might help to shield youths from smoking cannabis during the interval of late childhood to early adolescence, and that the estimated influence of monitoring is stable over these years, even as the incidence of CAN rises sharply during adolescence.

This is the first study of cannabis smoking that has examined the main processes presented by Patterson et al. (1989) in the model of developmental progression for antisocial behavior, sometimes called the social context theory. Prior evidence has examined components of the theory (Patterson et al., 1989). Namely, Lloyd and Anthony (2003) observed a robust association linking prior levels of PM with subsequent levels of DPA (Lloyd & Anthony, 2003). Other studies have documented the association between deviant peers and drug use (Kaplan, Martin, & Robbins, 1984) and PM and cannabis and other drug use (Chilcoat & Anthony, 1996). No prior study has tested these paths simultaneously with respect to cannabis smoking. At earlier ages, when CAN was less common, the estimated direct effect for level of PM on the odds of CAN was not statistically significant by conventional standards (i.e., at $p < 0.05$). This finding might be a result of low power to detect an association, as there were very few cannabis smokers at earlier ages. Nonetheless, at later ages, when CAN became more common, level of DPA did not appreciably mediate the predictive paths from level of PM to CAN. That is, the direct paths from level of PM to subsequent CAN were not markedly attenuated when level of DPA was included in the SEM. Therefore, the commonly held and argued belief that the primary manner in which PM prevents drug use is by thwarting relationships with

deviant peers is not wholly supported by the evidence of this study. This finding is foreshadowed in two previous studies on PM and tobacco initiation and PM and cannabis initiation in a southeast Michigan cohort (Bohnert, Anthony, & Breslau; Bohnert et al., 2009). In the two previous studies, having a tobacco using peer, a measure related to DPA, was an important predictor of initiation; however, it did not appreciably attenuate the PM-drug initiation link.

The possible implications of these observational findings should be considered in light of the following potential limitations. First, causal interpretations are limited due to the observational and self-reported nature of the data. Second, in the GEE analysis statistical adjustment was made for a broad array of covariates; however, there remains the possibility that some unmeasured variable related to both level of PM and cannabis smoking might account for the observed association. Third, in the logistic GEE analysis it is possible that the attempt to statistically control for a host of covariates might have led to over-adjustment. This especially might be the case with respect to prior tobacco and alcohol use (i.e., before cannabis smoking). These variables might be mediators on a PM-influenced path to CAN and adjustment might not have been warranted. Fourth, due to complexity and sample size constraints, we were not able to adjust for other covariates in the SEM.

It is important to note that PM is only one component in a constellation of parenting behaviors. Nonetheless, the cumulative evidence suggests that PM is a vital component. The results from the present investigation help to shed new light on the potential protective influence of parenting practices on cannabis smoking that starts before mid-adolescence. The findings suggest that PM might help to prevent cannabis

smoking at every age in late childhood and early adolescence. In addition, contrary to a conventionally held hypothesis, evidence from the present study suggests that the predictive association linking PM with CAN is not appreciably mediated by level of DPA. Replication of these findings in other settings and samples is necessary. The potential influence of PM evaluated in relation to other facets of parenting is also needed. Longitudinal studies with more finely-grained assessments might help to elucidate other complex and varied mechanisms along the hypothesized path from PM to drug use and beyond, perhaps testing the influence of PM on other later adverse health and social outcomes that might be consequences of early cannabis smoking (Fergusson & Boden, 2008a, 2008b; D. B. Kandel, 1984; M. Lynskey & Hall, 2000; Tien & Anthony, 1990). The length of the potential influence of PM remains unknown; i.e., when monitoring and its potential influence effectively end. Population-based trials aimed at teaching parents how to adeptly monitor their children are also needed (Spoth et al., 2009).

6. CHAPTER SIX

FINAL CONCLUSIONS

6.1 Summary of Findings

The study in Chapter Three estimated the predictive relationship linking parental monitoring, as reported by children at age 11, with the cumulative incidence of cannabis use up to age 17 years. Children with higher levels of parental monitoring were less likely to initiate cannabis use from the ages of 11 to 17 years. Specifically, for every unit increase on the parental monitoring scale there was a 4% reduction in the risk of cannabis initiation, even after statistical adjustment for other covariates. In the study presented in Chapter Three, children in the low monitoring group were consistently more likely than children in the high monitoring group to initiate cannabis use at each age interval from 11 to 17 years.

The investigation in Chapter Four estimated the influence of level of parental monitoring assessed at age 11 on level of drug use at age 17, simultaneously probing potential paths through levels of drug use and affiliation with drug using peers at age 11. The study found that level of parental monitoring, level of affiliation with drug using peers, and level of drug use were all robustly correlated with one another at age 11 years. Parental monitoring at age 11 predicted level of drug use at age 17; namely, higher levels of monitoring predicted lower levels of drug use. In contrast, level of affiliation with drug using peers at age 11 years did not robustly predict level of drug use at age 17 years.

In Chapter Five, a GEE logistic regression was used to estimate the association between prior levels of parental monitoring and subsequent recently active cannabis smoking. The findings indicated that for every one point increase on the monitoring scale, there was a 4% decrease in the odds of recently active cannabis use, even after statistical adjustment for other covariates. A statistical interaction between level of parental monitoring and year of assessment was not detected, indicating that the association between level of parental monitoring and odds of later cannabis use might not vary across the study interval (i.e., from middle to late childhood through early adolescence). In Chapter Five, potential mediation of the parental monitoring-cannabis use path by level of affiliation with deviant peers was tested (i.e., the model of developmental progression of antisocial behavior was tested). Although the results were statistically significant, mediation by level of affiliation with deviant peers was minimal and not appreciable.

6.2 Limitations

In both longitudinal samples used for the dissertation research, drug use was assessed via standardized, self-reported items, which are subject to biases (e.g., recall and reporting). Nevertheless, empirical evidence suggests that self-reported drug use data is relatively accurate, especially in epidemiologic settings. For example, although Harrison (1995) found evidence for a small underreporting bias in the National Household Survey on Drug Abuse and the Monitoring the Future Survey, the author suggested that the overall effects of the bias are small (Harrison, 1995). Nonetheless, it should be noted that

in other settings, such as the juvenile justice system, self-reported measures may be less reliable (Magura & Kang, 1996).

Similarly, parental monitoring was collected via child self-report, which could have influenced the results. For example, children who use cannabis might report that they are more highly monitored than those who do not use cannabis. The studies in the present dissertation did try to overcome this limitation. For example, the possibility of this circumstance occurring in the first manuscript was reduced via the exclusion of children who had previously initiated cannabis use from the analysis. In the third study, multiple measurements of parental monitoring and cannabis use were used, which might have improved reliability. The best possible measurement for parental monitoring might come from an independent observer (e.g., a fieldworker observing and recording monitoring in the participants' homes); however, the burden on participants, the length of time a fieldworker would have to observe a given home, and the expense are prohibitive. Nonetheless, future studies might benefit from using multiple informants of parental monitoring.

In the present dissertation studies, there is the possibility that sample attrition influenced the findings. Evidence for this prospect was not supported; there were no differences on demographic characteristics between the full sample who participated in baseline assessments and those from the sub-sample who participated in follow-up assessments, for either of the two cohort samples used.

The data used for the studies on parental monitoring and cannabis use were observational in nature; therefore, causal interpretations are cautioned. Randomized trials aimed at increasing parental monitoring are needed. Nonetheless, with respect to

guidelines for epidemiologic causal criteria, the results do show a modest association, temporality, a dose-response relationship, the findings have been replicated, and there is plausibility and consistency.

6.3 Future Directions

The findings from the present dissertation research have potential implications for future research. First, the duration of the potential influence of adept parental monitoring on drug use is unknown. In Chapter Three and Chapter Four, higher parental monitoring in childhood predicted lower occurrence of drug initiation and use into late adolescence. Dishion and McMahon (1998) have hypothesized that the influence of parental monitoring might extend to even later developmental intervals and older ages, namely, young adulthood (Dishion & McMahon, 1998). However, empirical tests of that hypothesis have not been conducted. Second, there has been less investigation on the potential impact of parental monitoring on the possible sequelae of early cannabis and other drug use. For example, it might be of interest to test if parental monitoring relates to academic outcomes through early drug use. Third, the finding in Chapter Five of no appreciable mediation by level of deviant peer affiliation needs to be replicated in other studies. If the findings are replicated in other samples, other potential mechanisms in the parental monitoring-drug use association will need to be explored. Fourth, new evidence is emerging with respect to the complex interrelationships between parental monitoring, genetic predisposition, and other environmental influences on externalizing and smoking behaviors (Dick et al., 2009; Dick et al., 2007). For example, using a twin sample, Dick

and colleagues (2007) observed that environmental influences were more important for adolescent smoking when children were highly monitored but genetic influences accounted for greater explanatory power at low parental monitoring levels (Dick et al., 2007). These findings will need to be replicated in other diverse settings. It is unknown if this observation might extend to other drugs or drug use disorders. Moreover, as new candidate genes for drug use and dependence emerge, it will be necessary to test for potential interactions with environmental influences, such as parental monitoring. Fifth, longitudinal studies with more finely-grained assessments might help to elucidate other complex and varied mechanisms along the paths from parental monitoring to drug use, and could help explain the mechanisms underlying the association between parental monitoring and drug use. Sixth, with the exception of a few recent noteworthy studies (e.g. (Spoth et al., 2009)), there is a dearth of epidemiologic intervention trials aimed at improving parenting with the goal of preventing early drug use. Most of the work to this point has been in schools; however, new trials might aim at improving parental monitoring in other study settings, e.g., community settings. These trials should also test outcomes on suspected paths to early drug use, such as affiliation with deviant peers, and possibly from early drug use to other potential consequences of early use. Seventh, most investigations of the potential protective influence of parental monitoring have been conducted in the United States. It is unknown if the generally observed associations in the US-based research would hold in other countries.

6.4 Conclusions

Parental monitoring is one component in a constellation of parenting behaviors. Nevertheless, the cumulative evidence suggests that parental monitoring is a vital parenting element, and might shield youths from a host of delinquent behavior, including cannabis and other drug use. Consistent with evidence from prior studies, the findings from the present research help confirm the theory that parenting and familial characteristics might exert long-lasting influences on a child's risk of initiating and using illegal drugs. The influence of parental monitoring was durable over the interval from childhood to the end of adolescence, covering the ages when cannabis initiation and use is most likely to occur. No appreciable mediation of the parental monitoring-cannabis use association was detected by level of deviant peer affiliation; i.e., the direct effect from parental monitoring to cannabis use remained relatively unchanged in the mediational models that included a path through deviant peer affiliation. If future prevention trials aimed at improving parental monitoring show efficacy, the interventions used in the trials might be relatively inexpensive, mass-action methods for reducing drug initiation and use before adulthood.

APPENDICIES

APPENDIX A

Technical Appendix for Chapter Three

In this study the research design is a prospective cohort with three waves of assessment. The study population was designated to include low birth weight (LBW) and normal birth weight (NBW) newborns from southeast Michigan. The sampling approach involved randomly selecting LBW and NBW children from 1983-1985 newborn discharge lists of two hospitals in southeast Michigan, one serving an inner-city community and the other serving a suburban community. Forty-seven children with severe neurologic impairments were excluded from the initial sample. The resulting sample consisted of 1095 children. Of the 1095 children eligible for the study, 823 (75.2%) participated in the initial assessment from 1990 to 1992, when they were six years of age. Follow-up assessments were conducted at 11 years ($n=717$; 87.1%) and 17 years of age ($n=713$; 86.6%). Six hundred and fifty-seven children completed both age 11 and age 17 follow-up assessments. Some of the designated participants for this study (i.e., the 657 children) had missing or invalid responses to key study variables. For this reason, the effective sample size for the present investigation and the proportion of designated participants with useable data are 641 and 77.9%, respectively. The study protocol was reviewed and approved by the cognizant institutional review board for protection of human subjects in research.

The key response variable for the study was the cumulative incidence of cannabis use up to age 17 years. It was assessed during the age 17 assessment via youth self-report

using the following standardized dichotomous (coded *Yes (1)* or *No (0)*) question about cannabis use: “Have you ever, even once, used marijuana or hashish?” Data from the age 11 assessment were used to identify children who had initiated cannabis smoking by age 11 and were therefore no longer at risk for initiation during the 11 to 17 years age-span. In addition, for those respondents who had initiated cannabis by age 17, a follow-up question queried the age of their first cannabis use.

The suspected causal determinant of interest was level of parental monitoring. It was assessed by child self-report at age 11 via a standardized 10-item scale (the full scale is attached in Appendix D) (Capaldi & Patterson, 1989; Chilcoat et al., 1995). The items encompass child supervision and tracking of activities outside the school environment (e.g., whether an adult was present within 1 hour of the child arriving home from school, how often the child talked with the parents about plans for the coming day, and whether the child knew how to contact the parents if they were not at home after school). On seven of the items, children responded on a five-point scale ranging from *All of the time (1)* to *Never (5)*; responses were coded either *Clear (1)* or *Unclear (2)* on two items; and on a single item the responses were coded *Yes (1)* or *No (2)*. A parental monitoring score was constructed by reversing the coding and summing the scores on the 10 items.

Possible scores ranged from a low of 10 to a high of 41.

The guiding conceptual model was one in which the risk of cannabis initiation from age 11 to 17 years was predicted by level of parental monitoring at age 11 years. The plan for data analysis was organized in relation to standard "explore, analyze, explore" cycles, in which the first exploratory steps involve Tukey-style box-and-whisker plots and other exploratory data analyses to shed light on the underlying distributions of

each response variable and covariate of interest. In the initial analysis step, the task was to estimate the cumulative occurrence of cannabis initiation from ages 11 to 17 years. First, using a method described by Zou (2004), unadjusted and adjusted Poisson regressions with robust error variances were conducted to estimate the predictive association linking parental monitoring at age 11 years with the initiation of cannabis use from the ages of 11 to 17 years (Zou, 2004). Estimates using this method have been shown to yield more precise estimates of relative risk than odds ratios derived in traditional logistic regression, especially when the outcome of interest occurs in greater than 10% of the sample (i.e., when estimated odds ratios may not approximate relative risk) (Zou, 2004). The general equation for the Poisson regression is given by the equation:

$$\log [\pi(x_i)] = \alpha + \beta x_i + \dots$$

where $\pi(x_i)$ is the probability of the outcome; β is the slope of the main covariate of interest. The exponentiation of β gives the relative risk, and the robust sandwich estimator is used to give the appropriate variance. Male-female subgroup variation in the parental monitoring-cannabis initiation association was evaluated via product terms, as was subgroup variation associated with race. None were detected at the alpha level of 0.05.

For a subsidiary survival analysis, a dichotomous variable was constructed from the original parental monitoring scale using the median as the cutoff (*low parental monitoring* (<37) and *high parental monitoring* (≥ 37)). Next, Kaplan-Meier curves were derived for the above- and below-median monitoring groups in order to inspect the failure rates of first cannabis use, year by year from age 11 to 17 years. Specifically, the

approach involved specifying the elapsed time from the age at the age 11 assessment until the age of first cannabis use. Adolescents who never used cannabis contributed person-years up to the time of their age at the age 17 interview. A log-rank test was conducted to formally test whether the two survival curves differed from one another ($\alpha = 0.05$).

In a post-estimation exploratory step, the method of plotting fractional polynomials, as described by Royston and Altman (1994), was employed to probe into the issue of possible non-linearity in the parental monitoring – cannabis initiation association (Royston & Altman, 1994). All analyses were conducted using Stata 11 (StataCorp, 2009). In this work, precision of the study estimates are stressed with a focus on 95% confidence intervals; p-values are presented as an aid to interpretation.

APPENDIX B

Technical Appendix for Chapter Four

In this study the research design is a prospective cohort with three waves of assessment. The study population was designated to include low birth weight (LBW) and normal birth weight (NBW) newborns from southeast Michigan. The sampling approach involved randomly selecting LBW and NBW children from 1983-1985 newborn discharge lists of two hospitals in southeast Michigan, one serving an inner-city community and the other serving a suburban community. Forty-seven children with severe neurologic impairments were excluded from the initial sample. The resulting sample consisted of 1095 children. Of the 1095 children eligible for the study, 823 (75.2%) participated in the initial assessment from 1990 to 1992, when they were six years of age. Follow-up assessments were conducted at 11 years ($n=717$; 87.1%) and 17 years of age ($n=713$; 86.6%). Of the original cohort of 823 children, 774 (94.0%) who had information on at least one outcome variable contributed data to the present investigation. The institutional review boards of the participating institutions approved the study.

There were several key variables for this study. Parental monitoring (PM 11) was assessed via child self-report at 11 years of age using a standardized 10-item scale (Appendix D) (Capaldi & Patterson, 1989; Chilcoat et al., 1995). The items encompass child supervision and tracking of activities outside the school environment (e.g., whether an adult was present within 1 hour of the child arriving home from school, how often the child talked with the parents about plans for the coming day, and whether the child knew

how to contact the parents if they were not at home after school). On seven of the items, children responded on a five-point scale ranging from *All of the time* (1) to *Never* (5); responses were coded either *Clear* (1) or *Unclear* (2) on two items; and on a single item the responses were coded *Yes* (1) or *No* (2). A summary score was generated by reversing the coding and summing the scores. Scores ranged from a low of 10 to a high of 41, with higher scores indicating higher levels of monitoring.

Level of drug use at age 11 (Drug 11) was constructed as a continuous latent variable from the following standardized, child-reported age of onset questions which were coded into dichotomous used/never used variables for alcohol, tobacco cigarette smoking and cannabis smoking, respectively: (1) “How old were you the first time you drank beer, wine, wine coolers, liquor, or any other drink with alcohol in it?”; (2) “How old were you when you first smoked a tobacco cigarette, even just a puff?”; (3) “How old were you the first time you smoked (marijuana/reefer)?”

Level of affiliation with drug using peers at age 11 (DUP 11) was constructed as a continuous latent variable from the following standardized, child-reported dichotomous (yes/no) questions on peer alcohol, tobacco cigarette smoking, and cannabis smoking, respectively: (1) “Do you have any friends around your age who ever drink alcohol?”; (2) “Do you have any friends around your age who ever smoke tobacco cigarettes?”; (3) “Do you have any friends around your age who ever smoke (marijuana/reefer)?”

Level of drug use at age 17 (Drug 17) was constructed as a continuous latent variable from the following three standardized, child-reported dichotomous (yes/no) questions on alcohol, tobacco cigarette smoking and cannabis smoking, respectively: (1) “Have you ever, even once, had a drink of any type of alcoholic beverage? *Do not*

include sips from another person's drink..”; (2) “Have you ever smoked a cigarette, even one or two puffs?”; (3) “Have you ever, even once, used marijuana or hashish?”

Structural equation modeling (SEM) was used to simultaneously test paths of PM11 with DUP11 and Drug 11 and 17. All models were fit in Mplus 6.0 using the weighted least squares means and variance adjusted estimator (Muthén & Muthén, 1998-2010). The latent variable SEM approach provides several advantages over traditional regression techniques. First, it allows for simultaneous testing of multiple regression paths of complex relationships. Second, common sources of variation between outcome variables are taken into account and the path estimates are unencumbered by assumptions about error. Third, Mplus handles missing data using multiple imputation methods to use all available data (Muthén & Muthén, 1998-2010). Model fit was assessed via the Comparative Fit Index (CFI) (where good model fit is denoted by a score >0.90) (Bentler, 1990), the Tucker-Lewis Index (TLI) (where good model fit is denoted by a score >0.90) (Bentler & Bonett, 1980), and the Root Mean Square Error of Approximation (RMSEA) (where good model fit is denoted by a score <0.05) (Bentler, 1990).

APPENDIX C

Technical Appendix for Chapter Five

The present study builds on an epidemiology and prevention research program initiated by Professors Sheppard Kellam, James C. Anthony, and their colleagues at the Prevention Research Center of the Johns Hopkins University Bloomberg School of Public Health. The data come from a prospective longitudinal study completed within the context of a group-randomized trial. At the time of entry into first grade, children were randomly assigned to either an intervention classroom, with a Good Behavior Game program or Mastery Learning Program, or to a standard classroom. The study population was designated to include all first graders entering 19 public elementary schools of a single urban school system located in the mid-Atlantic United States during two successive school years (cohort 1 entering in 1985 and cohort 2 entering in 1986). Sub-sampling did not take place; efforts were made to recruit all incoming first-graders ($n=2,311$; cohort 1 = 1196, cohort 2 = 1115).

The 19 participating schools were located within the catchment area of five pre-identified urban areas of the school system. The composition of these areas encompassed very poor to middle class families who were mainly non-Hispanic black and white. Moreover, the 19 schools and city neighborhoods where the children were growing up were conceptualized as an ecological niche. Therefore, follow-up assessments were focused on the first-graders who remained in the niche.

From 1985 through 1994, children were assessed via regular standardized face-to-face interviews with teachers. Starting at third grade (1989), and continuing through 1994, there were standardized face-to-face interviews with the children themselves, which covered a variety of health and behavioral outcomes, including drug use. The present investigation focuses on the child assessments from 1989 through 1994, when the children ranged in age from 8 and 9 to 13 and 14 years old.

Each year in the Spring from 1989 through 1993, the interview was repeated. The number of students interviewed varied each year by the maximum number of assessments allowed by the school that respective Spring. In the Spring of 1994, the investigators decided to focus resources on the second cohort of students who remained in the school system, and they only interviewed cohort 1 students when cohort 2 students were not available. Therefore, there were lower numbers of participants in 1994. The sample sizes for each year from 1989 through 1994 were: 1530, 1233, 1543, 1416, 1251, and 816, respectively.

The main outcome of interest was recently active (past-year) cannabis smoking. It was assessed in the interviews every spring from 1990 through 1994 via a single child-reported item: “When was the last time you smoked (marijuana/Reefer)?” Children were coded into a dichotomous variable; those who had used cannabis since June 1 of the previous year (1) and those who had not (0). Past-year use in 1990 was coded to include individuals who were new initiates and did not use cannabis in 1989.

The main suspected causal determinant was level of parental monitoring. Parental monitoring was assessed each Spring from 1989 through 1994 via a standardized, child-reported 10-item scale. As described elsewhere, the items were drawn from the Oregon

Social Learning Center Parent Monitoring Scale and adapted for age-appropriateness (Capaldi & Patterson, 1989; Chilcoat et al., 1995). The items in the scale encompass parental supervision, tracking, and knowledge of child whereabouts (e.g. “How often, before you go out, do you tell your parents when you will be back?”). The complete scale is included in Appendix D. Possible scores ranged from a low of 10 to a high of 41.

Another central variable of interest was level of deviant peer affiliation. The variable was assessed each Spring from 1989 through 1994 via a standardized, child-reported five-item scale. As previously described, the items were drawn from the Oregon Social Learning Center Peer Behavior Scale and adapted for age-appropriateness (Capaldi & Patterson, 1989; Chilcoat et al., 1995; Lloyd & Anthony, 2003). The five items assess friendships with children who participate in deviant behaviors such as cheating on tests and hitting others. The scale is included in the appendix (Appendix B). Possible score ranged from a low of 5 to a high of 25.

The present investigation employed several analytic steps. First, a logistic regression approach was used, applying generalized estimating equations (GEE), to estimate the time-series relationship linking prior level of parental monitoring with the probability of recent cannabis use across the five outcome time points (i.e., 1990-1994) (Zeger et al., 1988). The model is a population-averaged model. The general equation is as follows:

$$\text{logit}(\mu_{ij}) = \beta_0 + \sum_{h=1}^p \beta_h X_h$$

where μ_{ij} is the marginal probability of the outcome; β_0 is the intercept; β_h is the slope for a given covariate of interest from the array of covariates under study. In the present

study, the log-odds of recently active cannabis use was modeled as a function of prior parental monitoring (i.e. the data was set up so that each row was time-lagged; the log-odds of recently active cannabis use for time point t was regressed on parental monitoring for time point $t-1$). Next, the model was elaborated to include potentially confounding covariates. These covariates included: sex, racial/ethnic minority status, free/subsidized school lunch status at first grade, teacher-rated aggression in first grade, prior recent tobacco and alcohol use (i.e., time-lagged, $t-1$), cohort, intervention group, and year of outcome assessment. A few children ($n=11$) tried cannabis before the time of initial PM assessment; a variable to indicate this history of cannabis smoking was also introduced as a covariate. Product-terms between the covariates and parental monitoring were also evaluated. None were detected at the alpha level of 0.05. The GEE produces population-averaged estimates; taking into account interdependencies of repeated observations for the same subject over the survey assessment time points. It also offers the advantage of using all available data (Zeger et al., 1988). For the present analysis, 1,448 individuals had available data for these GEE analyses, which were performed using Stata 11 (StataCorp, 2009).

Second, the method of alternating logistic regression (ALR) was used to check the estimates obtained from the adjusted GEE model and examine the potential influence of clustering at the classroom/school level (Bobashev & Anthony, 2000). ALR is similar to GEE in that it takes into account interdependencies of correlated data and produces population averaged estimates. Unlike GEE, however, ALR produces a directly interpretable estimate of the amount of clustering in the data in the form of the pair-wise odds ratio (PWOR). The PWOR is analogous to the cross-product ratio in a two-by-two

table. Furthermore, ALR is capable of handling two levels of clustering; here, the individual and the classroom/school. The general equation for a two level nested design is as follows:

$$\log(PWOR_{jklm}) = \alpha_0 Z_{0jk} + \alpha_1 Z_{1lm}$$

where Z_{0jk} is 1 if the pair (j,k) belongs to the same cluster (classroom/school) or 0 if not, and Z_{1lm} is 1 if the pair (l,m) belongs to the same subcluster (individual) or 0 if not.

Exponentiation of (α_0) and $(\alpha_0 + \alpha_1)$ gives the PWORs for the cluster and subcluster, respectively. The ALR model was fit using SAS 9.1 (SAS Institute, Cary, NC).

Third, structural equation modeling (SEM)/path analysis was used for simultaneous examination of prospective paths from level of PM to level of DPA and onward to CAN. Following the steps outlined by Baron and Kenny (1986), three separate models were estimated to examine mediation (Baron & Kenny, 1986). In the first model, paths from level of PM at each time t to CAN at each time $t+2$ were estimated to test for direct effects. The model also simultaneously adjusted for prior and subsequent assessments of PM and CAN. In the second step, paths from level of PM at each time t to level of DPA at each time $t+1$ were modeled to test for the predictive association linking prior PM, the initial variable, with subsequent DPA, the hypothesized mediator. In the second model, statistical adjustment was made for prior and subsequent assessments of level of PM and level of DPA. In the comprehensive third model, effects were estimated for the paths from level of PM at time t to level of DPA at time $t+1$ to CAN at time $t+2$, as well as for the direct path from level of PM at t to CAN at $t+2$. The third model also simultaneously adjusted for prior and subsequent assessments of PM, DPA, and CAN. For all three models, SEM was implemented under maximum

likelihood estimation with robust standard errors (MLR) using Monte Carlo integration with 1000 integration points in Mplus 6.0 (Muthén & Muthén, 1998-2010). The MLR SEM approach in Mplus uses multiple imputation methods that allow for the inclusion of subjects with incomplete data (Muthén & Muthén, 1998-2010). The SEM analyses were conducted for 1,302 individuals with available data. Mplus produced linear regression slopes for the meditational path when level of DPA was modeled as the outcome (i.e., Gaussian) and logistic regression slopes in the form of log-odds with corresponding odds ratios when CAN was the outcome (i.e., binary).

Fourth, main predictive paths that were statistically robust in all three models (i.e., where the paths were robust from PM to recent cannabis use, PM to DPA, and PM to DPA to recent cannabis use) were tested for mediation using the `binary_mediation` program with the bootstrap command with 500 replications in Stata 11 (StataCorp, 2009; UCLA). In accordance with Kenny's approach for testing mediation with dichotomous outcomes, the program standardizes the coefficients before using the product of coefficients method to compute indirect effects (Kenny, 2009; UCLA). This allows for the direct comparison of coefficients from one model to another in computing the direct and indirect effects (Kenny, 2009; UCLA). It should be noted, however, that some of the complexities of the data that are accounted for in the SEM analyses (e.g., repeated measurements) are lost in the traditional regression framework of the `binary_mediation` program.

APPENDIX D

Parental Monitoring Scale

1. What time does your mom or dad or parents expect you to come home from school? (clear, unclear)
2. What time do they expect you to come home on a weekend night? (clear, unclear)
3. How often would your mom or dad or parents or a sitter know if you came home an hour late on weekends? (always, most of the time, sometimes, hardly ever, never)
4. Are there kids your mom or dad or parents don't allow you to play with? (always, most of the time, sometimes, hardly ever, never)
5. How often, before you go out, do you tell your parents when you will be back? (always, most of the time, sometimes, hardly ever, never)
6. If your parents or a sitter are not at home, how often do you leave a note for them about where you are going? (always, most of the time, sometimes, hardly ever, never)
7. How often do you check in with your parents or a sitter after school before going to play? (always, most of the time, sometimes, hardly ever, never)
8. When you get home from school, how often is someone there within 1 hour? (always, most of the time, sometimes, hardly ever, never)
9. If you are at home when your parents are not, how often do you know how to get in touch with them? (always, most of the time, sometimes, hardly ever, never)
10. How often do you talk with your mom or dad or parents about your plans for the coming day? For instance, talk about what will happen at school or with friends? (always, most of the time, sometimes, hardly ever, never)

APPENDIX E

Deviant Peer Affiliation Scale

1. During the last school year, how many of your friends have cheated on school tests? (none, very few of them, some of them, most of them, all of them)
2. During the last year, how many of your friends have ruined or damaged something on purpose that wasn't theirs? (none, very few of them, some of them, most of them, all of them)
3. During the last year, how many of your friends have stolen something worth less than five dollars? (none, very few of them, some of them, most of them, all of them)
4. During the last year, how many of your friends have hit or threatened to hit someone without any real reason? (none, very few of them, some of them, most of them, all of them)
5. During the last year, how many of your friends have suggested that you do something against the law? (none, very few of them, some of them, most of them, all of them)

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