EXAMINING BIDIRECTIONAL RELATIONSHIPS BETWEEN PARENTAL SOCIALIZATION BEHAVIORS AND ADOLESCENT ALCOHOL MISUSE ACROSS EARLY AND MIDDLE ADOLESCENCE

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ABSTRACT

Melissa J. Cox: Examining Bidirectional Relationships Between Parental Socialization Behaviors and Adolescent Alcohol Misuse Across Early and Middle Adolescence
(Under the direction of Susan Ennett)

While numerous studies have examined the influence of parenting on adolescent alcohol use, relatively few have examined how adolescents influence parental behavior or the reciprocal nature of parent-adolescent behavior relative to alcohol use. Jointly guided by socialization theories and transactional models of development, the purpose of this dissertation was to examine reciprocal effects between parental socialization behaviors and adolescent alcohol misuse across early and middle adolescence. The study assessed bidirectional relationships between adolescent alcohol misuse and three general parental socialization behaviors (closeness, support, behavioral control), three alcohol-specific socialization behaviors (alcohol-specific monitoring, negative and permissive communication messages about alcohol) and parental alcohol use. To address developmental considerations underlying parent-adolescent relationships, the study also examined the stability and change of the reciprocal relations across early and middle adolescence. Data were from 1645 parent-adolescent dyads drawn from a longitudinal study of adolescent health risk behaviors spanning grades 6 through 10. A multivariate latent curve model with structured residuals, an extension of the autoregressive latent trajectory model, was used to test study hypotheses. This model was chosen to disaggregate developmental processes underlying the proposed relationships that occur across parent-adolescent dyads and over time within each dyad. Results suggest that increased adolescent alcohol misuse leads to greater alcohol-specific monitoring behaviors by parents across all grades. This finding substantiates the theoretical expectation that parental behavior is partially determined by the actions of their child, the direction of influence often left out of previous socialization research. No other relations between adolescent alcohol misuse and parental socialization behaviors were found after accounting for underlying developmental processes and necessary controls. While the few significant results limit implications for practice, results from this study

provide a basis for future research to examine more dynamic transactional processes between parents and adolescents relative to alcohol use.

To my parents, who have always supported my wildest dreams.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER 1: INTRODUCTION

Adolescent alcohol misuse describes a pattern of drinking comprised of high-risk drinking, problems associated with drinking and high alcohol consumption levels.[1] This type of alcohol use is harmful to a range of health outcomes both during adolescence and into adulthood.[1,2] A substantial body of research on adolescent alcohol use has examined how parental behaviors, such as fostering a supportive parent-child relationship and parents' own drinking, impacts adolescent drinking. However, limited research has examined how adolescents influence parental behaviors and the reciprocal nature of parent and child behaviors relative to alcohol use. For example, research has indicated that when adolescents drink more alcohol, their parents subsequently exhibit less behavioral control, a known risk factor for adolescent alcohol use.[3] Studies that clarify how the relationship between parental behaviors and adolescent alcohol misuse develops during adolescence may help inform prevention efforts across this critical part of the lifespan. This dissertation assesses bidirectional influences between adolescent alcohol misuse and seven parental socialization behaviors using longitudinal data that spanned early and middle adolescence.

Socialization is the process by which an individual learns the norms and accepted behaviors of society. The process occurs through a continual exchange between the individual and influential others. For the developing child, family is the most proximal source of influence, and parents play a critical role in socializing their child to the use of alcohol, a culturally accepted substance used by most adults in the United States. What parents do and say, both generally and specific to alcohol, influences adolescent alcohol use and misuse. Parents who facilitate strong bonds with their children, demonstrate to their child they are loved and supported, exert developmentally appropriate levels of control over their child's activities and who limit their own alcohol use decrease the risk their child will engage in alcohol use during adolescence.[4-11] In addition, behaviors that parents employ specific to alcohol use, such as setting rules about alcohol use, produce unique effects on adolescent alcohol use beyond their general approach to parenting.[12]

The relationship between parents and teens regarding to alcohol use, however, is not limited to the influence of parents on teens. Transactional models of development state that a person and his or her environment are engaged in a continual process of mutual influence. That is, a person is developing in response to the changing environment, which in turn develops based on the changing individual. Applied to the parent-child dynamic regarding alcohol use, transactional theories suggest that an adolescent's misuse of alcohol and the socialization behaviors of his or her parent are responsive to each other. To date, empirical studies on transactional models of development have largely focused on infant and early childhood outcomes.[13-17] Few studies have examined reciprocal relationships between parents and adolescents,[18-20] and no studies have examined bidirectional processes regarding parental socialization behaviors and the particularly consequential outcome of alcohol misuse during adolescence.

Using longitudinal data collected from both parents and adolescents, the current study addresses this gap by examining bidirectional relationships across early and middle adolescence between adolescent alcohol misuse and seven parental socialization behaviors. The study assesses three general parenting behaviors common to socialization research on adolescent alcohol use, but not yet examined with regard to their reciprocity with adolescent alcohol misuse: parental closeness, support, and behavioral control. The study extends examination of parental socialization behaviors to incorporate three behaviors specific to alcohol use that are supported by socialization theories, but not frequently examined in empirical research on the socialization of adolescent alcohol use: alcohol-specific monitoring and negative and permissive messages about alcohol. Additionally, the study assesses reciprocal relations between parental alcohol use and adolescent alcohol misuse.

Not only are the relationships between parental socialization behaviors and adolescent alcohol misuse potentially reciprocal in nature, they are also subject to developmental changes that occur as an adolescent matures. With increasing age, adolescents' sense of self and personal autonomy develop and exposure to new experiences within and outside the family increases. These changes shape the relationship adolescents have with their parents. For example, adolescents may begin to rebel if they feel their parents are encroaching on the expanded independence they seek.[21,22] Because of these developmental considerations, the study also tests whether reciprocal influences between parental socialization behaviors and adolescent alcohol misuse change across adolescence.

Adolescent alcohol misuse is a particularly salient case for testing bidirectional pathways to determine whether the behaviors of parents and teens are dynamically linked across adolescence. First, alcohol is the most commonly used substance by adolescents. Initiation of alcohol use, as well as escalation of use, often occurs during adolescence. Second, alcohol misuse is an exceptionally risky form of drinking during adolescence that holds severe consequences both during adolescence and into adulthood. Third, parents are concerned about the potential negative consequences of adolescent alcohol use and want to prevent risky drinking by their teens. Thus, better understanding the dynamics of parenting and alcohol misuse is informative to family-based prevention programs.

Theory and empirical research present a rationale for examining bidirectional processes between parents and adolescents to more fully understand the development of adolescent alcohol misuse. These processes are multifaceted and include those that are driven by characteristics of individuals as well as those specific to the timing of when a behavior occurs. Research on bidirectional processes relating parent and adolescent behavior therefore requires specific analytical techniques to disentangle multiple mechanisms of influence. Those processes related to characteristics of individuals encompass between-person, or interindividual processes. Processes that unfold within an individual, termed within-person or intraindividual processes, explain potentially meaningful relations between time-specific deviations from a person's average level of behavior. This study focused on within-person processes as I sought to understand how the behavior of a parent or teen at one point in time influences the behavior of the other at a subsequent time. These time-specific relations represent reactions of an individual to his or her environment as indicated by transactional models of development.

This dissertation utilizes a theoretically informed framework with appropriately matched analytical methods to assess bidirectional relationships between adolescent alcohol misuse and parental socialization behaviors across early and middle adolescence. The study extends previous socialization research on adolescent alcohol use by examining reciprocal relations between alcohol misuse and three general parental behaviors, three socialization practices specific to alcohol use and parental alcohol use. Results from this study are intended to inform strategies for family-based prevention programs. In the chapters that follow I detail the theoretical and empirical evidence supporting the need to examine bidirectional processes between adolescent alcohol misuse and parental socialization behaviors. I then

describe the methods for the research, including the analytical framework used to test study hypotheses. Following the results, I discuss study findings, provide implications of the research for public health practice and suggest future directions for research on the dynamics of familial processes relative to youth alcohol misuse.

CHAPTER 2: THEORETICAL AND EMPIRICAL EVIDENCE FOR THE RELATIONSHIP BETWEEN PARENTAL SOCIALIZATION BEHAVIORS AND ADOLESCENT ALCOHOL MISUSE

The Problem of Adolescent Alcohol Misuse

Currently in the United States, more than 50% of teenagers have had a drink of alcohol by the age of 15, a figure that rises to 70% by age 18.[23] Approximately 10% of youth have initiated drinking by 10 years old,[24] with nearly one third of American youth beginning to drink by age 13.[25] Initiation of alcohol use is most likely to occur during 7th and 8th grades, when youth are 12-13 years old.[26] Early initiation of alcohol use is particularly concerning, as youth who begin drinking at earlier ages are more likely to develop problem alcohol use and misuse during adolescence[24,27,28] and later in life.[29]

Alcohol misuse describes a pattern of drinking behavior that is harmful to a range of health outcomes, as well as interpersonal relationships and social responsibilities.[1,2] Multiple components of drinking and its consequences comprise alcohol misuse. These include high-risk drinking, problems associated with drinking and high alcohol consumption levels.[1] For adolescents, high-risk drinking reflects circumstances that place youth at particularly elevated risk for detrimental consequences such as drinking before or after school, being drunk, using alcohol with other drugs, and drinking while driving. Problems associated with drinking include missing school or work, getting in a fight or feeling sick due to drinking.[30] High consumption levels reflect excessive alcohol use such as heavy episodic drinking, or binge drinking, defined as four drinks for women and five drinks for men in a singular episode, as well as longer patterns of excessive use.[31] Binge drinking is common among adolescents who drink alcohol, accounting for the majority of alcohol consumed among 12-20 year olds in the United States.[32,33] Thus, when adolescents drink alcohol, they do so in an exceptionally risky manner.

Alcohol misuse is of significant public health concern, as episodes of heavy drinking and patterns of heightened drinking escalate risk for negative health and social outcomes. When adolescents consume large quantities of alcohol, they are simultaneously at risk for riding in the car with someone who has been drinking and more severely, being involved in automobile accidents and fatalities.[33-37] High intoxication levels also make adolescents more vulnerable to risky sexual encounters and heighten risk

for dating violence and aggression.[33,37-40] In addition, alcohol misuse increases the likelihood that adolescents will use other substances including cigarettes and illicit drugs, [33] as well as experience suicidal ideation and behavior.[33,41]

Not only does alcohol misuse amplify risk of these negative outcomes at the time of drinking, the consequences persist into adulthood. For both men and women, excessive drinking during adolescence is significantly associated with similar drinking patterns in young adulthood.[42] Binge drinking during adolescence places youth on a problematic trajectory towards alcohol abuse and dependence in adulthood.[34] Such persistent drinking behavior also detracts from adolescents' ability to maximize their educational, social and economic potential, consequences that have lifelong ramifications.[28]

Parental Socialization of Adolescent Alcohol Misuse

Socialization is the process by which individuals learn the norms, values, and accepted behaviors of their society. The socialization process constitutes a continual exchange between individuals and those who seek to influence them. Primary socialization theory,[43] which draws on social control and social learning theories, points to the primacy of the family as the socialization source from early life that carries forward to adolescence.[44] "The family is, after all, the most proximal social system in which patterned exposure occurs; it generally guarantees a continuity of exposure extending back in time to the earliest consciousness of social meanings; and it is the single milieu that encompasses, at pre-adolescence, the widest range of experiences and involvements for the child"[45] (p.119). Norms held by family members, in particular parents, are passed on to children and serve as the basis for how the child learns what is expected and valued in his or her society.

Underlying primary socialization theory is the premise that human social behaviors are mostly, if not fully, learned behaviors and that this learning process occurs through interactions with proximal sources of influence. The theory is intended to explain adolescent substance use and other risk behaviors. Adolescents in the US grow up within a society in which responsible adult drinking is generally accepted, and where most adults do drink alcohol. Accordingly, alcohol use can be considered a learned social behavior. As adolescents are increasingly exposed to opportunities for drinking alcohol, the foundation of socialization built in childhood, and the particular parental socialization behaviors enacted

during adolescence are of critical importance to preventing adolescent alcohol use and its escalation to misuse.

For years researchers have sought to define the conditions that support optimal parenting and effective parental socialization. How parents influence child outcomes is the focus of a large body of theory on parenting style. Early work by Baldwin and colleagues advocated for democratic parenting, which entailed maximum participation of children in family decision-making.[46,47] From this came the formative work of Diana Baumrind[48,49] that defined three parenting style typologies based on the dimensions of responsiveness, or the degree to which parents respond to their child's needs, and demandingness, which reflects parental expectations for mature and responsible behavior consistent with the child's developmental stage. Baumrind's conclusion that authoritative parenting, marked by high levels of responsiveness along with consistent and reinforced demands for meeting limits and expectations, is the most conducive for effective parenting has remained central to studies of familial socialization.[50] Responsiveness and demandingness are two dimensions of parenting that have defined parental socialization theories and the many empirical studies of parental influence on adolescent substance use that followed. Others have extended the typology to a four-fold scheme based on cross-classifying the two dimensions of responsiveness and demandingness,[44] and added further dimensions including psychological autonomy, a related construct to that of Baldwin's democracy.[51] However defined, this work demonstrated the parental role of socializing the child in a way that reflects reciprocity between the needs and demands of self and other. Parents reflect this reciprocal dynamic by altering their own parenting behavior to meet the needs of their growing child. That is, parents alter their behaviors to support the changing needs of a developing child while relaying increasingly complex messages about their expectations for responsible behavior as the child assumes more mature social roles.

Parental socialization theories predicated on dimensions of responsiveness and demandingness describe the general approach parents take to parenting, which is independent of the content of a particular parenting behavior or the specific outcome the parent is intending to influence.[52] In addition to these global parenting characteristics, parents also employ behaviors intended to influence a specific child outcome, such as academic achievement, dietary habits, religious norms or alcohol use behaviors. Parents employ specific behaviors to help children meet specific socialization goals.[52] Thus, general and

behavior-specific parenting tactics are related, yet distinct components of parental socialization. For example, two equally authoritative parents may employ different parenting practices related to alcohol use: one may allow the child to have a drink at family occasions while the other does not permit the child to drink alcohol under any circumstance. Assessing parental socialization of adolescent alcohol use requires an examination of both general and alcohol-specific parenting behaviors.

Socialization theories regarding general and behavior-specific parenting have focused on how parenting influences child behavior, with little attention paid to the alternative direction, how a child influences parental behavior. To more effectively prevent risky adolescent behaviors including alcohol misuse, scholars have called for a movement from a focus on the individuals involved, namely teens and their parents, to a focus on the interactions between the two.[53] This shift in focus underscores the need to investigate the processes that occur within the family that shape adolescent drinking. Fundamental to this investigation is an assessment of bidirectional relationships, or the specific ways in which parents and teens influence one another relative to alcohol use. These bidirectional relationships are the core of transactional models of development.

Transactional Models of Development

The socialization literature through the mid twentieth century provided extensive support for the role of family in shaping a child's development. Until this point, however, conceptualizations of the parent-child relationship depicted unidirectional influence from parent to child. In this respect, children were passive agents responding to the behaviors of their parents or primary caregivers. In 1968, Bell[54] published a seminal review paper that reinterpreted the putative parent effects findings of previous studies. Bell offered an alternative view that included not only parent effects on child behaviors and outcomes, but also the effects of child characteristics on parental behaviors and practices. To support his claims, Bell examined both human and animal studies to find evidence for a broader approach than the unilateral perspective. Rather than a fixed-effects approach to parenting, Bell argued that parents modified their own behavior based on child characteristics, such as assertiveness and temperament. He contended that parents have a repertoire of behaviors from which they select based on stimulation and reinforcement from the child. For example, the cry of an infant activates different parental responses

based on the level of distress exhibited by the crying infant. Not only do children activate types of parental behavioral responses, but they also affect the level and reinforcement of those behaviors.

Building on the work of Bell, researchers began to put forth developmental models that portrayed the child as having an active role in the socialization process. These models incorporated dynamic exchanges between parent and child that shape the growth and subsequent behavior of each.[55,56] Kuczynski and colleagues provided a conceptual model for bidirectional socialization that incorporated both parent and child as 'interdependent, active agents in a process of mutual influence'[56] (p.27). Sameroff[57] distinguished mechanistic and dialectical exchange orientations to describe processes in which the dynamic interplay between parent and child occurs. From a mechanistic stance, there is a linear prediction of one variable on another, from organism to environment or environment to organism. Contrastingly, a dialectical approach addresses transactions between an organism and environment that mutually alters development of each, either in form or level[58] In addition, Lerner and Spanier[59]documented a dynamic interactional model of development that centered on a continuous interdependency between an individual and social change processes. Development occurs due to these exchanges, a process that is based on a series of interactions between an individual and his or her environment such that an individual is constantly adjusting to a changing environment and the environment is continuously responding to the changing individual.

An emphasis on dynamic processes has extended previous theories widely used in family-based research. For example, early iterations of social learning theory[60] framed the learning process as one in which parents shaped their child's growth via their own behavior. Interaction theorists extended this model to include the reciprocal processes that underlie socialization. These new conceptualizations of mutual exchange centered on feedback patterns to incorporate both action and reaction within the exchange system.[61] A much studied and replicated example of the reciprocal effects between parent and child is suggested by coercion theory. Simply, coercion theory explores how parents and children mutually train each other in ways that increase risk for aggressive behavior. A cascade of destructive events ensues as negative child behaviors are reinforced and parents' control over these behaviors diminishes.[62-65] The severity of this process and its consequences are due to the ongoing negative exchanges between parent and child.

The severity of the immediate and long-term consequences of alcohol misuse makes this type of drinking an important case for studying bidirectional relationships. As suggested by coercion theory, negative cycles of behavior between a parent and child may prolong deviant actions, and the consequences of those actions. It may be that parents and teens perpetuate a cycle across adolescence in which weak parental socialization behaviors escalate alcohol misuse and vice versa, thereby reinforcing negative behaviors by both the parent and teen. Knowing how parents and adolescents interact relative to alcohol misuse may illuminate particular socialization processes, which in turn might suggest specific strategies for family-based prevention programs to prevent and intervene on multiple types of adolescent alcohol use.

General Parental Socialization Behaviors and Adolescent Alcohol Misuse

I now review the extant literature on the relationship between parental socialization behaviors and adolescent alcohol misuse. When applicable, I add findings from studies of bidirectional relationships to the more substantial literature base on parental influence on adolescent alcohol use. First, I review three aspects of general parental socialization: closeness, support and behavioral control.

Parental closeness. Because norms are passed on through links with socialization sources, the strength of the bond between a parent and child determines how effectively norms are transmitted; strong bonds are necessary for effective transmission of norms. Social control theory (SCT) posits that every individual holds deviant tendencies, which are manifested only when there are weak bonds between the individual and conventional society.[66-68] Secure attachments, derived from emotional connectedness to conventional role models, are indications of strong bonds and effective socialization. Parents who facilitate secure attachments with responsive actions are more likely to have children willing to comply with their expectations.[69] Adolescents who feel detached or isolated from their parents are less likely to internalize parental values and standards for pro-social behavior, and are therefore at greater risk for deviant behavior such as alcohol misuse. In contrast, children who feel close to their parents trust them to provide appropriate guidance as they mature and encounter new experiences.[69,70]

Cross-sectional studies have demonstrated that strong parent-child bonds increase the likelihood of delayed initiation into drinking[4,71] and lower levels of use during adolescence.[4,5,72] However, longitudinal studies have not replicated these findings.[73] Examining the alternative direction of this

relationship, use of alcohol in early adolescence weakens an adolescent's perceived closeness to his or her parents.[73] Early initiation into alcohol use can distance a child emotionally from their parents, decreasing the ability of the parents to fully relay pro-social expectations and values. This weakening of parental closeness due to problematic substance use also holds true in later adolescence and young adulthood,[74] indicating a persistent effect with long-term implications. In a study of reciprocal relations of alcohol use and the related construct of parental attachment, adolescent alcohol use had a negative influence on subsequent levels of parental attachment, although attachment did not influence later alcohol use.[73] More broadly, however, full reciprocal relations have been demonstrated between parental attachment and adolescent externalizing behavior.[75]

Parental support and behavioral control. Socialization research has long regarded support and control as two parenting elements applicable to a range of adolescent behaviors.[76,77] Socialization theories specific to parenting and the empirical studies that followed largely focused on these two parenting behaviors, which are derived from the dimensions of responsiveness and demandingness that underlie authoritative parenting. Parental support comprises behaviors that indicate to the child they are accepted and loved. Parental support is a related, yet distinct, construct from parental closeness. Closeness measures the emotional connectedness between parent and child, which facilitates the delivery and receipt of specific supportive behaviors such as giving praise and encouragement or physical affection. Parental control describes a set of actions used by parents to regulate their child's activities and direct their behavior in a manner acceptable to the parent. When parents exert control over their child's behaviors and activities, they restrict what the child can and cannot do without permission, and place boundaries on their activities. In doing so, children learn what is considered appropriate behavior.

For effective socialization, the family must function with adequate levels of parental support and control. Research regarding both parental support and control has concluded that effective socialization occurs with high levels of support and moderate levels of control.[77,78] Overall, parental support shows a consistent relationship with adolescent alcohol use such that higher levels of support are associated with lower levels of drinking.[6,7,76,78,79] Parental support may also indirectly influence adolescent alcohol use by increasing the strength of the bond between parent and child,[80] resulting in children being more receptive to the specific socialization behaviors discussed below.[9]

In contrast to parental support, results regarding parental control are much less definitive, in some research showing no significant relationship with adolescent alcohol use.[76] Researchers have found both negative linear[81,82] and quadratic[77,78,83] relationships between parental control and adolescent alcohol use. Curvilinear results can be explained in that too little and too much control may both be detrimental to adolescent outcomes.[7,76] A lack of control doesn't provide the child with adequate guidance and may interfere with a child's attachment to their parent, and in turn the extent to which they internalize parental values and norms. Too much control may result in rebellious acts by the adolescent, including devious behaviors such as alcohol use. More recent longitudinal studies provide equivocal evidence regarding the association between parental behavioral control and adolescent drinking. On the one hand, higher levels of parental behavioral control reduced adolescent drinking; [8,9,84] other evidence indicates that parental control had no effect on adolescent drinking.[85,86]

The limited research that has assessed bidirectional associations between parental behavior and adolescent alcohol use has largely focused on parental support and control. Assessing substance use generally, full reciprocal effects have been found between both parental support and control and adolescent substance use such that lower levels of these parental behaviors prospectively predicted higher levels of adolescent substance use, which in turn predicted lower subsequent levels of parental support and control.[18] Similarly with regard to alcohol use only, greater parental control resulted in less alcohol consumption by older adolescents, and when these teens drank more, parents exhibited less subsequent control.[3] Extending to related constructs of behavioral control, bidirectional effects were found between parental knowledge and monitoring of child whereabouts and adolescent alcohol consumption specifically,[73] substance use generally,[19] and overall problem behavior.[8,87] However, recent studies of reciprocal effects using modeling approaches similar to those I use in this study did not find bidirectional pathways between parental knowledge and heavy episodic drinking[20] nor parental monitoring and substance use generally.[88]

Alcohol-Specific Parental Socialization Behaviors and Adolescent Alcohol Misuse

Much of the literature argues for the importance of the general approach parents take to socializing their children; however, socialization also occurs as a result of more specific attempts to influence particular behaviors.[52,69] In fact, alcohol-specific socialization practices have produced unique

effects above and beyond general socialization behaviors.[12,84,89] Alcohol-specific parenting reflects a range of behaviors parents employ to deter or prevent their child from consuming alcohol.[90,91] This study examines three alcohol-specific socialization behaviors: alcohol-specific monitoring, negative communication messages and permissive communication messages about alcohol.

Alcohol-specific monitoring. Most studies of parental control operationalize the construct to include general rules and regulations on a wide range of adolescent activities, which is also the manner in which many studies define parental monitoring. In this way, parental monitoring reflects more general control efforts that pertain to multiple risk behaviors. However, it may be that monitoring practices specific to a behavior have unique effects on that behavior. While general parental monitoring has been the subject of substantial inquiry,[89,92] surprisingly few studies have examined monitoring behaviors specific to alcohol use.

Van der Vorst and colleagues[93] found that supervisory practices specific to alcohol use did not moderate the study's primary relationship between adolescents' drinking inside and outside the home. That is, the positive association between drinking inside and outside the home did not differ based on the level of parental supervision of their child's alcohol use. Examining students in fifth grade, Jackson et al[90] found that those students who believed their parents did not monitor their activity for alcohol use were at significantly greater odds for reporting alcohol use as compared to students who believed their parents would know if they drank alcohol. Only one study has examined reciprocal effects with regard to alcohol-specific monitoring and adolescent alcohol use. Results from this study of younger adolescents suggested that adolescent alcohol use resulted in lower subsequent parental alcohol-specific behavioral control and alternatively, increased alcohol-specific behavioral control led to decreased adolescent alcohol use.[3] Relatedly, rules regarding alcohol use decreased the likelihood of alcohol initiation, though the impact of alcohol-specific rules declined as an adolescent aged.[94,95] Bidirectionally, alcohol-specific rules decreased subsequent alcohol use, but only for those adolescents who had not already initiated alcohol use at baseline. For both drinking and non-drinking adolescents at baseline, alcohol use by the adolescent did not influence subsequent alcohol-specific rules.[94]

Parental communication regarding alcohol: permissive and negative messages*.* Specific socialization behaviors apply to not only what parents do to prevent their child from using alcohol, but also

what they say. To date, empirical literature on the influence of alcohol-related parent-child communication remains mixed. Inconsistent findings coupled with differences in methodological approaches make it difficult to conclusively interpret this body of literature. Encompassed within parent-child communication is the frequency, content, quality, and timing of the communication. Assuming that communication is fundamental to the parent-child relationship, it might be expected that greater frequency and quality of communication regarding alcohol would protect the adolescent from risky drinking behaviors. However, the evidence remains equivocal at best.

Several studies have found that higher frequency and better quality of parent-child communication were related to lower risk for both initiation and escalation of drinking across adolescence.[96] Others have found no significant effect of communication on adolescent drinking.[97,98] Some studies have shown potentially detrimental effects of communication on adolescent alcohol use. Assessing multiple components of parent-child communication on alcohol, Ennett et al's[99] study found no influence on the initiation of alcohol use, but some evidence to suggest that conversations about the rules and consequences of alcohol may have increased alcohol use for those who had already tried alcohol, a result similar to that of van der Vorst et al. [91] Additionally, more frequent communication, especially of the negative consequences of alcohol use, resulted in greater risk for initiation and escalation of use[100] as well as problematic alcohol use.[101]

Regarding specific content of parent-child communication related to alcohol use, Reimuller et al[102] studied parent-adolescent dyads and found two distinct domains of alcohol-specific communication. The first was permissive messages, or messages about when and under what circumstances drinking alcohol is okay. The second was negative messages, or messages related to parental rules and health consequences of alcohol use. While neither permissive nor negative messages predicted future alcohol use in the study, there was a significant interaction between permissive messages and adolescents' baseline alcohol use such that the positive relationship between permissive messages and alcohol use was stronger for those with higher versus lower levels of baseline alcohol use. Similarly, in a recent study of alcohol-specific parenting, parental permission to drink was related to higher initial levels and more rapid increases in drinking during adolescence.[103] Demonstrating a protective effect on adolescent

drinking, this study found that frequent messages regarding the consequences of alcohol use were associated with lower likelihood of initial drinking and a slower increase in drinking across adolescence.[103]

Parental Alcohol Use and Adolescent Alcohol Misuse

Parental drinking is related to adolescent alcohol consumption in several ways. First, it has been shown that children model their parents' behavior as described by social learning theory.[60] Adolescents whose parents drink regularly or heavily are at increased risk for alcohol use themselves.[10,104-107] Witnessing parents drink alcohol may lead adolescents to drink themselves or adopt pro-social norms regarding alcohol consumption.[108-110] The more often adolescents witness their parents drunk, the more likely they are to drink themselves.[111] Furthermore, those children that drink with their parents inside the home are more likely to also drink outside the home.[93]

Second, parental drinking may indirectly impact their adolescent's drinking in several important ways. One such mechanism is that parents who drink heavily are more lenient with regard to rules and monitoring practices, known risk factors for adolescent drinking.[112,113] Second, when parents drink in the home, alcohol is more readily available which also increases risk for adolescent alcohol use.[114] Additionally, it can be argued that regularly drinking parents consider themselves less credible in enforcing drinking rules on their children and are more permissive to teen drinking. Finally, parental drinking may also change the family environment to heighten teen alcohol use due to increased family conflict and less interaction between family members.[109]

Developmental Considerations in the Relationship Between Parental Socialization and Adolescent Alcohol Misuse

Not only is the relationship between parental socialization and adolescent alcohol misuse complex given the need to examine bidirectional processes and both general and alcohol-specific parenting behaviors, it is also influenced by developmental changes that occur during adolescence. The substantial physical, social and emotional growth that occurs during adolescence has implications for parent-child interactions. As adolescents develop, they navigate changing environments and work toward fulfilling more adult roles, and their relationships change as well. Physical and cognitive maturation impacts adolescents' perception of themselves and their relationships with parents and peers. Maturation can lead to shifts in the stability and strength of the parent-child bond, which as described previously, underlies the interactions between parent and child. Altogether, these developmental changes indicate a

need to examine parental and adolescent behavior at multiple time points across adolescence to assess stability and change within the relationship between parenting and adolescent alcohol misuse.

Adolescents undergo many changes during adolescence that prepare them to assume adult roles in society. Underlying this development is growth in personal autonomy, with the expectation that adolescents will seek greater independence as they get older, both in the roles they play as well as their relationships to those who have socialized them, including parents.[115] Expanded independence allows for greater personal control and autonomy, which are important characteristics for the transition to adulthood. While seeking independence may be normative, this shift holds implications for the parent-child relationship, and subsequently other outcomes such as alcohol use that are shaped by parental interaction.

As an adolescent gains greater autonomy, parents must adjust their control efforts to balance the need to monitor their adolescent's behavior while respecting the independence sought by their child.[116,117] Person-environment fit theory suggests that optimal results occur when personal factors are in congruence with environmental characteristics.[118,119] Applied to child development outcomes, the match between an adolescent's desire for independence and the level of parental control exerted is highly important. Children who feel too controlled by parents may feel resentment towards their parents, or even rebel against parental control.[21,22] Such rebellion could turn an adolescent away from pro-social activities encouraged through parents' socialization behaviors and to more deviant behaviors such as drinking. Optimal developmental outcomes occur when parental control is gradually reduced in response to a child's desire for more autonomy,[120] a process critically important during early and middle adolescent years.

Beyond specific control efforts, the overall stability of the parent-adolescent bond during adolescence has been subject to much research.[50,121-123] Processes associated with adolescent maturation have been theorized as factors underpinning destabilization of the parent-adolescent bond.[124-127] Cognitively, adolescents develop greater capacity for critical and abstract thinking. They may want or be offered a greater role in decision-making within the family.[127,128] These cognitive changes provide increased opportunity for the adolescent to influence the parent-child relationship, viewing the relationship within reciprocal terms, potentially realigning the hierarchal structure of the relationship established during

childhood.[126] Additionally, adolescents may begin to reinterpret previous social conventions held by parents, viewing them as subjective rather than absolute. This may bring about tensions in the relationship as parents and adolescents struggle to define and agree upon rules and acceptable behaviors governed by the morals and beliefs of each.[127,129,130] Physical maturation affects the way adolescents view themselves and how others, including their parents, treat them. Hormonal fluctuations caused by puberty necessitate recalibration of relationships to respond to adolescents' heightened emotional states.[127] Such pubertal changes may result in increased emotional distance between parent and child.

Instability in the parent-child bond may occur as both parties seek to find appropriate levels of independence. As a result, closeness to parents diminishes across adolescence, as does the interdependence of parents and children.[124,126,131] Rather than an abrupt decline, closeness to parents gradually decreases.[132] High-quality relationships built on strong bonds, however, may change little across adolescence as both parties adapt to changing circumstances and individual development in a manner that is mutually beneficial.[81,126] The stability of parental closeness is paramount to the influence of parental behaviors on adolescent behavior. This holds true in the reverse as well, closeness underlies the influence of adolescent actions on parental behavior.

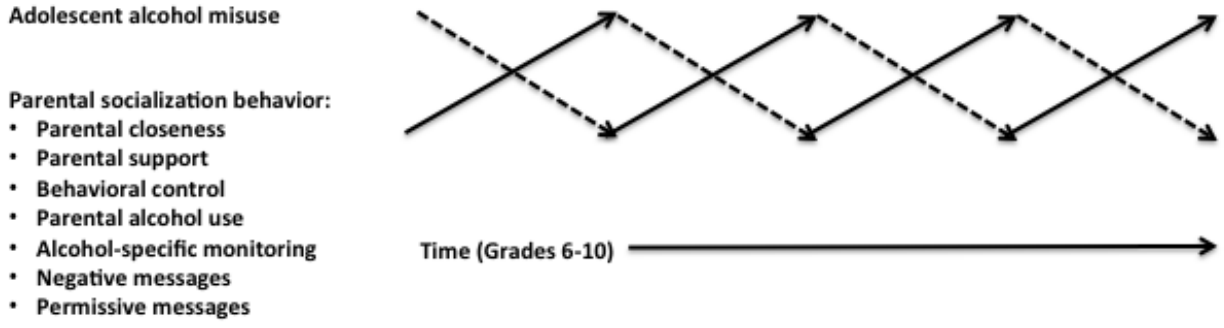
In addition to the changing dynamics of the parent-child relationship, adolescence also brings a shift in importance of peer relationships.[133-135] It is well documented that adolescents have greater involvement and more intense relationships with their peers as they progress through adolescence.[115,136-138] Greater involvement with peers is important in that it often occurs simultaneously with decreased parental involvement and relationship quality.[115,133,139] The relative importance of peer relationships during adolescence is beyond the purview of this study; however, specific elements are significant with regard to alcohol misuse. Foremost, tensions in the parent-adolescent relationship, whether due to changes in closeness, disagreements regarding control or other factors, increases the likelihood the adolescent engages with deviant peers.[6,140,141] According to primary socialization theory, weak familial bonds increase an adolescent's risk of bonding with a deviant peer cluster and engaging in deviant behaviors.[43] Association with deviant peers remains one of the strongest predictors for alcohol use and misuse in adolescence.[82,110,142-144]

In summary, physical, cognitive and emotional development during adolescence impacts the strength and nature of the relationships teenagers have with their parents. This maturation has implications for the interactions between adolescents and parents, and consequently the behaviors of each. To address development change, this study assesses the relationship between parental socialization behaviors and adolescent alcohol misuse at multiple time points across early and middle adolescence.

Conceptual Model

My dissertation extends previous research on parental socialization and adolescent alcohol use by examining reciprocal relations between seven parental socialization behaviors and the particularly consequential behavior of adolescent alcohol misuse. I test bidirectional pathways between three well-documented general parenting behaviors (parental closeness, support and behavioral control), three less studied aspects of alcohol-specific parenting (alcohol-specific monitoring, negative communication messages about alcohol and permissive communication messages about alcohol) and parental alcohol use. Additionally, I assess how the reciprocal relations between each parenting behavior and alcohol misuse unfold across early and middle adolescence. The conceptual model for this research is presented in Figure 1.

Figure 1: Bidirectional relationships of child effects on parent behavior (dashed lines) and parent effects on child behavior (solid lines)



Aims and Hypotheses

Aim 1: To understand bidirectional mechanisms of influence between parental socialization behaviors and adolescent alcohol misuse.

- [Parent effects on adolescent] At times when the parent exhibits weaker socialization behaviors (H1: lower parental closeness, H2: lower parental support, H3: lower behavioral control, H4: higher alcohol use, H5: lower alcohol-specific monitoring, H6: higher permissive messages about alcohol, and H7: lower negative messages about alcohol), the adolescent will subsequently misuse alcohol more than is typical for him/her.
- [Adolescent effects on parent] At times when the adolescent misuses alcohol more than his or her typical level, the parent will subsequently exhibit weaker socialization behaviors (H8: lower parental closeness, H9: lower parental support, H10: lower behavioral control, H11: higher alcohol use, H12: lower alcohol-specific monitoring, H13: higher permissive messages about alcohol, and H14: lower negative messages about alcohol).

Aim 2: To assess whether bidirectional influences between parental socialization behaviors and adolescent alcohol misuse change from early through middle adolescence.

- [Parent effects on adolescent]: The magnitude of the effect of parent H15: closeness, H16: support, H17: behavioral control, H18: alcohol use, H19: alcohol-specific monitoring, H20: permissive messages, and H21: negative messages on adolescent alcohol misuse will decrease from 6-10th grade.
- [Adolescent effects on parent]: The magnitude of the effect of adolescent alcohol misuse on parent H22: closeness, H23: support, H24: behavioral control, H25: alcohol use, H26: alcohol-specific monitoring, H27: permissive messages and H28: negative messages will differ from 6-10th grade.

CHAPTER 3: METHODS

Participants

Data for this study are from the Context of Adolescent Substance Use (Context Study, R01 DA13459, PI: S. Ennett) and Violence against Peers, Dates, and Self: A Developmental Focus (Linkage Study, CDC R49 CCV423114, PI: V. Foshee) that together supported Context/Linkages, a longitudinal study of adolescent health risk behaviors. The study was designed to examine the development of adolescent health risk behaviors, and the individual and contextual factors that contribute to their development.

Context/Linkages used a cohort sequential design that included seven waves of data collection from three cohorts of adolescents in three non-metropolitan counties in North Carolina; the three cohorts were enrolled in grades 6, 7, and 8 at baseline. Targeted schools for Context/Linkages were all public schools that contained grades 6-8, as well as the high schools they fed into, in the three counties. At baseline, all adolescents enrolled in grades 6-8 were eligible for the study except for those who could not complete the survey in English, were in self-contained special education classrooms, or were out of school for long-term suspension. Analyses for this study included data from Waves 1-5. Data collection for Waves 1-5 took place every spring and fall beginning in spring 2002. The study also included a parent sample that was a simple random sample of parents of adolescents who completed the adolescent questionnaire at Wave 1. Additional eligibility required parents to have only one child in the school-based study and to be able to complete the interview in English. By design, for 98% of the cases, the mother or mother surrogate was the parent interviewed. Parents completed annual telephone interviews at Waves 1, 3, and 5.

Data from adolescents were collected through a self-administered paper questionnaire facilitated by study staff during school hours in classrooms or other designated school locations such as the cafeteria. Adolescents whose parents refused study participation were released from the classroom and sent to a pre-specified location. Trained data collectors followed a written protocol to describe the study,

obtain assent and provide directions on completing the questionnaire. Those adolescents who refused assent were excused. Adolescents completed the questionnaire in approximately one hour. To maintain order, teachers remained in classrooms but did not walk around the classroom or ask any questions regarding the study. Students were spread across the classroom and instructed not to talk with one another to ensure privacy. Once completed, students placed the questionnaires in envelopes before returning them to data collectors. Study staff returned to the schools as many as four additional days beyond the primary data collection day in attempt to reach absent adolescents. Trained data collectors conducted the parent interviews via telephone, each of which lasted approximately 25 minutes. The Institutional Review Board at the University of North Carolina at Chapel Hill approved all study protocols.

The core sample, used in these analyses, is the subsample of parent-adolescent dyads identified in Wave 1. At Wave 1, 2215 parents were eligible for data collection, which 79.8% completed the Wave 1 interview (N=1663). All adolescents of these parents completed the Wave 1 questionnaire. Pairs were excluded if 1) the metric of time for this study (grade) could not be confirmed for the adolescent (N=15) or 2) adolescents were missing data for alcohol use across all waves of data collection (N=3), resulting in an analytical sample of 1645 parent-adolescent dyads. Response rates for adolescents and parents in the core sample across Waves 1-5 are presented in Table 1.

Table 1: Context/Linkages study design across waves 1-5

	Wave 1 Spring 2002 6,7,8	**Wave 2 Fall 2002 7,8,9**	**Wave 3 Spring 2003 7,8,9**	**Wave 4 Fall 2003 8,9,10**	**Wave 5 Spring 2004 8,9,10**
Core Sample Adolescent Survey	N=1663 (100%)	N=1377 (82.8%)	N=1417 (85.2%)	N=1331 (80.0%)	N=1265 (76.1%)
Parent Interview	N=1663 (79.8%)		N=1372 (82.5%)		N=1194 (71.8%)

Approximately 48% of the adolescent sample was male and 57% self-reported as White, 36% Black and 7% other races (Hispanic/Latino, American Indian/Native American, Asian/Pacific Islander, multiracial, other). 40% of adolescents reported the highest education for either parent was high school or less, and 19% of adolescents reported living in a household in which there was only one parent at any

wave. At baseline, 50% of the adolescent sample had ever had alcohol while 27% had consumed alcohol in the past 30 days.

Measures

Primary measures for this study included adolescent alcohol misuse and seven parental socialization behaviors, three general parental socialization behaviors (closeness, support and behavioral control), three alcohol-specific parental socialization behaviors (alcohol-specific monitoring, permissive messages about alcohol, negative messages about alcohol) and parental alcohol use. The measure for adolescent alcohol misuse was based on adolescent self-report. All measures of parental socialization behaviors were derived from parental report with the referent being the responding parent only. The adolescent and parent outcomes measures were identically constructed at each wave of data collection. Covariate demographic and behavioral measures were generated from adolescent or parent responses, as appropriate, across all waves of data collection, described in detail below. As applicable, Cronbach's alpha averaged across waves is provided.

Adolescent Alcohol Misuse. This outcome was measured by an existing variable that was constructed using item response theory (IRT).[145] Adolescents responded to eight questions related to recent alcohol use, measuring both problematic levels and negative consequences of alcohol use (e.g. 'how many times have you had 5 or more drinks in a row', 'gotten into a sexual situation that you later regretted because you had been drinking', 'gotten into a physical fight because you had been drinking'). Responses fell into five categories ranging from 0 to 10 or more occurrences in the past 3 months. IRT[146] methods were used to account for the skewed distribution of the responses. One observation per respondent was randomly chosen from all repeated measures to ensure independent cases in constructing the IRT scores. A nominal, one-factor model was most appropriate for the data. IRT scale scores were computed for all participants using the item parameter estimates, resulting in a continuous distribution with lesser skewness and kurtosis than a scale derived from summary scores.

Parental Closeness. Parents were asked 'how close do you feel toward (name)' and 'how close do you think (name) feels towards you?'. Response options were very close (4), somewhat close (3), not very close (2) or not close at all (1). Parents were also asked 'how often do you kiss and hug (name)'.

Response options were often (4), sometimes (3), rarely (2) or never (1). The three items were averaged to create a composite parental closeness score (average Cronbach's α=.69).

Parental Support. Parents were asked in reference to their child, how often they 'make (name) feel better when he/she is upset', 'tell (name) they did a good job on things', and 'want to hear about his/her problems'. Response options were often (4), sometimes (3), rarely (2) or never (1). The three items were averaged to create a composite parental support score. The resulting variable was highly skewed (skew=-3.836); therefore the variable was transformed to the exponential value for analyses to reduce skewness (average Cronbach's α=.54). The items used in this measure of parental support have been used in a similar study elsewhere.[90]

Parental Behavioral Control. Parents were asked in reference to their child, how often they 'tell him/her what time to come home', 'have rules he/she must follow', 'makes sure he/she doesn't stay up too late', 'monitor what he/she watches on tv', and 'put restrictions on music or videogames he/she can play'. Response options were often (4), sometimes (3), rarely (2) or never (1). The five items were averaged to create a composite behavioral control score (average Cronbach's α=.62). The items used in this measure of behavioral control have been used in a similar study, though with additional items included.[90]

Parental Alcohol Use. Parents were asked how many days in the past three months they had one or more drinks of alcohol. Response options ranged from 1=less than one day a month to 6=almost every day. Parents were also asked how much they drank on those days they did drink. Response options ranged from 1=1 drink to 5=5 or more drinks. The two items were multiplied to create a parental alcohol use score.

Parental Alcohol-Specific Monitoring. Parents were asked in reference to their child, whether they had 'done anything to discourage him/her from drinking', 'checked his/her room or other places for evidence of tobacco, alcohol or other drug use' and 'looked for signs that he/she might have smoked or used other kinds of tobacco, drank, or used marijuana or other drugs'. Response options were yes (1) or no (0). The three items were averaged to create an alcohol-specific monitoring score (average Cronbach's α=.57).

Parental Permissive Messages. Parents were asked whether they had told their child 'if he/she ever wants to try a drink, he/she can have a few sips at home in front of you', 'under certain circumstances it's okay to have a few sips of a drink, like with parents or special family occasions', and 'drinking in moderation is okay'. Response options were yes (1) or no (0). The three items were averaged to create a permissive messages score (average Cronbach's α= 0.64).[102]

Parental Negative Messages. Parents were asked whether they had told their child 'drinking is not healthy', 'drinking can lead to alcoholism', 'drinking can cause loss of control', 'he/she cannot ride with someone who has been drinking', 'he/she cannot drink and drive when old enough to drive', and 'he/she should call home to be picked up if he/she does drink'. Response options were yes (1) or no (0). The six items were averaged to create a negative messages score (average Cronbach's α= 0.77).[102]

Covariates. An average measure of each parental socialization behavior across time points was constructed and included as a time-invariant control in models of all other socialization behaviors. Adolescent-reported sex was included, with females defined as the reference category. Race/ethnicity was based on the modal response reported by adolescents across all waves of data collection and dummy coded as White (reference), Black, or Other race/ethnicity. Parent-reported parental education consisted of six categories ranging from less than high school to graduate school or more, and was assessed as the highest level of education attained by either parent across all waves of data collection. Adolescent-reported family structure was coded as either two parents in the household (biological or not) as the referent or other family structure. Age was included as a covariate. Additionally, a variable was created to control for whether the responding parent changed across any wave of assessment.

Missing Data

All analyses used maximum likelihood estimation (MLE) techniques, which utilize all available data, and can be used under the assumption that missing data are missing at random (MAR). In this case, data on the outcomes of interest (alcohol misuse and parental socialization behaviors) were considered missing at random if the probability of missingness did not depend on the value of the outcome variable, controlling for covariates in the analytic model.[147,148] While there is no way to empirically test the assumption of missingness at random, I explored patterns of missing data by assessing associations between 1) study dropout and observed scores on demographic covariates and

baseline alcohol misuse for adolescents and 2) study dropout and demographic covariates and baseline socialization behaviors for parents. Dropout was coded as '1' for adolescents or parents who did not have complete data, and '0' for those with complete data. In multivariate analyses, the association between dropout and baseline alcohol misuse was not significant after controlling for sex, parental education, family structure and race/ethnicity. In multivariate analyses among parents, baseline levels of closeness, support, behavioral control, alcohol-specific monitoring, negative and permissive messages and parental alcohol use were not significantly associated with parental dropout after controlling for demographic covariates. These analyses suggest that study dropout was not related to baseline alcohol misuse or parental socialization variables after controlling for demographic covariates.

Cohort Sequential Design

To capitalize on the cohort sequential design of the study, data were reorganized such that grade level of the child, rather than wave of assessment, was used as the metric of time. This structure allowed variables to be modeled continuously from the spring of 6th grade to the spring of 10th grade, using half-year intervals resulting in nine discrete data points, see Table 2. Cohort sequential designs combine data from multiple, shorter longitudinal datasets into one dataset under the assumption that there is one common growth curve for which the separate cohorts do not differ. I tested for differences in the three cohorts (i.e., grades 6, 7, and 8 at baseline) in growth patterns for adolescent alcohol misuse following established multi-group methods,[149] and found no evidence of cohort differences in the latent trajectory of adolescent alcohol misuse, indicating that it is appropriate to merge the data across cohorts.[150]

Table 2: Cohort sequential design for adolescent and parent data by grade, rather than wave of assessment

	6.5 Spring	7 Fall	7.5 Spring	8 Fall	8.5 Spring	9 Fall	9.5 Spring	10 Fall	10.5 Spring
Adolescent Cohort 1	Wave 1	Wave 2	Wave 3	Wave 4	Wave 5				
Parent Cohort 1	Wave 1		Wave 3		Wave 5				
Adolescent Cohort 2			Wave 1	Wave 2	Wave 3	Wave 4	Wave 5		
Parent Cohort 2			Wave 1		Wave 3		Wave 5		
Adolescent Cohort 3					Wave 1	Wave 2	Wave 3	Wave 4	Wave 5
Parent Cohort 3					Wave 1		Wave 3		Wave 5

*solid lines represent parental effects on child; dashed lines represent child effects on parent

Analytic Approach

The overall goal of this study was to assess bidirectional effects between parental socialization behaviors and adolescent alcohol misuse across grades 6-10. In this way, the purpose of the study is to model dynamic relations that link parental socialization behaviors and adolescent alcohol misuse over time. Such an investigation provides insight as to how these behaviors mutually play out across adolescence. There is more than one way to examine how these relationships develop over time.

The first is how the relationship unfolds across individuals, describing *interindividual* processes. A between-person level of analysis explores interindividual differences, or those differences that exist across persons.[151-154] Generally, between-person effects examine whether individuals who, on average, experience a higher level of one construct tend to behave in a certain way on another construct; or, whether individuals who report a systematic increase in one behavior are more likely to report a systematically higher or lower level of a second, related behavior. For example, on average, adolescents whose parents monitor their activities tend to misuse alcohol less than adolescents whose parents are not monitoring their whereabouts. Therefore, between-person processes provide information on *who* is at risk; specifically, which adolescents are at risk for alcohol misuse based on parental socialization behaviors? And, which parents demonstrate socialization behaviors reflective of teen drinking behaviors?

The second method of examination is how the relationship unfolds within an individual, describing *intraindividual* processes. This component, referred to as the within-person effect, reflects unique components of both the individual and specific points in time, thereby examining differences within the same person across points in time.[151-155] The within-person model assesses whether at times when an individual is above or below his or her own average level of one construct, he or she is above or below their average on a second construct. This model examines whether a time-specific level at one time point is meaningfully related to another time-dependent level at a later point. Thus, with-in person effects examine time-specific deviations relative to an individual's underlying trajectory.

For this study, parental and adolescent behaviors are linked according to time-specific deviations. This provides information on *when* an adolescent is most at risk for alcohol misuse based on their parents' socializing behaviors. Due to the bidirectional nature of the study, the within-person model also explores *when* parents alter socialization behaviors based on their child's alcohol use. As outlined in transactional models of development, this study focuses on such intraindividual effects to explain the relationship between general and alcohol-specific parental socialization behaviors, parental alcohol use and adolescent alcohol misuse. All models control for the between-level effect.

The goal of the analyses was to model the over-time interrelationships between adolescent alcohol misuse and parental socialization behaviors, assessed one at a time, specifically examining the time-specific component of these relations within each adolescent-parent dyad. To simultaneously explore the hypothesized relationships of the two outcome variables over time, I utilized a latent curve model with structured residuals (LCM-SR)[156], an extension of the autoregressive latent trajectory (ALT) model.[157] The approach of the ALT model assumes that each outcome (adolescent alcohol misuse and parental socialization behavior) is dictated by an underlying growth process unique to that outcome. Thus, a separate latent curve model is estimated for each outcome to reflect its distinctive growth process and to align with a developmental perspective that change is a continuous growth process.[158] The latent curve approach assumes that observed repeated measures are generated from this underlying growth trajectory, which is unique to each individual. The parameters (i.e. means and variances) that describe the unobserved (or latent) factors of the growth trajectory (i.e. intercept and slope) are empirically generated from the observed repeated measures.

The ALT model combines the strength of two common methods for the analysis of panel data: the autoregressive model and the random coefficients growth curve model. Doing so allows for the simultaneous assessment of time-stable and time-specific associations between the two outcomes. The time-stable association is reflected through the covariation of the latent factors that govern the underlying developmental process of each outcome. Time-specific relations are assessed by the inclusion of autoregressive paths and prospective cross-lag paths between repeated measures of time-varying outcomes.[157]

The current study specifically examines the within-person processes of the two outcomes, which are the time-specific relationships between adolescent alcohol misuse and parental socialization behavior for each parent-adolescent dyad. For this study, the time-specific relationships include 1) autoregressive stability paths from the observed measure of adolescent alcohol misuse at one time point to the next time point, 2) autoregressive stability paths between repeated measures of the parental socialization behavior at one time point to the next time point , 3) prospective paths from adolescent alcohol misuse at one time point to the parental socialization behavior at the next time point, and 4) prospective paths from parental behavior to the time-adjacent measure of adolescent alcohol misuse. To fully capture these four within-person effects, the between- and within- person effects of the model must be disaggregated.

The latent curve model with structured residuals (LCM-SR) was chosen because of its unique ability to disaggregate between- and within-person effects on the stability and change of the proposed relationship between the variables over time.[152,156] The LCM-SR used in this study deviates from the ALT model in that whereas the ALT model specifies the autoregressive and prospective paths at the level of the observed variables, the LCM-SR does so at the level of the time-specific residuals. Since the ALT model specifies these relations among the manifest variables, it can be interpreted that the repeated measures of one construct are mediators for the influence of the latent curve of that construct on the indicators of the second construct. Because of this, the ALT model does not provide a pure disaggregation of between- and within-person effects. The reparameterization of the LCM-SR allows for such disaggregation, as the inclusion of time-specific regressions among the residuals (the prospective, reciprocal, within-person component of the model) does not influence the fixed-effect characteristics (e.g. mean structure) of the underlying latent curve. The application of regression coefficients at the level of the

time-specific residuals is not a typical parameterization of a structural equation model (SEM); however, this approach has been used previously in SEM analyses and is sometimes referred to as 'phantom variables'. These are latent variables with no indicators used as a methodological means for imposing constraints on parameter estimates.[159,160] With the imposition of a higher order structure among the residuals, the mean structure of the repeated measures is uniquely determined by the latent curve factors while the covariance of the repeated measures is jointly modeled by the latent curve factors as well as the structure of the residuals. This parameterization allows for estimation of unique between and within person components of the model.

I present a series of equations to demonstrate the reconceptualization of the residual structure and function for the LCM-SR[156]. For a univariate LCM with no structured residuals, the residual ($\xi_{y_{it}}$) represents the deviation of the observed measure (y_{it}) from its underlying trajectory:

$$\xi_{y_{it}} = y_{it} - (\alpha_{y_i} + \lambda_t \beta_{y_i})$$

The reparameterization of the residuals in the LCM-SR conceptualizes the residual as a time-specific estimate of the above deviation. Rather than allowing the residuals to covary in an unstructured way, the model defines the regression of a later residual on a prior residual:

$$\xi_{y_{it}} = \rho_{yy}\xi_{y_{i(t-1)}} + \nu_{y_{it}}$$

Where ρ_{yy} is the regression parameter and $\nu_{yit} \sim N(0, \sigma^2_{vy})$. Based on this equation, a later residual is partly determined by a prior residual, beyond the influence of the underlying trajectory.

Given that the goal of the research is to estimate the within-person processes that link two constructs over time, a multivariate model is necessary. The multivariate LCM-SR defines a residual based on the regression of both a prior autoregressive and cross-lagged residual:

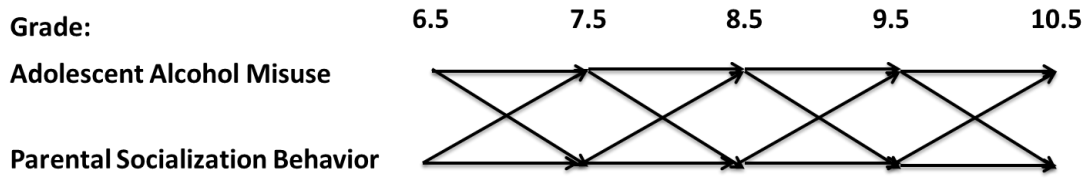
$$\xi_{y_{it}} = \rho_{yy}\xi_{y_{i(t-1)}} + \rho_{yz}\xi_{z_{i(t-1)}} + \nu_{y_{it}}$$

Where ρ_{yy} is the autoregressive regression parameter for the residual at time t on the residual at time t-1 and ρ_{yz} is the similar cross-lagged regression parameter. This residual structure holds for all residuals except for T=1, which differs in that it is not conditioned on a prior residual. A similar equation for the residual of the second construct in the multivariate model can be expressed as:

$$\xi_{z_{it}} = \rho_{zz}\xi_{z_{i(t-1)}} + \rho_{zy}\xi_{y_{i(t-1)}} + \nu_{z_{it}}$$

Due to model complexity, a separate model was estimated for the relation between each parental socialization behavior and adolescent alcohol misuse, controlling for all other socialization behaviors. To assess reciprocal relations within the same time period, autoregressive and cross-lagged paths were incorporated into the LCM-SR according to the time dimensions depicted in Figure 2.

Figure 2: Analytical time dimensions for autoregressive and cross-lagged paths



For nested models, I used a chi-square difference test to determine improvement in model fit. The comparative fit index (CFI), Tucker-Lewis Index (TLI) and the root mean square error of approximation (RMSEA) were used to assess model fit for all latent curves.[158] Good fit was denoted as indices greater than 0.95 for the CFI and TLI statistics and less than 0.05 for the RMSEA. Unstandardized parameter coefficients are presented throughout the results and discussion. Statistical significance is denoted at the following alpha levels: +p<.10, **p<.05, ***p<.01. I used the model building approach described below to assess increasingly complex models to test study hypotheses.

Step 1- Univariate LCM-SR for adolescent alcohol misuse:

The first step was to estimate an unconditional model for adolescent alcohol misuse to determine the functional form of time as well as test for autoregressions among the residuals and equality restraints among those autoregressions. I first tested a random intercept model, which included only a mean and variance of the intercept factor and residual variances for each repeated measure, allowed to vary across time. I then added a linear slope factor, which included a mean and variance for the intercept and slope factors, as well as an intercept-slope covariance, while maintaining freely varying time-specific residuals. If the addition of the linear slope significantly improved model fit, I tested the addition of a quadratic slope factor, again including a mean and variance for the quadratic growth factor and covariances with the intercept and linear slope factor. Only the growth factors that resulted in a significant improvement to model fit were retained. Next, an autoregressive path (AR) among the residuals was added, held to be equal over time. If the addition of equal autoregressive paths resulted in a significant improvement in

model fit, I allowed the autoregressive paths to vary across time, and tested that inclusion with a nested chi-square difference test. Finally, I generated a conditional univariate model for alcohol misuse by regressing the latent curve factors on the time-stable average of each parental socialization behavior and the demographic covariates (sex, age, race/ethnicity, parental education, family structure, responding parent). The best fitting model was chosen based on parsimony, theoretical inclusion of components and overall fit.

Step 2- Univariate LCM-SR for parental socialization behavior:

I repeated the process described in Step 1 to generate the unconditional univariate curve for each parental socialization behavior. To construct a conditional model for a focal parental behavior I regressed the latent factors for the parental socialization univariate curve on all demographic covariates and each of the other averaged parental behaviors. The best fitting model was chosen based on parsimony, theoretical inclusion of components and overall fit.

Step 3- Bivariate LCM-SR for parental behavior and adolescent alcohol misuse, no cross lags

Next, I combined the two univariate LCMs into a single bivariate LCM. To do so, I allowed the latent factors from each univariate curve to covary with each other. I included the autoregressive components among the residuals for adolescent alcohol misuse and parental behavior, retaining the structure of the autoregressive paths identified in Steps 1 and 2; however, I did not include any prospective paths between the repeated measures of the two outcomes. I allowed the time-specific residuals to covary between alcohol misuse and parental behavior, and set these covariances to be equal across times 2-5. I then tested this equality constraint by allowing the covariance between the time-specific residuals to freely vary across time and assessing whether the model was significantly improved by freeing the equality constraint.

Step 4- Bivariate LCM-SR for parental behavior and adolescent alcohol misuse with parent on child cross lags

In the fourth step, I tested the inclusion of prospective relations between adolescent alcohol misuse at time T and the parental socialization behavior at time T+1, holding all prospective relations from parental behavior to adolescent alcohol use at zero. First, I retained the bivariate structure from Step 3 and added the cross-lagged paths from alcohol misuse to parental behavior, holding the paths to be equal across time. Because the final bivariate model from Step 3 and this model with equal prospective

paths are nested, I used a chi-square difference test to assess significant improvement in model fit. Next, I tested whether the magnitude of these relations changed over time by applying a model constraint such that each sequential cross-lag changed by a fixed amount and tested this model in comparison to the previous model in which the prospective relations were held to be equal. I assessed the direction of the new lag parameter generated by the model constraint as well as the significance of the prospective path between each subsequent cross lag to determine whether the prospective paths systematically changed over time. Finally, I allowed the prospective relations to freely vary and tested improvement in model fit.

Step 5- Bivariate LCM-SR for parental behavior and adolescent alcohol misuse with child on parent cross lags

I then removed the prospective relations from child to parent and repeated the same process outlined in Step 4 for the relation between the parental behavior at time T to adolescent alcohol misuse at time T+1. First, I tested a model with equal prospective relations, comparing it to the final bivariate model in Step 3. Then I imposed a model constraint to test for a systematic change in the magnitude of the prospective effect over time. Finally, I allowed the prospective relations to freely vary over time and tested improvement in model fit.

Step 6- Combined bivariate LCM-SR for parental behavior and adolescent alcohol misuse, all cross lags

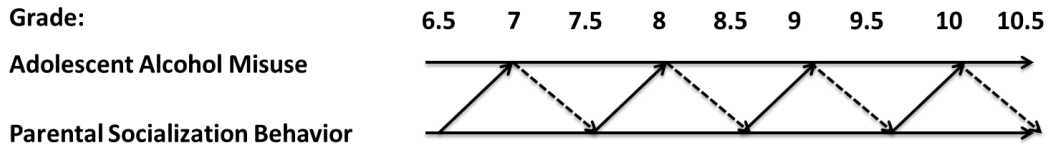
The last step in the model building approach was to combine the bivariate structure from Step 3 with the prospective relations from child to parent identified in Step 4 and the prospective relations from parent to child identified in Step 5. I determined the results of study hypotheses from this model, assessing the significance of the autoregressive and prospective reciprocal paths between the constructs, as well as variation in the magnitude of the reciprocal relation over time.

Sensitivity Analyses: Cross-Lag Time Interval

As currently arranged according to Figure 2, reciprocal relationships are examined within the same time frame (1 year). This data structure was chosen to make inferences regarding the magnitude of the reciprocal effects within a given developmental time period (i.e. assessing both child influence on parental behavior and parental influence on child behavior from time T to T+1). The lag time between subsequent time points needed to be one year to account for the data collection schedule in which adolescents were assessed every six months while parents were assessed annually. Sensitivity analyses were conducted by repeating the same model building process, altering the lag between time points to be

six months rather than one year. To do this, however, reciprocal relationships cannot be assessed in the same time frame. Figure 3 depicts the time relations for the sensitivity analyses. Data from Wave 6 was included in this analysis to include an additional repeated measure of adolescent alcohol misuse.

Figure 3: Alternative analytical time dimensions for autoregressive and cross-lagged paths



Despite the mismatch of assessment intervals for adolescents and parents, the sensitivity analysis was added to provide insight into whether a six-month versus one-year time interval was more informative for assessing prospective reciprocal paths.

Sensitivity Analyses: Comparison of Adolescent Versus Parental Report of Parental Socialization Behaviors

Parent reports of socialization behaviors were used in the current study to maintain informant consistency across parental socialization behaviors given that all three alcohol-specific socialization behaviors (alcohol-specific monitoring, permissive and negative messages) were only available through parent report. The use of parent-reported behavior is less common than in most research, which uses adolescent report of parental behavior. Implications for each informant are discussed below.

Parents may over-report certain behaviors due to social desirability bias, or the desire to respond in a manner perceived as preferable by society. This may be especially true for general parenting behaviors as they are widely discussed and highly regarded as central parenting practices for a variety of adolescent outcomes, including alcohol use. Parents may respond more positively to questions regarding support, closeness, control and their own alcohol use than is actually the case, thus biasing the measurement of these constructs. Several empirical studies have assessed the correspondence between parental and adolescent reports of behavior to assess these types of biases. In regards to alcohol use, both parents and children underestimate consumption of alcohol by the other, particularly the quantity of alcohol consumed. Parents more accurately estimate when their child is not drinking rather than when they are drinking.[161] Further, parents are less aware of substance use problems as compared to use.[162]

Examining parental socialization behaviors and adolescent drinking, Latendresse et al[163] found that profiles of parenting behaviors derived from adolescent report were more predictive of adolescent drinking than comparable profiles derived from parental report of their own behavior; however, the degree to which the two profiles provided unique explanations of variance in adolescent drinking was not decipherable given the methods used in the study. Adolescent perceptions of their parents' behavior may reflect the saliency of that behavior for the adolescent, which may have important implications for how the parent behavior truly affects adolescent behavior. On the other hand, adolescent report of both independent and dependent variables results in shared measurement variance. Data from adolescent report of parental behaviors were available for the general socialization behaviors (closeness, support and behavioral control) and parental alcohol use. I repeated the analyses described above with these four adolescent-reported parental socialization behaviors.

Perceived parental closeness: At each wave, adolescents were asked 'how close do you feel toward your mother/father' and 'how close do you think your mother/father feels towards you?'. Response options were very close (4), somewhat close (3), not very close (2) or not close at all (1). Adolescents were also asked 'how often does your mother/father kiss and hug you?'. Response options were often (4), sometimes (3), rarely (2) or never (1). The three items regarding the responding parent were averaged to create a composite perceived parental closeness score (average Cronbach's α=.80).

Perceived parental support: At each wave, adolescents were asked how often their responding parent 'makes me feel better when I am upset', 'tells me I did a good job on things', 'wants to hear about my problems'. Response options were often (4), sometimes (3), rarely (2) or never (1). The three items were averaged to create a composite perceived parental support score (average Cronbach's α=.86).

Perceived behavioral control: At each wave, adolescents were asked how often their responding parent 'has rules they must follow', 'tells me times I must come home in the evenings' and 'makes sure I don't stay up too late'. Response options were 'just like him/her ' (4), 'a lot like him/her ' (3), 'somewhat like him/her ' (2), and 'not at all like him/her ' (1). The three items were averaged to create a composite perceived parental behavioral control score (average Cronbach's α=.80).

Perceived parental alcohol use: At each wave, adolescents were asked regarding the responding parent ‘about how many days a week do you think she/he drinks?’. Response options were none (1), 1-2 days (2), 3-4 days (3) 5-7 days (4).

CHAPTER 4: RESULTS

Tables 3-9 provide the means, standard deviations and correlations between repeated measures of adolescent alcohol misuse and parental closeness, support, behavioral control, parental alcohol use, alcohol-specific monitoring, permissive messages, and negative messages, respectively. Because of the differences in starting grade, the descriptive statistics are presented for each cohort of students in the sample. Overall, mean scores of alcohol misuse increased across grades. Mean values for parental closeness and behavioral control decreased slightly across grades, support remained relatively consistent, and alcohol-specific monitoring, permissive messages and negative messages slightly increased over time. Parental alcohol use scores were not consistent across cohort and grade level. Across grades and cohorts, the correlations between parental socialization behaviors and adolescent alcohol misuse were low (<0.20), though some were significant at $p<.05$ even with the low correlation value.

Table 3: Parental closeness and adolescent alcohol misuse correlations by grade level and cohort

Cohort 1 (N=589)							
Outcome	**Grade**	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	6.5	1.00					
2. Alcohol misuse	7.5	0.19*	1.00				
3. Alcohol misuse	8.5	0.12*	0.23*	1.00			
4. Closeness	6.5	-0.07	-0.01	0.00	1.00		
5. Closeness	7.5	-0.13*	0.01	-0.03	0.60*	1.00	
6. Closeness	8.5	-0.08	0.02	-0.02	0.58*	0.70*	1.00
Mean		-0.10	0.24	0.48	3.85	3.81	3.80
SD		0.31	0.48	0.58	0.30	0.36	0.35
Cohort 2 (N=537)							
	Grade	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	7.5	1.00					
2. Alcohol misuse	8.5	0.31*	1.00				
3. Alcohol misuse	9.5	0.16*	0.38*	1.00			
4. Closeness	7.5	-0.14*	-0.11*	-0.09	1.00		
5. Closeness	8.5	-0.10	-0.09	0.03	0.56*	1.00	
6. Closeness	9.5	-0.19*	-0.02	0.03	0.55*	0.65*	1.00
Mean		-0.01	0.33	0.58	3.84	3.79	3.79
SD		0.52	0.59	0.68	0.30	0.34	0.35
Cohort 3 (N=519)							
	Grade	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	8.5	1.00					
2. Alcohol misuse	9.5	0.31*	1.00				
3. Alcohol misuse	10.5	0.11*	0.37*	1.00			
4. Closeness	8.5	-0.07	0.06	-0.05	1.00		
5. Closeness	9.5	-0.12*	-0.05	-0.06	0.66*	1.00	
6. Closeness	10.5	-0.01	0.04	-0.02	0.67*	0.68*	1.00
Mean		0.08	0.47	0.70	3.80	3.78	3.75
SD		0.64	0.73	0.74	0.35	0.39	0.41

*p<.05

Table 4: Parental support and adolescent alcohol misuse correlations by grade level and cohort

Cohort 1 (N=589)							
Outcome	**Grade**	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	6.5	1.00					
2. Alcohol misuse	7.5	0.19*	1.00				
3. Alcohol misuse	8.5	0.12*	0.23*	1.00			
4. Support	6.5	-0.02	0.06	-0.03	1.00		
5. Support	7.5	-0.02	0.04	0.03	0.40*	1.00	
6. Support	8.5	-0.02	-0.03	0.01	0.44*	0.58*	1.00
Mean		-0.10	0.24	0.48	3.91	3.91	3.94
SD		0.31	0.48	0.58	0.26	0.26	0.17
Cohort 2 (N=537)							
	Grade	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	7.5	1.00					
2. Alcohol misuse	8.5	0.31*	1.00				
3. Alcohol misuse	9.5	0.16*	0.38*	1.00			
4. Support	7.5	-0.03	-0.03	-0.11*	1.00		
5. Support	8.5	-0.08	-0.06	-0.03	0.44*	1.00	
6. Support	9.5	-0.08	-0.06	-0.02	0.38*	0.47*	1.00
Mean		-0.01	0.33	0.58	3.91	3.92	3.91
SD		0.52	0.59	0.68	0.22	0.21	0.21
Cohort 3 (N=519)							
	Grade	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	8.5	1.00					
2. Alcohol misuse	9.5	0.31*	1.00				
3. Alcohol misuse	10.5	0.11*	0.37*	1.00			
4. Support	8.5	-0.07	0.07	0.00	1.00		
5. Support	9.5	-0.04	-0.01	-0.02	0.37*	1.00	
6. Support	10.5	0.01	0.10	0.01	0.49*	0.51*	1.00
Mean		0.08	0.47	0.70	3.90	3.91	3.89
SD		0.64	0.73	0.74	0.23	0.21	0.24

*p<.05

Table 5: Parental behavioral control and adolescent alcohol misuse correlations by grade level and cohort

Cohort 1 (N=589)							
Outcome	**Grade**	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	6.5	1.00					
2. Alcohol misuse	7.5	0.19*	1.00				
3. Alcohol misuse	8.5	0.12*	0.23*	1.00			
4. Behavioral control	6.5	-0.05	0.01	0.09	1.00		
5. Behavioral control	7.5	-0.03	-0.01	-0.08	0.37*	1.00	
6. Behavioral control	8.5	-0.08	0.00	0.02	0.46*	0.48*	1.00
Mean		-0.10	0.24	0.48	3.72	3.79	3.70
SD		0.31	0.48	0.58	0.35	0.34	0.37
Cohort 2 (N=537)							
	Grade	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	7.5	1.00					
2. Alcohol misuse	8.5	0.31*	1.00				
3. Alcohol misuse	9.5	0.16*	0.38*	1.00			
4. Behavioral control	7.5	0.02	-0.04	-0.06	1.00		
5. Behavioral control	8.5	0.00	0.01	0.05	0.36*	1.00	
6. Behavioral control	9.5	0.01	-0.02	0.00	0.45*	0.34*	1.00
Mean		-0.01	0.33	0.58	3.71	3.80	3.61
SD		0.52	0.59	0.68	0.38	0.36	0.47
Cohort 3 (N=519)							
	Grade	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	8.5	1.00					
2. Alcohol misuse	9.5	0.31*	1.00				
3. Alcohol misuse	10.5	0.11*	0.37*	1.00			
4. Behavioral control	8.5	-0.01	0.03	-0.01	1.00		
5. Behavioral control	9.5	-0.09	-0.13*	-0.08	0.52*	1.00	
6. Behavioral control	10.5	0.01	-0.01	0.07	0.49*	0.58*	1.00
Mean		0.08	0.47	0.70	3.66	3.68	3.54
SD		0.64	0.73	0.74	0.42	0.43	0.53

*p<.05

Table 6: Parental alcohol use and adolescent alcohol misuse correlations by grade level and cohort

Cohort 1 (N=589)							
Outcome	**Grade**	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	6.5	1.00					
2. Alcohol misuse	7.5	0.19*	1.00				
3. Alcohol misuse	8.5	0.12*	0.23*	1.00			
4. Parental alcohol use	6.5	0.12	0.03	-0.02	1.00		
5. Parental alcohol use	7.5	0.09	0.04	-0.07	0.55*	1.00	
6. Parental alcohol use	8.5	0.11*	0.11	0.04	0.54*	0.55*	1.00
Mean		-0.10	0.24	0.48	2.52	2.36	2.69
SD		0.31	0.48	0.58	4.39	4.02	4.55
Cohort 2 (N=537)							
	Grade	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	7.5	1.00					
2. Alcohol misuse	8.5	0.31*	1.00				
3. Alcohol misuse	9.5	0.16*	0.38*	1.00			
4. Parental alcohol use	7.5	0.02	0.11*	0.15*	1.00		
5. Parental alcohol use	8.5	-0.07	0.06	0.09	0.64*	1.00	
6. Parental alcohol use	9.5	-0.00	0.00	0.11*	0.55*	0.56*	1.00
Mean		-0.01	0.33	0.58	2.85	2.31	2.63
SD		0.52	0.59	0.68	4.52	3.90	4.30
Cohort 3 (N=519)							
	Grade	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	8.5	1.00					
2. Alcohol misuse	9.5	0.31*	1.00				
3. Alcohol misuse	10.5	0.11*	0.37*	1.00			
4. Parental alcohol use	8.5	0.10*	-0.00	0.03	1.00		
5. Parental alcohol use	9.5	0.03	0.06	0.08	0.54	1.00	
6. Parental alcohol use	10.5	0.08	0.17*	0.12	0.59	0.55	1.00
Mean		0.08	0.47	0.70	2.68	2.66	3.24
SD		0.64	0.73	0.74	4.52	4.22	4.58

*p<.05

Table 7: Parental alcohol-specific monitoring and adolescent alcohol misuse correlations by grade level and cohort

Cohort 1 (N=589)							
Outcome	**Grade**	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	6.5	1.00					
2. Alcohol misuse	7.5	0.19*	1.00				
3. Alcohol misuse	8.5	0.12*	0.23*	1.00			
4. Alcohol-specific monitoring	6.5	0.13*	0.04	0.07	1.00		
5. Alcohol-specific monitoring	7.5	0.14*	0.05	0.12*	0.51*	1.00	
6. Alcohol-specific monitoring	8.5	0.14*	-0.01	0.13*	0.49*	0.51*	1.00
Mean		-0.10	0.24	0.48	0.31	0.33	0.34
SD		0.31	0.48	0.58	0.34	0.35	0.35
Cohort 2 (N=537)							
	Grade	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	7.5	1.00					
2. Alcohol misuse	8.5	0.31*	1.00				
3. Alcohol misuse	9.5	0.16*	0.38*	1.00			
4. Alcohol-specific monitoring	7.5	0.03	0.04	-0.03	1.00		
5. Alcohol-specific monitoring	8.5	0.09	0.10	0.01	0.48*	1.00	
6. Alcohol-specific monitoring	9.5	0.18*	0.15*	0.08	0.50*	0.48*	1.00
Mean		-0.01	0.33	0.58	0.37	0.36	0.41
SD		0.52	0.59	0.68	0.35	0.35	0.37
Cohort 3 (N=519)							
	Grade	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	8.5	1.00					
2. Alcohol misuse	9.5	0.31*	1.00				
3. Alcohol misuse	10.5	0.11*	0.37*	1.00			
4. Alcohol-specific monitoring	8.5	0.17*	0.05	-0.01	1.00		
5. Alcohol-specific monitoring	9.5	0.07	0.07	0.18*	0.47*	1.00	
6. Alcohol-specific monitoring	10.5	0.07	0.13*	0.23*	0.46*	0.52*	1.00
Mean		0.08	0.47	0.70	0.34	0.36	0.39
SD		0.64	0.73	0.74	0.36	0.36	0.36

*p<.05

Table 8: Parental permissive messages and adolescent alcohol misuse correlations by grade level and cohort

Cohort 1 (N=589)							
Outcome	**Grade**	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	6.5	1.00					
2. Alcohol misuse	7.5	0.19*	1.00				
3. Alcohol misuse	8.5	0.12*	0.23*	1.00			
4. Permissive messages	6.5	-0.01	0.13*	-0.03	1.00		
5. Permissive messages	7.5	0.07	0.08	-0.09	0.70*	1.00	
6. Permissive messages	8.5	0.01	-0.00	0.00	0.63*	0.73*	1.00
Mean		-0.10	0.24	0.48	0.17	0.22	0.22
SD		0.31	0.48	0.58	0.27	0.31	0.30
Cohort 2 (N=537)							
	Grade	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	7.5	1.00					
2. Alcohol misuse	8.5	0.31*	1.00				
3. Alcohol misuse	9.5	0.16*	0.38*	1.00			
4. Permissive messages	7.5	0.03	0.04	0.07	1.00		
5. Permissive messages	8.5	0.06	0.08	0.10	0.72*	1.00	
6. Permissive messages	9.5	-0.02	0.02	0.09	0.67*	0.73*	1.00
Mean		-0.01	0.33	0.58	0.22	0.26	0.27
SD		0.52	0.59	0.68	0.31	0.33	0.33
Cohort 3 (N=519)							
	Grade	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	8.5	1.00					
2. Alcohol misuse	9.5	0.31*	1.00				
3. Alcohol misuse	10.5	0.11*	0.37*	1.00			
4. Permissive messages	8.5	0.08	0.07	0.13*	1.00		
5. Permissive messages	9.5	0.05	0.08	0.20*	0.66*	1.00	
6. Permissive messages	10.5	0.09	0.17*	0.23*	0.66*	0.74*	1.00
Mean		0.08	0.47	0.70	0.21	0.25	0.27
SD		0.64	0.73	0.74	0.32	0.33	0.35

*p<.05

Table 9: Parental negative messages and adolescent alcohol misuse correlations by grade level and cohort

Cohort 1 (N=589)							
Outcome	**Grade**	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	6.5	1.00					
2. Alcohol misuse	7.5	0.19*	1.00				
3. Alcohol misuse	8.5	0.12*	0.23*	1.00			
4. Negative messages	6.5	-0.01	0.04	0.06	1.00		
5. Negative messages	7.5	0.06	0.01	0.09	0.61*	1.00	
6. Negative messages	8.5	0.01	0.03	0.10	0.50*	0.62*	1.00
Mean		-0.10	0.24	0.48	0.84	0.85	0.87
SD		0.31	0.48	0.58	0.25	0.23	0.23
Cohort 2 (N=537)							
	Grade	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	7.5	1.00					
2. Alcohol misuse	8.5	0.31*	1.00				
3. Alcohol misuse	9.5	0.16*	0.38*	1.00			
4. Negative messages	7.5	-0.01	0.10*	0.06	1.00		
5. Negative messages	8.5	0.04	0.07	0.04	0.62*	1.00	
6. Negative messages	9.5	0.06	0.05	0.09	0.53*	0.72*	1.00
Mean		-0.01	0.33	0.58	0.85	0.88	0.87
SD		0.52	0.59	0.68	0.24	0.22	0.22
Cohort 3 (N=519)							
	Grade	**1.**	**2.**	**3.**	**4.**	**5.**	**6.**
1. Alcohol misuse	8.5	1.00					
2. Alcohol misuse	9.5	0.31*	1.00				
3. Alcohol misuse	10.5	0.11*	0.37*	1.00			
4. Negative messages	8.5	0.04	0.03	0.08	1.00		
5. Negative messages	9.5	0.04	0.04	0.02	0.59*	1.00	
6. Negative messages	10.5	0.05	0.01	0.06	0.51*	0.65*	1.00
Mean		0.08	0.47	0.70	0.85	0.89	0.91
SD		0.64	0.73	0.74	0.23	0.20	0.19

*p<.05

Adolescent Alcohol Misuse

Univariate LCM-SR for Adolescent Alcohol Misuse

I first tested a random intercept-only model that included a mean and variance of the intercept factor and residual variances for each of the repeated measures of adolescent alcohol misuse, which were allowed to vary over time. This model fit the data poorly ($\chi^2(11)=901.58$; CFI=0.00, TLI=-1.93, RMSEA=.22). I then added a random linear slope factor, estimating a mean and variance for the intercept and linear slope factor as well as a covariance between the two latent factors. A random slope could not be estimated; therefore I fixed the linear slope variance and covariance to zero. The model fit to the data was poor ($\chi^2(10)=96.86$; CFI=.61; TLI=.69, RMSEA=.07), yet the addition of the linear slope relative to the

intercept-only model was significant ($\Delta\chi^2(1)=804.72$; $p<.01$) and therefore was retained. Next, I added a quadratic growth factor including a mean and variance for the intercept, linear and quadratic growth factors and covariances between all latent factors. This model could not be estimated.

The estimated means for the retained latent factors indicate the model-implied mean trajectory for the sample was characterized by an initial adolescent alcohol misuse score of -.14 ($p<.01$) and a significant positive linear growth factor ($b=.23$, $p<.01$). Thus, the average developmental trajectory of adolescent alcohol misuse is increasing over time. In addition to these fixed effects, there was significant individual variability around the initial level of alcohol misuse ($b=.07$, $p<.01$).

Next, I added autoregressive paths among the residuals of the repeated measures of alcohol misuse, setting these paths to be equal over time. Overall, the fit of this model to the data was excellent ($\chi^2(9)=10.17$; CFI=1.00, TLI=1.00, RMSEA=.01). The inclusion of the autoregressive paths significantly improved model fit relative to the intercept and linear slope model ($\Delta\chi^2(1)=86.69$, $p<.01$). Next, I allowed the autoregressive paths to vary across time, but this did not lead to significant improvement in model fit ($\Delta\chi^2(3)=2.20$, $p=.53$), therefore I retained the model with equal autoregressive paths across time. Finally, I regressed the latent factors on the average parental socialization behavior and demographic covariates. I retained those covariates in which the regression parameter was significantly different from zero (intercept: average closeness, average alcohol-specific monitoring, age, parental education; slope: average permissive messages, family structure, age, race/ethnicity). A summary of the model building process for the univariate LCM-SR for adolescent alcohol misuse is presented in Table 10. This univariate LCM-SR for alcohol misuse was used in all subsequent bivariate models with each parental socialization behavior.

Table 10: Model building process for univariate LCM-SR for adolescent alcohol misuse

	χ^2 (df)	Comparison Model	$\Delta\chi^2$ (df)	CFI	TLI	RMSEA
Step 1: Univariate LCM for adolescent alcohol misuse						
1. Random intercept	901.58 (11)			0.00	-1.93	0.22
2. 1 + fixed linear slope	96.86 (10)	1	804.72 (1)***	0.61	0.69	0.07
3. 2 + quadratic slope	--					
Latent Factor		*Mean*	*Variance*			
Intercept		-.14***	.07***			
Linear slope		.23***	n/a			
4. 2+ AR paths equal	10.17 (9)	2	86.69 (1)***	1.00	1.00	0.01
5. 2+ AR paths free	7.97 (6)	4	2.20 (3)	0.99	0.99	0.01
Final model Step 1: 4 + covariates Intercept: average closeness, average alcohol-specific monitoring, age, parental education Slope: average permissive messages, family structure, age, race						

AR= autoregressive paths; +p<.10, **p<.05,***p<.01

Parental Closeness

Univariate LCM-SR for Parental Closeness

I first estimated a random intercept model that included a mean and variance of the intercept factor and residual variances for each of the repeated measures of closeness, which were allowed to vary over time (the freely-varying time-specific residuals were kept throughout the model-building process). This model fit the data moderately well ($\chi^2(11)=105.74$; CFI=.92, TLI=.94, RMSEA=.07). Next, I added a random linear slope factor to the model, estimating a mean and variance for the intercept and slope factors as well as a covariance between the two. These additions resulted in significant improvement to model fit relative to the intercept-only model ($\Delta\chi^2(3)=101.15$, p<.01), and the model itself was an excellent fit to the data ($\chi^2(8)=4.59$; CFI=1.00, TLI=1.00, RMSEA=.00). Next, I added a quadratic growth factor including a mean and variance for the intercept, linear and quadratic growth factors and covariances between all latent factors. This did not result in significant improvement to model fit relative to the intercept-linear slope model ($\Delta\chi^2(4)=2.32$, p=.68), and was not retained.

The estimated means for the retained latent factors indicate the model-implied mean trajectory for the sample was characterized by an initial parental closeness score of 3.86 (p<.01) and a significant negative linear growth factor (b=-.03, p<.01). Thus, the average developmental trajectory of parental closeness is decreasing over time. In addition to these fixed effects, there was significant individual variability around the initial level of parental closeness (b=.06, p<.01), but not in rates of change of

parental closeness over time. However, while the linear slope variance and intercept-slope covariance were not significantly different from zero, constraining the slope variance and the intercept-slope covariance to zero led to a significant decrement in model fit ($\Delta\chi^2(2)=53.31$, $p<.01$), thus the parameters were retained in the model.

I extended the intercept-linear slope model to include autoregressive paths between the residuals of the repeated measures of parental closeness, setting them to be equal over time. This model could not be estimated. I freed the equality constraint on the autoregressive paths, and this model fit the data well ($\chi^2(4)=3.67$; CFI=.99, TLI=.98, RMSEA=.04). The addition of the freely varying autoregressive parameters did not significantly improve model fit relative to the intercept-linear slope model ($\Delta\chi^2(4)=.92$,$p=.92$); however, I retained the freely varying autoregressive paths because they are theoretically hypothesized to exist. Finally, I regressed the latent factors on the average parental socialization behavior and demographic covariates. I retained those in which the regression parameter was significantly different from zero (intercept: average support, average negative messages; slope: average behavioral control).

Bivariate LCM-SR for Parental Closeness and Adolescent Alcohol Misuse

I then combined the univariate LCM-SRs for adolescent alcohol misuse and parental closeness into a single bivariate LCM-SR. I allowed the intercept and slope factors for parental closeness to covary with each other as well as with the intercept factor for alcohol misuse. I allowed the time-specific residuals to covary with each other and set the covariances to be equal across Times 2-5. I removed the regression of the intercept factor for alcohol misuse on the average parental closeness covariate for this series of bivariate LCM-SRs. This model fit the data well ($\chi^2(110)=128.17$; CFI=.99, TLI=.99, RMSEA=.01). Next, I allowed the time-specific residuals to freely covary across time. This did not lead to significant improvement in model fit ($\Delta\chi^2(3)=.10$, $p=.99$), and was not retained. I then tested the inclusion of prospective relations between the constructs in multiple steps. First, I added the regression of parental closeness on adolescent alcohol misuse, holding the regression of alcohol misuse on parental closeness to zero. I constrained the prospective regressions to be equal across time. While the model itself fit the data well ($\chi^2(109)=128.10$; CFI=.99, TLI=.99, RMSEA=.01), the inclusion of the prospective regressions of parental closeness on alcohol misuse did not significantly improve model fit relative to the bivariate model with no cross lags ($\Delta\chi^2(1)=.07$, $p=.80$), and thus were not retained. Next, I added the prospective

regressions of adolescent alcohol misuse on parental closeness, holding the regression of closeness on adolescent alcohol misuse to zero. Similarly, this model fit the data well ($\chi^2(109)=127.65$; CFI=.99, TLI=.99, RMSEA=.01) but did not significantly improve model fit relative to the bivariate model with no cross lags ($\Delta\chi^2(1)=.52$, p=.47) and therefore was not retained.

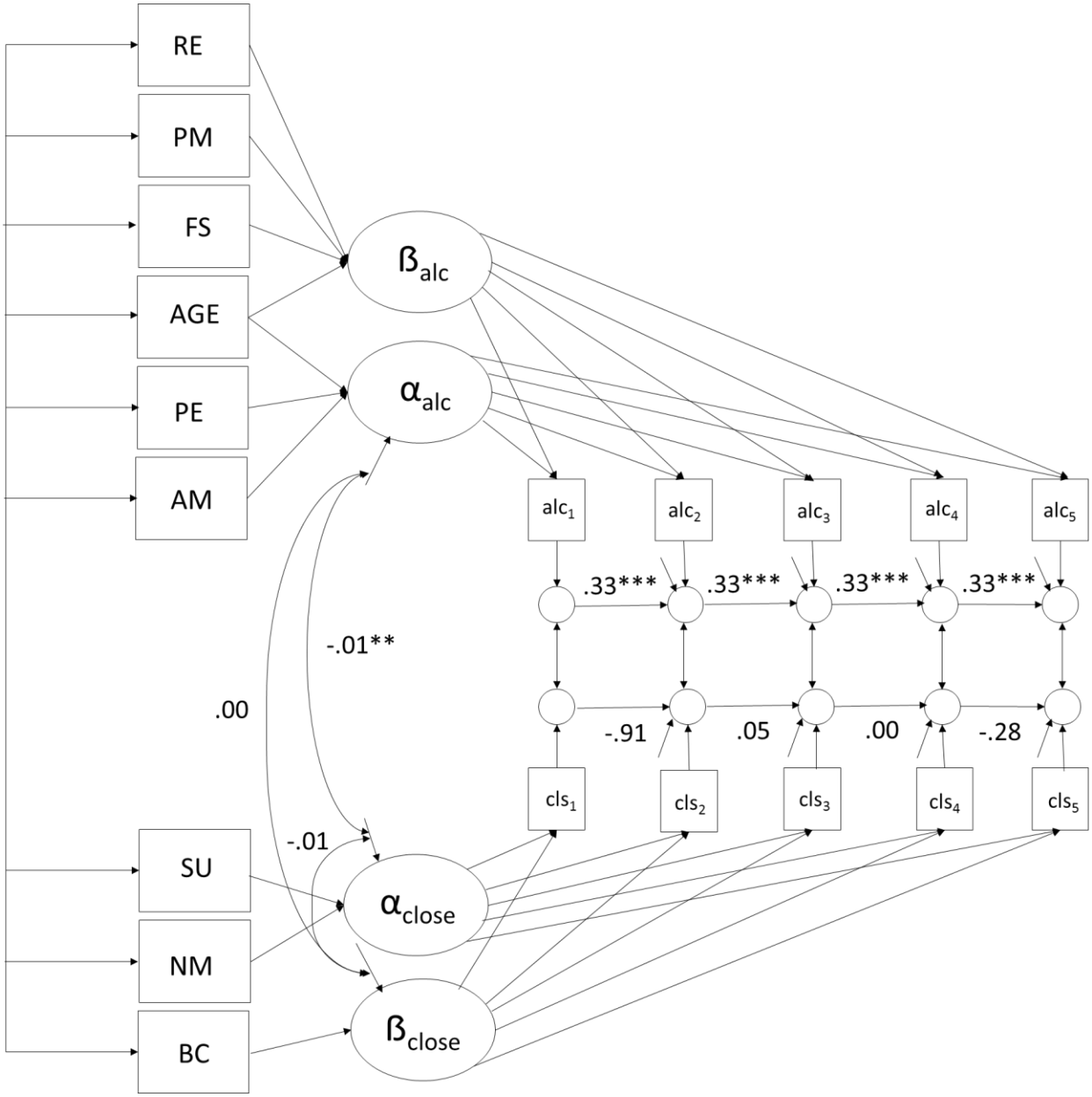
A summary of the model building process for the bivariate LCM-SR for parental closeness and adolescent alcohol misuse is provided in Table 11. Figure 4 presents the results of the final bivariate model of parental closeness and adolescent alcohol misuse for which the autoregressive paths among the residuals for adolescent alcohol misuse are significant (b=.33, p<.01) but the autoregressive paths among the residuals for parental closeness are not significantly different from zero. Additionally, the covariance between the latent intercept factors was negative and statistically significant (b=-.01, p<.05); however, the covariances between slope of parental closeness with intercepts of closeness and alcohol misuse were not significantly different from zero. Given that the prospective cross-lagged regressions were not retained in either direction, hypotheses related to the presence of reciprocal effects between parental closeness and adolescent alcohol misuse (H1 and H8) as well as to changes in the magnitude of those reciprocal effects (H15 and H22) were not supported.

Table 11: Model-building process for bivariate LCM-SR between parental closeness and adolescent alcohol misuse

	χ^2 (df)	Comparison Model	$\Delta\chi^2$ (df)	CFI	TLI	RMSEA
Step 2: Univariate LCM for closeness						
6. Random intercept	105.74 (11)***			0.92	0.94	0.07
7. 6 + random linear slope	4.59 (8)	6	101.15 (3)***	1.00	1.00	0.00
8. 7+ random quadratic slope	2.27 (4)	7	2.32 (4)	1.00	1.00	0.00
Latent Factor	*Mean*	*Variance*				
Intercept	3.86***	0.06***				
Linear slope	-.03***	.00				
9. 7+ AR paths equal	--					
10. 7+ AR paths free	3.67 (4)	7	.92 (4)	0.99	0.98	0.04
Final model Step 2: 10 + covariates Intercept: average support, average negative messages Slope: average behavioral control						
Step 3: Bivariate LCM-SR for closeness and adolescent alcohol misuse, no cross lags						
11. Residual covariances equal	**128.17 (110)**			**0.99**	**0.99**	**0.01**
12. Residual covariances free	128.07(107)	11	.10 (3)	0.99	0.99	0.01
Step 4- Bivariate LCM-SR for closeness and adolescent alcohol misuse with parent on child cross lags						
13. 11 + cross lags equal	128.10 (109)	11	.07 (1)	0.99	0.99	0.01
Prospective regressions	*Beta*	*SE*	*p-value*			
P2 on C1	.00	.02	.80			
P3 on C2	.00	.02	.80			
P4 on C3	.00	.02	.80			
P5 on C4	.00	.02	.80			
Step 5- Bivariate LCM-SR for closeness and adolescent alcohol misuse with child on parent cross lags						
14. 11 + cross lags equal	127.65 (109)	11	.52 (1)	0.99	0.99	0.01
Prospective regressions	*Beta*	*SE*	*p-value*			
C2 on P1	.07	.09	.47			
C3 on P2	.07	.09	.47			
C4 on P3	.07	.09	.47			
C5 on P4	.07	.09	.47			

AR= autoregressive paths; +p<.10, **p<.05,***p<.01; **Bold=final model**

Figure 4: Final model for bivariate LCM-SR between parental closeness and adolescent alcohol misuse



+p<.10, **p<.05,***p<.01; RE=race/ethnicity; PM= average permissive messages; FS= family structure; PE=parental education; AM= average alcohol-specific monitoring; SU= average parental support; NM= average negative messages; BC= average behavioral control

Parental Support

Univariate LCM-SR for Parental Support

I began by estimating a random intercept-only model that included a mean and variance of the intercept factor and residual variances for each of the repeated measures of support, which were allowed to vary over time (the freely-varying time-specific residuals were maintained throughout the model-building process). This model fit the data very well ($\chi^2(11)=17.04$; CFI=.99, TLI=.99; RMSEA=.02). I extended this model with the addition of a random linear slope by estimating a mean and variance for the intercept and slope factor as well as a covariance between the two latent factors. While this model also fit the data well ($\chi^2(8)=14.24$; CFI=.99, TLI=.99; RMSEA=.02), the inclusion of the linear slope factor did not significantly improve model fit relative to the intercept-only model ($\Delta \chi^2(3)=2.80$, p=.42), and therefore was not retained. The model-implied mean trajectory for the sample was characterized by an initial parental support score of 50.89 (p<.01), for which there was significant individual variability around that mean (b=31.50, p<.01).

I extended the intercept-only model to include autoregressive paths between the residuals of the repeated measures of parental support, setting them to be equal over time. Overall, this model fit the data well ($\chi^2(10)=16.75$; CFI=.99, TLI=.99; RMSEA=.02). The addition of the equal autoregressive parameters did not significantly improve model fit relative to the intercept-only model ($\Delta\chi^2(1)=.29$,p=.59); however, I retained the autoregressive paths because they are theoretically hypothesized to exist. Finally, I regressed the latent factors on the average parental socialization behavior and demographic covariates. I retained those in which the regression parameter was significantly different from zero (intercept: average closeness, average behavioral control, average negative messages, parental education, race/ethnicity).

Bivariate LCM-SR for Parental Support and Adolescent Alcohol Misuse

Next, I combined the univariate LCM-SRs for adolescent alcohol misuse and parental support into a bivariate LCM-SR. I allowed the intercept factors for parental support and adolescent alcohol misuse to covary. I allowed the time-specific residuals to covary with each other and set the covariances to be equal across Times 2-5. This model fit the data well ($\chi^2(114)=129.93$; CFI=.99, TLI=.99, RMSEA=.01). Next, I allowed the time-specific residuals to freely covary across time. This did not lead to significant improvement in model fit ($\Delta\chi^2(3)=3.00$, p=.39), and was not retained. Following the steps outlined

previously, I tested the inclusion of prospective relations between the constructs over time. First, I added the regression of parental support on adolescent alcohol misuse, holding the regression of alcohol misuse on parental support to zero and constraining the prospective regressions to be equal across time. While the model itself fit the data well ($\chi^2(113)=129.46$; CFI=.99, TLI=.99, RMSEA=.01), the inclusion of the prospective regressions of parental support on alcohol misuse did not significantly improve model fit relative to the bivariate model with no cross lags ($\Delta\chi^2(1)=.47$, p=.49), and thus were not retained. Next, I added the regressions of adolescent alcohol misuse on parental support, holding the regression of support on alcohol misuse to zero. Again, this model fit the data well ($\chi^2(113)=127.43$; CFI=.99, TLI=.99, RMSEA=.01) but did not significantly improve model fit relative to the bivariate model with no cross lags ($\Delta\chi^2(1)=2.50$, p=.11) and therefore was not retained.

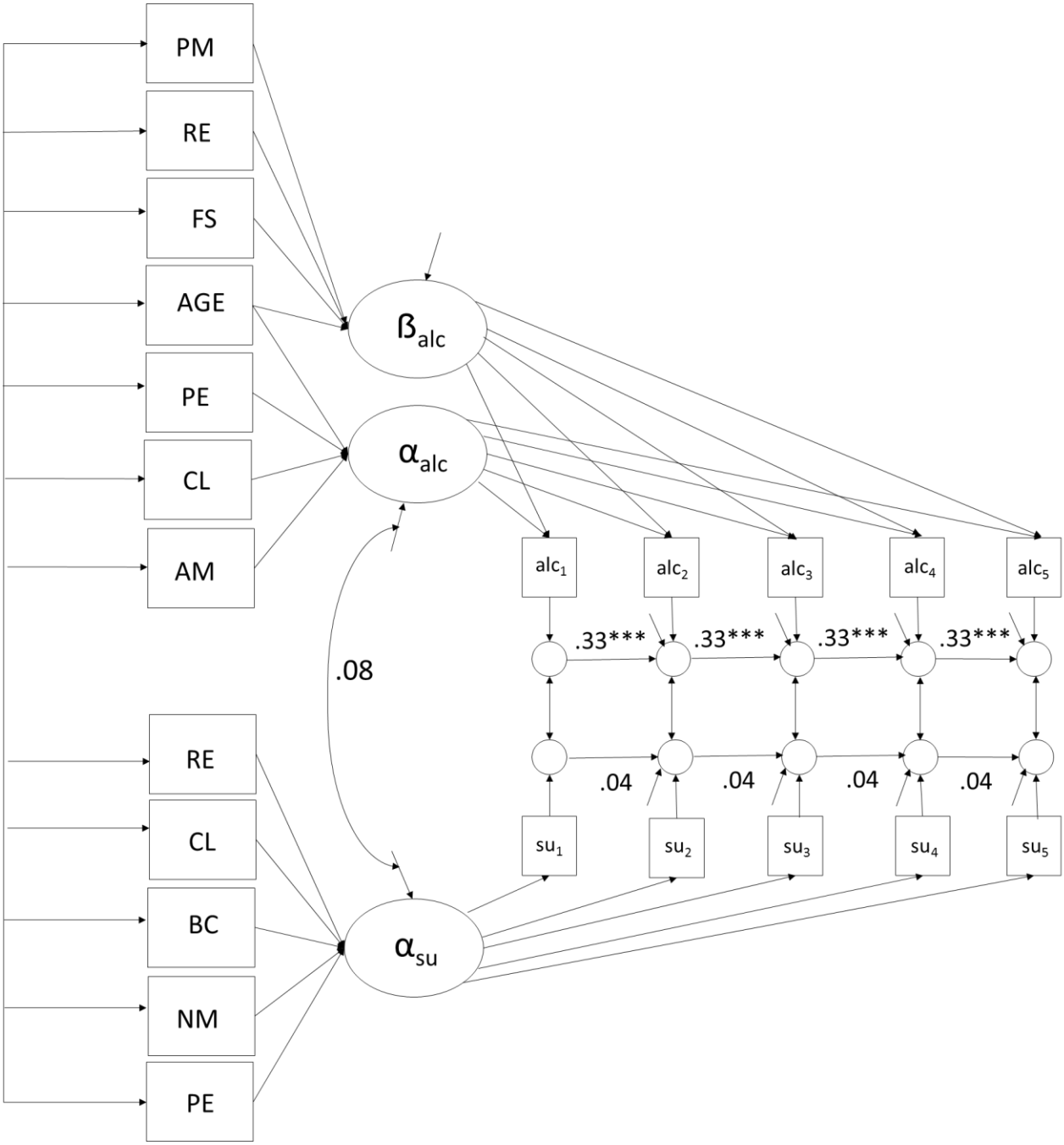
A summary of the model building process for the bivariate LCM-SR for parental support and adolescent alcohol misuse is provided in Table 12. Figure 5 presents the results of the final bivariate model for parental support and adolescent alcohol misuse for which the autoregressive paths among the residuals for adolescent alcohol misuse are significant (b=.33, p<.01) but the autoregressive paths among the residuals for parental support are not significantly different from zero. The covariance between the latent intercept factors is not statistically different from zero. Given that the prospective cross-lagged regressions were not retained in either direction, hypotheses related to the presence of reciprocal effects between parental support and adolescent alcohol misuse (H2 and H9) as well as to changes in the magnitude of those reciprocal effects (H16 and H23) were not supported.

Table 12: Model-building process for bivariate LCM-SR between parental support and adolescent alcohol misuse

	χ^2 (df)	Comparison Model	$\Delta\chi^2$ (df)	CFI	TLI	RMSEA
Step 2: Univariate LCM for support						
6. Random intercept	17.04 (11)			0.99	0.99	0.02
7. 6 + random linear slope	14.24 (8)	6	2.80 (3)	0.99	0.99	0.02
Latent Factor	*Mean*	*Variance*				
Intercept	50.89***	31.50***				
8. 6 + AR paths equal	16.75 (10)	6	.29 (1)	0.99	0.99	0.02
Final model Step 2: 8 + covariates Intercept: average closeness, average behavioral control, average negative messages, parental education, race						
Step 3: Bivariate LCM-SR for support and adolescent alcohol misuse, no cross lags						
9. Residual covariances equal	**129.93 (114)**			**0.99**	**0.99**	**0.01**
10. Residual covariances free	126.93 (111)	9	3.00 (3)	0.99	0.99	0.01
Step 4- Bivariate LCM-SR for support and adolescent alcohol misuse with parent on child cross lags						
11. 9 + cross lags equal	129.46 (113)	9	.47 (1)	0.99	0.99	0.01
Prospective regressions	*Beta*	*SE*	*p-value*			
P2 on C1	-.26	.38	.49			
P3 on C2	-.26	.38	.49			
P4 on C3	-.26	.38	.49			
P5 on C4	-.26	.38	.49			
Step 5- Bivariate LCM-SR for support and adolescent alcohol misuse with child on parent cross lags						
12. 9 + cross lags equal	127.43 (113)	9	2.50 (1)	0.99	0.99	0.01
Prospective regressions	*Beta*	*SE*	*p-value*			
C2 on P1	.00	.00	.11			
C3 on P2	.00	.00	.11			
C4 on P3	.00	.00	.11			
C5 on P4	.00	.00	.11			

AR= autoregressive paths; +p<.10, **p<.05,***p<.01; **Bold=final model**

Figure 5: Final model for bivariate LCM-SR between parental support and adolescent alcohol misuse



+p<.10, **p<.05,***p<.01; RE=race/ethnicity; PM= average permissive messages; FS= family structure; PE=parental education; AM= average alcohol-specific monitoring; NM= average negative messages; BC= average behavioral control; CL=average parental closeness

Parental Behavioral Control

Univariate LCM-SR for Parental Behavioral Control

I first estimated a random intercept-only model including a mean and variance for the intercept factor and residual variances for each of the repeated measures of behavioral control, which were allowed to vary over time (the freely-varying time-specific residuals were kept throughout the model-building process). The fit of the model to the data was moderate ($\chi^2(11)=97.49$, CFI=.84, TLI=.88, RMSEA=.07). Next, I added a random linear slope to the model and estimated a mean and variance for the intercept and slope as well as a covariance between the two latent factors. This model would not properly estimate due to the linear slope factor. The mean for the linear slope factor was significantly different from zero (b=-.03, p<.01) yet the variance of the slope factor was not (b=0.00, p=.96). I restricted the variance and covariance of the linear slope factor to zero, resulting in a model which also had moderate fit to the data ($\chi^2(10)=68.79$, CFI=.89, TLI=.91, RMSEA=.06). The addition of the fixed linear slope resulted in significant improvement to model fit relative to the intercept-only model ($\Delta\chi^2(1)=28.70$; p<.01) and was retained. I extended this model by adding a quadratic growth factor, estimating a mean and variance for each latent factor, and a covariance between the intercept and quadratic factor. Overall, this model fit the data very well ($\chi^2(7)=7.92$, CFI=.99, TLI=.99, RMSEA=.01) and was a significant improvement to model fit relative to the intercept-linear slope model ($\Delta\chi^2(3)=60.87$; p<.01); therefore, the quadratic growth factor was retained.

The estimated means for the latent factors indicate that the model-implied mean trajectory for the sample was characterized by an initial parental behavioral control score of 3.71 (p<.01), a significant positive linear growth component (b=0.04, p<.01), and a significant negative quadratic growth component (b=-0.02, p<.01). Taken together, these results reflect that the average developmental trajectory of parental behavioral control is increasing over time and that the magnitude of change decreases at later grades. In addition to these significant fixed effects, the latent factor variance estimates for the model indicate that there was substantial individual variability in initial levels of parental behavioral control (b=0.05, p<.01), but not for rates of change in behavioral control over time. However, while the quadratic slope variance and intercept-slope covariance were not significantly different from zero, constraining the

quadratic slope variance and covariances to zero led to a significant decrement in model fit ($\Delta\chi^2(2)=28.50$, $p<.01$), thus the parameters were retained in the model.

Next I added autoregressive parameters among the residuals to the model, restricted to be equal over time. The addition of the equal autoregressive parameters did not significantly improve model fit relative to the quadratic model ($\Delta\chi^2(1)=3.40$,$p=.10$); however, I retained the autoregressive paths because they are theoretically hypothesized to exist. Finally, I regressed the latent factors on the average parental socialization behavior and demographic covariates. I retained those in which the regression parameter was significantly different from zero (intercept: average support, average alcohol-specific monitoring; quadratic slope: average alcohol-specific monitoring).

Bivariate LCM-SR for Parental Behavioral Control and Adolescent Alcohol Misuse

Next, I combined the univariate LCM-SRs for adolescent alcohol misuse and parental behavioral control into a single bivariate LCM-SR. I allowed the intercept factor for behavioral control to covary with both the quadratic growth factor and the intercept for alcohol misuse as well as a covariation between the quadratic growth factor for behavioral control and the intercept for alcohol misuse. I allowed the time-specific residuals to covary with each other and set the covariances to be equal across Times 2-5. This model fit the data moderately well ($\chi^2(101)=205.12$; CFI=.91, TLI=.90, RMSEA=.03). Next, I allowed the time-specific residuals to freely covary across time. This did not lead to significant improvement in model fit ($\Delta\chi^2(3)=6.47$, $p=.10$), and was not retained. Following the steps outlined previously, I tested the inclusion of prospective relations between the constructs over time. First, I added the regression of behavioral control on adolescent alcohol misuse, holding the regression of alcohol misuse on behavioral control to zero and constraining the prospective regressions to be equal across time. While the model itself fit the data moderately well ($\chi^2(100)=204.58$; CFI=.91, TLI=.90, RMSEA=.03), the inclusion of the prospective regressions of behavioral control on alcohol misuse did not significantly improve model fit relative to the bivariate model with no cross lags ($\Delta\chi^2(1)=.54$, $p=.46$), and thus were not retained. Next, I added the regressions of adolescent alcohol misuse on behavioral control, holding the regression of behavioral control on alcohol misuse to zero. This model fit the data moderately well ($\chi^2(100)=205.06$; CFI=.91, TLI=.89, RMSEA=.03) but did not significantly improve model fit relative to the bivariate model with no cross lags ($\Delta\chi^2(1)=.06$, $p=.86$) and therefore was not retained.

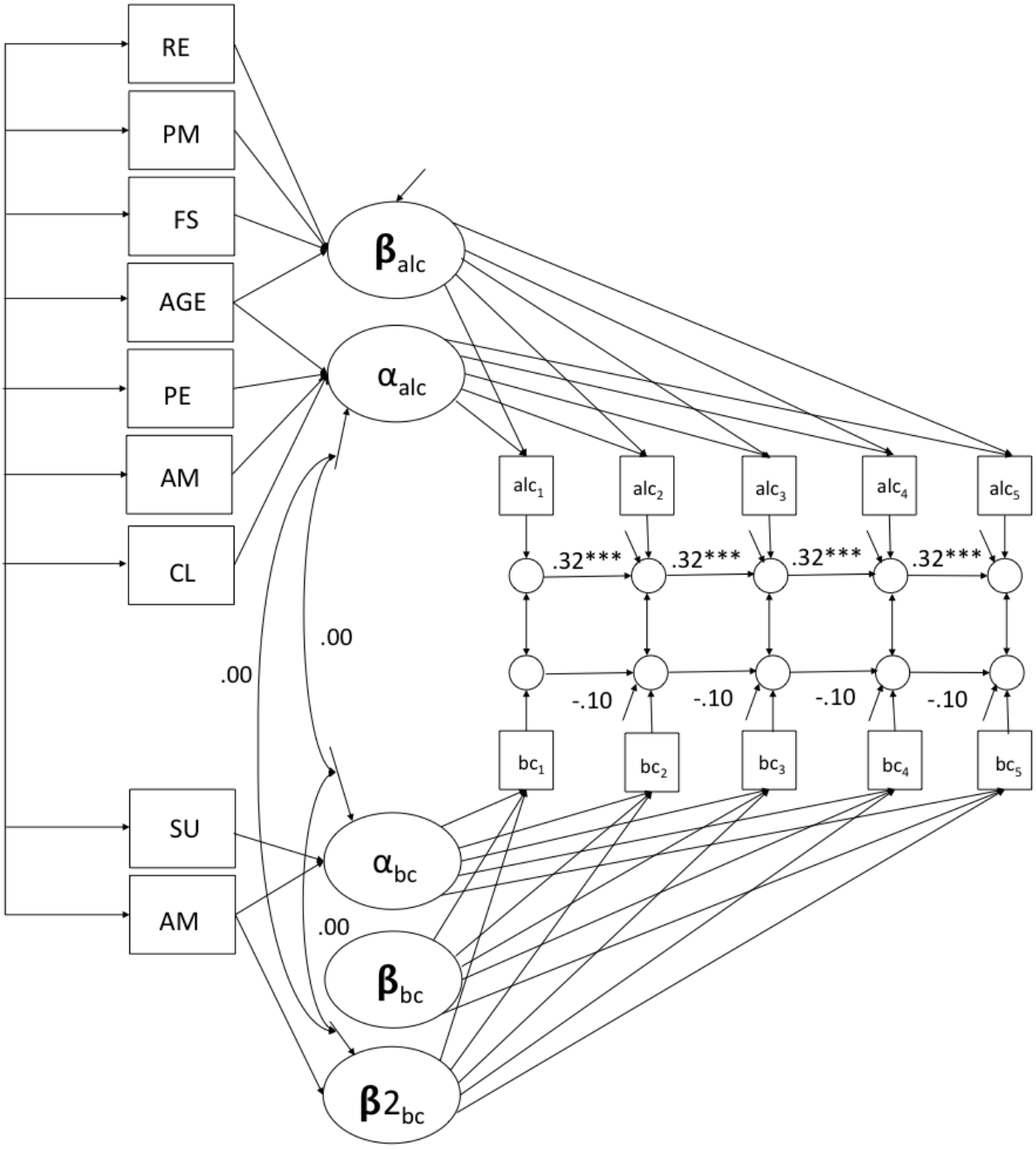
A summary of the model building process for the bivariate LCM-SR for parental behavioral control and adolescent alcohol misuse is provided in Table 13. Figure 6 presents the results of the final bivariate model for parental behavioral control and adolescent alcohol misuse for which the autoregressive paths among the residuals for adolescent alcohol misuse are significant ($b=.32$, $p<.01$), but the autoregressive paths among the residuals for parental behavioral control are not significantly different from zero. None of the covariances between latent factors of the growth curves (intercept of alcohol misuse with intercept and quadratic slope of behavioral control, intercept and quadratic slope of behavioral control) were statistically significant. Given that the prospective cross-lagged regressions were not retained in either direction, hypotheses related to the presence of reciprocal effects between behavioral control and adolescent alcohol misuse (H3 and H10) as well as to changes in the magnitude of those reciprocal effects (H17 and H24) were not supported.

Table 13: Model-building process for bivariate LCM-SR between parental behavioral control and adolescent alcohol misuse

	χ^2 (df)	Comparison Model	$\Delta\chi^2$ (df)	CFI	TLI	RMSEA
Step 2: Univariate LCM for behavioral control						
6. Random Intercept	97.49 (11)			0.84	0.88	0.07
7. 6 + fixed linear slope	68.79 (10)	6	28.70 (1)***	0.89	0.91	0.06
8. 7 + random quadratic slope	7.92 (7)	7	60.87 (3)***	0.99	0.99	0.01
Latent Factor	*Mean*	*Variance*				
Intercept	3.71***	.05***				
Linear slope	.04***	n/a				
Quadratic slope	-.02***	.00				
9. 8 + AR paths equal	4.52 (6)	8	3.40 (1)	1.00	1.00	0.00
Final model Step 2: 9+ covariates Intercept: average support, average alcohol-specific monitoring Quadratic slope: average alcohol-specific monitoring						
Step 3: Bivariate LCM-SR for behavioral control and adolescent alcohol misuse, no cross lags						
10. Residual covariances equal	**205.12 (101)+**			**0.91**	**0.90**	**0.03**
11. Residual covariances free	198.65 (98)	10	6.47 (3)	0.91	0.90	0.03
Step 4- Bivariate LCM-SR for behavioral control and adolescent alcohol misuse with parent on child cross lags						
12. 10 + cross lags equal	204.58 (100)	10	.54 (1)	0.91	0.90	0.03
Prospective regressions	*Beta*	*SE*	*p-value*			
P2 on C1	-.02	.02	.46			
P3 on C2	-.02	.02	.46			
P4 on C3	-.02	.02	.46			
P5 on C4	-.02	.02	.46			
Step 5- Bivariate LCM-SR for behavioral control and adolescent alcohol misuse with child on parent cross lags						
13. 10 + cross lags equal	205.06 (100)	10	.06 (1)	0.91	0.89	0.03
Prospective regressions	*Beta*	*SE*	*p-value*			
C2 on P1	-.02	.06	.80			
C3 on P2	-.02	.06	.80			
C4 on P3	-.02	.06	.80			
C5 on P4	-.02	.06	.80			

AR= autoregressive paths; +p<.10, **p<.05,***p<.01; **Bold=final model**

Figure 6: Final model for bivariate LCM-SR between parental behavioral control and adolescent alcohol misuse



+p<.10, **p<.05,***p<.01; RE=race/ethnicity; PM= average permissive messages; FS= family structure; PE=parental education; AM= average alcohol-specific monitoring; BC= average behavioral control; CL=average parental closeness; SU= average parental support

Parental Alcohol Use

Univariate LCM-SR for Parental Alcohol Use

I began by estimating a random intercept-only model that included a mean and variance of the intercept factor and residual variances for each of the repeated measures of parental alcohol use which were allowed to vary over time (the freely-varying time-specific residuals were kept through the model-building process). This model fit the data very well ($\chi^2(11)=12.34$; CFI=1.00, TLI=1.00; RMSEA=.01). I extended this model with the addition of a random linear slope by estimating a mean and variance for the intercept and slope factor as well as a covariance between the two latent factors. While this model also fit the data well ($\chi^2(8)=10.63$; CFI=1.00, TLI=1.00; RMSEA=.01), the inclusion of the linear slope factor did not significantly improve model fit relative to the intercept-only model ($\Delta \chi^2(3)=1.71$, p=.63), and therefore was not retained. The model-implied mean trajectory for the sample was characterized by an initial parental alcohol use score of 2.63 (p<.01), for which there was significant individual variability around that mean (b=10.76, p<.01).

I extended the intercept-only model to include autoregressive paths between the residuals of the repeated measures of parental alcohol use, setting them to be equal over time. Overall, this model fit the data well ($\chi^2(10)=11.73$; CFI=1.00, TLI=1.00; RMSEA=.01). The addition of the equal autoregressive parameters did not significantly improve model fit relative to the intercept-only slope model ($\Delta\chi^2(1)=.61$,p=.43); however, I retained the autoregressive paths because they are theoretically hypothesized to exist. Finally, I regressed the latent factors on the average parental socialization behavior and demographic covariates. I retained those in which the regression parameter was significantly different from zero (intercept: average behavioral control, average permissive messages, average negative messages, race/ethnicity, sex, family structure).

Bivariate LCM-SR for Parental Alcohol Use and Adolescent Alcohol Misuse

Next, I combined the univariate LCM-SRs for adolescent alcohol misuse and parental alcohol use into a bivariate LCM-SR. I allowed the intercept factors for parental alcohol use and adolescent alcohol misuse to covary. I allowed the time-specific residuals to covary with each other and set the covariances to be equal across Times 2-5. This model fit the data well ($\chi^2(123)=128.19$; CFI=1.00, TLI=1.00,

RMSEA=.01). Next, I allowed the time-specific residuals to freely covary across time. This did not lead to significant improvement in model fit ($\Delta\chi^2(3)=3.65$, p=.30), and was not retained.

Following the steps outlined previously, I tested the inclusion of prospective relations between the constructs over time. First, I added the regression of parental alcohol use on adolescent alcohol misuse, holding the regression of alcohol misuse on parental alcohol use to zero and constraining the prospective regressions to be equal across time. While the model itself fit the data well ($\chi^2(122)=127.32$; CFI=1.00, TLI=1.00, RMSEA=.01), the inclusion of the prospective regressions of parental alcohol use on alcohol misuse did not significantly improve model fit relative to the bivariate model with no cross lags ($\Delta\chi^2(1)=.87$, p=.35), and thus were not retained. Next, I added the regressions of adolescent alcohol misuse on parental alcohol use, holding the regression of parental alcohol use on alcohol misuse to zero. Again, this model fit the data well ($\chi^2(122)=127.06$; CFI=1.00, TLI=1.00, RMSEA=.01) but did not significantly improve model fit relative to the bivariate model with no cross lags ($\Delta\chi^2(1)=1.13$, p=.29) and therefore was not retained.

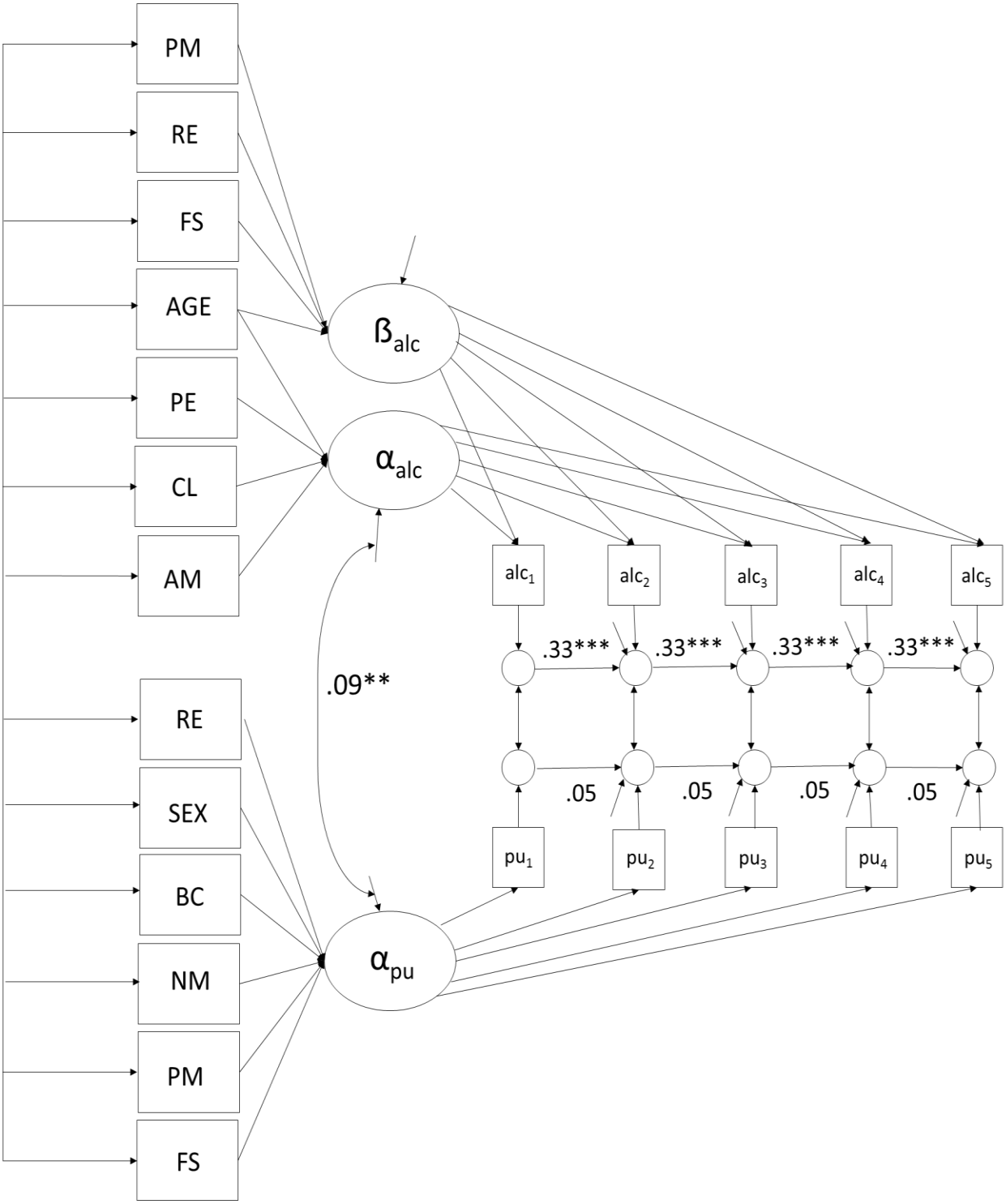
A summary of the model building process for the bivariate LCM-SR for parental alcohol use and adolescent alcohol misuse is provided in Table 14. Figure 7 presents the results of the final bivariate model for parental alcohol use and adolescent alcohol misuse for which the autoregressive paths among the residuals for adolescent alcohol misuse are significant (b=.33, p<.01) but the autoregressive paths among the residuals for parental alcohol use are not significantly different from zero. The covariance between the latent intercept factors was positive and significant (b=.09, p<.05). Given that the prospective cross-lagged regressions were not retained in either direction, hypotheses related to the presence of reciprocal effects between parental alcohol use and adolescent alcohol misuse (H4 and H11) as well as to changes in the magnitude of those reciprocal effects (H18 and H25) were not supported.

Table 14: Model-building process for bivariate LCM-SR between parental alcohol use and adolescent alcohol misuse

	χ^2 (df)	Comparison Model	$\Delta\chi^2$ (df)	CFI	TLI	RMSEA
Step 2: Univariate LCM for parental alcohol use						
6. Random Intercept	12.34 (11)			1.00	1.00	.01
7. 6 + random linear slope	10.63 (8)	6	1.71 (3)	1.00	1.00	.01
Latent Factor	*Mean*		*Variance*			
Intercept	2.63***		10.76***			
8.6 + AR paths equal	11.73 (10)	6	.61 (1)	1.00	1.00	.01
Final model Step 2: 8 + covariates Intercept: average behavioral control, average permissive and negative messages, race, sex, family structure						
Step 3: Bivariate LCM-SR for parental alcohol use and adolescent alcohol misuse, no cross lags						
9. Residual covariances equal	**128.19 (123)**			**1.00**	**1.00**	**.01**
10. Residual covariances free	124.54 (120)	9	3.65 (3)	1.00	1.00	.01
Step 4- Bivariate LCM-SR for parental alcohol use and adolescent alcohol misuse with parent on child cross lags						
11. 9 + cross lags equal	127.32 (122)	9	.87 (1)	1.00	1.00	.01
Prospective regressions	*Beta*	*SE*	*p-value*			
P2 on C1	.17	.18	.35			
P3 on C2	.17	.18	.35			
P4 on C3	.17	.18	.35			
P5 on C4	.17	.18	.35			
Step 5- Bivariate LCM-SR for parental alcohol use and adolescent alcohol misuse with child on parent cross lags						
12. 9 + cross lags equal	127.06 (122)	9	1.13 (1)	1.00	1.00	.01
Prospective regressions	*Beta*	*SE*	*p-value*			
C2 on P1	-.01	.01	.29			
C3 on P2	-.01	.01	.29			
C4 on P3	-.01	.01	.29			
C5 on P4	-.01	.01	.29			

AR= autoregressive paths; +p<.10, **p<.05,***p<.01; **Bold=final model**

Figure 7: Final model for bivariate LCM-SR between parental alcohol use and adolescent alcohol misuse



+p<.10, **p<.05,***p<.01; PM=average permissive messages; RE=race/ethnicity; FS= family structure; PE=parental education; AM= average alcohol-specific monitoring; BC= average behavioral control; NM=average negative messages; CL=average parental closeness

Parental Alcohol-Specific Monitoring

Univariate LCM-SR for Parental Alcohol-Specific Monitoring

I began by estimating a random intercept-only model for alcohol-specific monitoring that included a mean and variance for the intercept factor and residual variances for each of the repeated measures of alcohol-specific monitoring which were allowed to vary over time (the freely-varying time-specific residuals were kept throughout the model-building process). This model fit the data well ($\chi^2(11)=30.99$; CFI=.97, TLI=.98, RMSEA=.03). Next, I added a random linear slope and estimated a mean and variance for the intercept and slope factors as well as a covariance between the latent factors. The fit of the model to the data was excellent ($\chi^2(8)=5.75$; CFI=1.00, TLI=1.00, RMSEA=.00). The addition of the linear slope factor significantly improved model fit relative to the intercept-only model ($\Delta\chi^2(3)=25.24$, $p<.01$) and thus was retained. Next, I included a quadratic growth factor, which resulted in poor model fit ($\chi^2(6)=298.57$; CFI=.51, TLI=.18, RMSEA=.17) and was not retained.

The estimated means for the retained latent factors indicate the model-implied mean trajectory for the sample was characterized by an initial alcohol-specific monitoring score of .32 ($p<.01$) and a significant positive linear growth factor (b=.02, $p<.01$). Thus, the average developmental trajectory of alcohol-specific monitoring is increasing over time. In addition to these fixed effects, there was significant individual variability around the initial level of alcohol-specific monitoring (b=.06, $p<.01$), but not in rates of change of alcohol-specific monitoring over time. The linear slope variance and intercept-slope covariance were not significantly different from zero, and constraining the slope variance and the intercept-slope covariance to zero did not lead to a significant decrement in model fit ($\Delta\chi^2(2)=1.85$, $p=.40$). For parsimony, the linear slope variance and covariance were constrained to zero in all further models.

I then added autoregressive parameters among the residuals for alcohol-specific monitoring to the model and restricted them to be equal over time. The addition of the equal autoregressive parameters did not significantly improve model fit relative to an intercept-slope model with the slope variance fixed to zero ($\Delta\chi^2(1)=.33$,$p=.57$); however, I retained the autoregressive paths because they are theoretically hypothesized to exist. Finally, I regressed the latent factors on the average parental socialization behavior and demographic covariates. I retained those in which the regression parameter was significantly different

from zero (intercept: average behavioral control, average negative messages, race/ethnicity, sex, parental education).

Bivariate LCM-SR for Parental Alcohol-Specific Monitoring and Adolescent Alcohol Misuse

Next, I estimated a bivariate LCM-SR by combining the univariate LCM-SRs for adolescent alcohol misuse and alcohol-specific monitoring. I allowed the intercept factors for alcohol-specific monitoring and adolescent alcohol misuse to covary and the time-specific residuals to covary with each other, setting these covariances to be equal across Times 2-5. I removed the regression of the intercept factor for alcohol misuse on the average alcohol-specific monitoring covariate for this series of bivariate LCM-SRs. This model fit the data well ($\chi^2(114)=166.49$; CFI=.96, TLI=.96, RMSEA=.02). Next, I allowed the time-specific residuals to freely covary across time. This led to significant improvement in model fit ($\Delta\chi^2(3)=8.13$, p=.04), thus I retained the freely varying structure of residual covariances.

I then tested the inclusion of the prospective regressions between the constructs. First, I added the regression of alcohol-specific monitoring on adolescent alcohol misuse, holding the regression of alcohol misuse on alcohol-specific monitoring to zero and constraining the prospective regressions to be equal across time. The model fit the data well ($\chi^2(110)=154.73$; CFI=.97, TLI=.96, RMSEA=.02), and the inclusion of the prospective regressions of alcohol-specific monitoring on alcohol misuse led to a significant improvement of model fit relative to the bivariate model with no cross lags ($\Delta\chi^2(1)=3.63$, p=.05). Given this improvement in model fit with the inclusion of equal prospective regressions, I went on to test whether the magnitude of the prospective regressions systematically changed over time by including a new lag parameter within a model constraint. This model also fit the data well ($\chi^2(109)=152.39$; CFI=.97, TLI=.96, RMSEA=.02), but did not significantly improve model fit relative to the model with equal monitoring on alcohol misuse regressions ($\Delta\chi^2(1)=2.34$, p=.13) and therefore was not retained. The magnitude of the lag parameter (b=.03) indicated that, while not statistically significant, there was a systematic increase in the effect of adolescent alcohol misuse on alcohol-specific monitoring by parents over time. To fully assess the stability of the prospective regressions of alcohol-specific monitoring on alcohol misuse, I allowed the regressions to freely vary over time. This model fit the data well ($\chi^2(107)=150.41$; CFI=.97, TLI=.96, RMSEA=.02), yet did not significantly improve model fit relative

to the model with equal prospective regressions ($\Delta\chi^2(3)=4.32$, p=.23). Thus, I retained the model with equal prospective regressions of alcohol-specific monitoring on adolescent alcohol misuse.

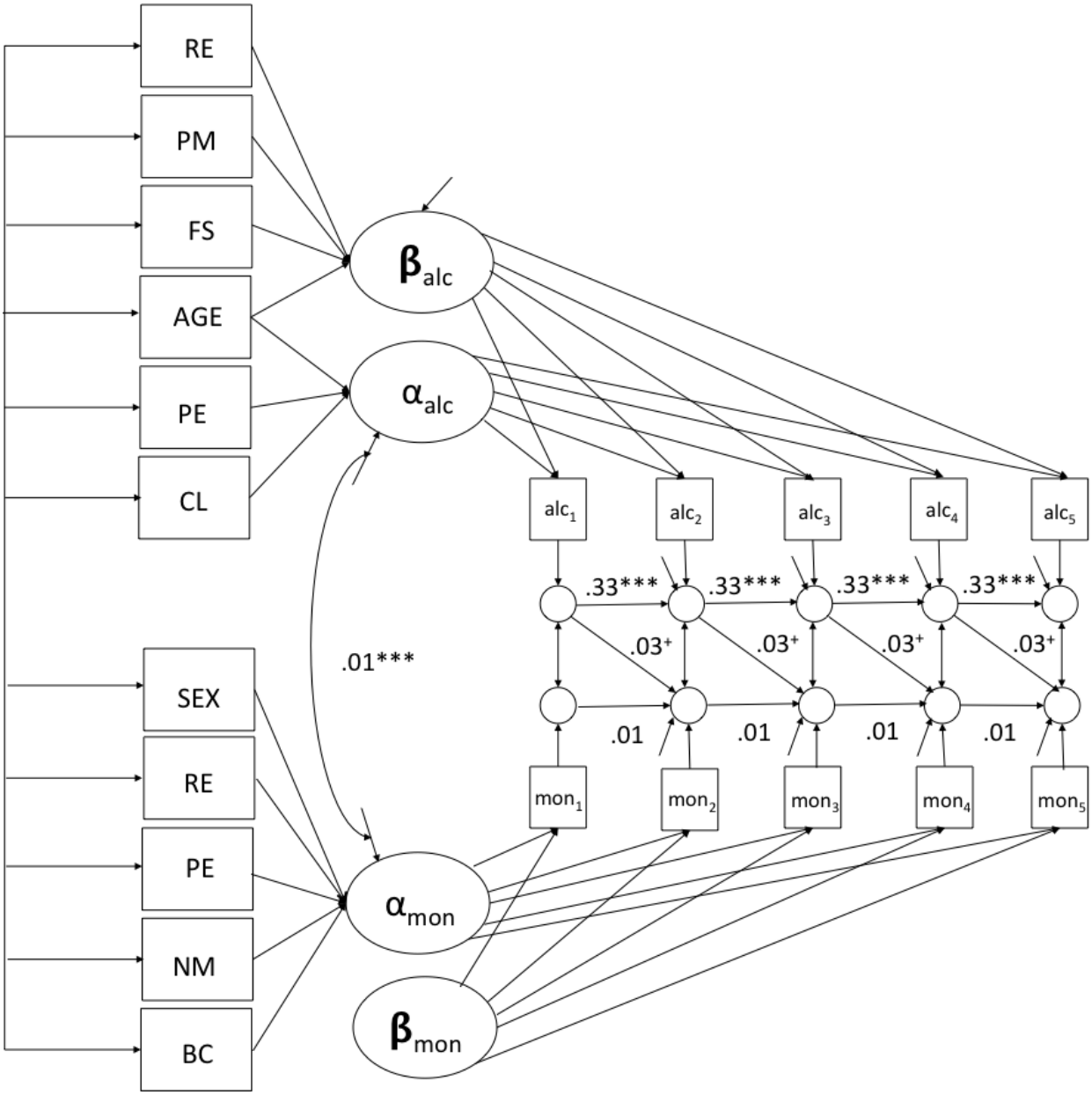
Next, I added the regressions of adolescent alcohol misuse on alcohol-specific monitoring, holding the regression of monitoring on alcohol misuse to zero. This model fit the data well ($\chi^2(110)=157.75$; CFI=.97, TLI=.96, RMSEA=.02) but did not significantly improve model fit relative to the bivariate model with no cross lags ($\Delta\chi^2(1)=.61$, p=.44) and therefore was not retained. A summary of the model building process for the bivariate LCM-SR for alcohol-specific monitoring and adolescent alcohol misuse is provided in Table 15. Figure 8 presents the results of the final model which included statistically significant autoregressive paths among the alcohol-misuse residuals (b=.33, p<.01), and significant prospective pathways from alcohol misuse to subsequent alcohol-specific monitoring (b=.03, p=.05); however, the autoregressive paths among the residuals for alcohol-specific monitoring were not statistically different from zero. The covariance between the latent intercept factors was positive and significant (b=.01, p<.01). The hypothesis that weaker alcohol-specific monitoring would lead to subsequent increases in alcohol misuse (H5) was not supported since no prospective regressions were identified from alcohol-specific monitoring to adolescent alcohol misuse. The significant prospective regression that indicated increases in alcohol misuse led to subsequent increases in alcohol-specific monitoring was in the opposite direction than hypothesized (H12). Neither hypothesis regarding changes in the magnitude of reciprocal effects across time were supported (H19 and H26).

Table 15: Model-building process for bivariate LCM-SR between parental alcohol-specific monitoring and adolescent alcohol misuse

	χ^2 (df)	Comparison Model	$\Delta\chi^2$ (df)	CFI	TLI	RMSEA
Step 2: Univariate LCM for alcohol-specific monitoring						
6. Random intercept	30.99 (11)			0.97	0.98	0.03
7. 6 + Random linear slope	5.75 (8)	6	25.24 (3)***	1.00	1.00	0.00
8. 7 + quadratic slope	298.57 (6)	7	n/a	0.51	0.18	0.17
Latent Factor	*Mean*	*Variance*				
Intercept	.32***	.06***				
Linear slope	.02***	.00				
9. 7 + AR paths equal (+ fixed monitoring slope variance)	7.28 (9)	7	.33 (1)	1.00	1.00	0.00
Final model Step 2: 9 + covariates Intercept: race, sex, parental education, average behavioral control, average negative messages						
Step 3: Bivariate LCM-SR for monitoring and adolescent alcohol misuse, no cross lags						
10. Residual covariances equal	166.49 (114)			0.96	0.96	0.02
11. Residual covariances free	158.36 (111)	10	8.13 (3)**	0.96	0.96	0.02
Step 4- Bivariate LCM-SR for monitoring and adolescent alcohol misuse with parent on child cross lags						
12. 11 + cross lags equal	**154.73 (110)**	**11**	**3.63 (1)+**	**0.97**	**0.96**	**0.02**
Prospective regressions	*Beta*	*SE*	*p-value*			
P2 on C1	.03	.02	.06			
P3 on C2	.03	.02	.06			
P4 on C3	.03	.02	.06			
P5 on C4	.03	.02	.06			
13. 11 + cross lags systematically change	152.39 (109)	12	2.34 (1)	0.97	0.96	0.02
14. 11 + cross lags free	150.41 (107)	12	4.32 (3)	0.97	0.96	0.02
Step 5- Bivariate LCM-SR for monitoring and adolescent alcohol misuse with child on parent cross lags						
15. 11 + cross lags equal	157.75 (110)	11	.61 (1)	0.97	0.96	0.02
Prospective regressions	*Beta*	*SE*	*p-value*			
C2 on P1	-.05	.07	.44			
C3 on P2	-.05	.07	.44			
C4 on P3	-.05	.07	.44			
C5 on P4	-.05	.07	.44			

AR= autoregressive paths; +p<.10, **p<.05,***p<.01; **Bold=final model**

Figure 8: Final model for bivariate LCM-SR between alcohol-specific monitoring and adolescent alcohol misuse



+p<.10, **p<.05,***p<.01; RE=race/ethnicity; PM= average permissive messages; FS= family structure; PE=parental education; BC= average behavioral control; NM=average negative messages; CL=average parental closeness

Parental Permissive Messages

Univariate LCM-SR for Parental Permissive Messages

I first estimated a random intercept model that included only a mean and variance for the intercept factor and residual variances for each of the repeated measures of permissive messages which were allowed to vary over time (the freely-varying time-specific residuals were kept throughout the model-building process). This model fit the data well ($\chi^2(11)=66.12$; CFI=.97, TLI=.98, RMSEA=.06). Next, I added a linear slope factor by estimating a mean and variance for both the intercept and slope factor and a covariance between the two latent variables. The fit of this model to the data was excellent ($\chi^2(8)=19.82$; CFI=.99, TLI=.99, RMSEA=.03). Relative to the intercept-only model, the inclusion of the random linear slope significantly improved model fit ($\Delta\chi^2(3)=46.30$, $p<.01$). I then added a quadratic growth factor to the model, estimating a mean and variance for each factor and covariances between them all. This model fit the data poorly ($\chi^2(6)=449.78$; CFI=.69, TLI=.49, RMSEA=.21) and was not retained.

The estimated means for the retained latent factors indicate the model-implied mean trajectory for the sample was characterized by an initial permissive messages score of 0.19 ($p<.01$) and a significant positive linear growth factor (b=.02, $p<.01$). Thus, the average developmental trajectory of permissive messages is increasing over time. In addition to these fixed effects, there was significant individual variability around the initial level of permissive messages (b=.06, $p<.01$), and in the rate of change in permissive messages over time (b=.003, $p<.01$).

Next, I added autoregressive paths among the residuals of the repeated measures of permissive messages, setting these paths to be equal over time. Overall, the fit of this model to the data was excellent ($\chi^2(7)=13.86$; CFI=1.00, TLI=1.00, RMSEA=.02). The inclusion of the autoregressive paths significantly improved model fit relative to the intercept and linear slope model ($\Delta\chi^2(1)=5.96$, $p=.01$). Next, I allowed the autoregressive paths to vary across time, however, this model would not estimate correctly due to the linear slope factor. The variance of the linear slope factor was not statistically different from zero (b=0.00, $p=.87$), therefore I re-estimated the model restricting the linear slope variance and covariance to zero. This did not lead to significant improvement in model fit relative to the model with equal autoregressive paths ($\Delta\chi^2(1)=.20$, $p=.66$); therefore, I retained the model with equal autoregressive

paths across time and a freely estimated linear slope variance. Finally, I regressed the latent factors on the average parental socialization behavior and demographic covariates. I retained those in which the regression parameter was significantly different from zero (intercept: average negative messages, average parental alcohol use, race/ethnicity, parental education).

Bivariate LCM-SR for Parental Permissive Messages and Adolescent Alcohol Misuse

I then combined the univariate LCM-SRs for adolescent alcohol misuse and permissive messages into a single bivariate LCM-SR. I allowed the intercept and slope factors for permissive messages to covary with each other as well as the intercept factor for alcohol misuse. I allowed the time-specific residuals to covary with each other and set the covariances to be equal across Times 2-5. I removed the regression of the slope factor for alcohol misuse on the average permissive messages covariate for this series of bivariate LCM-SRs. This model fit the data well ($\chi^2(102)=142.40$; CFI=.98, TLI=.98, RMSEA=.02). Next, I allowed the time-specific residuals to freely covary across time. Overall, this model fit the data well ($\chi^2(99)=133.80$; CFI=.98, TLI=.98, RMSEA=.02). This led to a significant improvement in model fit ($\Delta\chi^2(3)=8.60$, p=.04), thus I retained the freely varying residual covariance structure.

I then tested the inclusion of prospective relations between the constructs in multiple steps. First, I added the regression of permissive messages on adolescent alcohol misuse, holding the regression of alcohol misuse on permissive messages to zero. I constrained the prospective regressions to be equal across time. While the model itself fit the data well ($\chi^2(98)=133.41$; CFI=.98, TLI=.98, RMSEA=.02), the inclusion of the prospective regressions of permissive messages on alcohol misuse did not significantly improve model fit relative to the bivariate model with no cross lags ($\Delta\chi^2(1)=.39$, p=.53), and thus were not retained. Next, I added the regressions of adolescent alcohol misuse on permissive messages, holding the regression of permissive messages on alcohol misuse to zero. Similarly, this model fit the data well ($\chi^2(98)=132.08$; CFI=.99, TLI=.98, RMSEA=.02) but did not significantly improve model fit relative to the bivariate model with no cross lags ($\Delta\chi^2(1)=1.72$, p=.19) and therefore was not retained.

A summary of the model building process for the bivariate LCM-SR for permissive messages and adolescent alcohol misuse is provided in Table 16. Figure 9 presents the results of the final model in which the autoregressive paths for adolescent alcohol misuse (b=.33, p<.01) and permissive messages

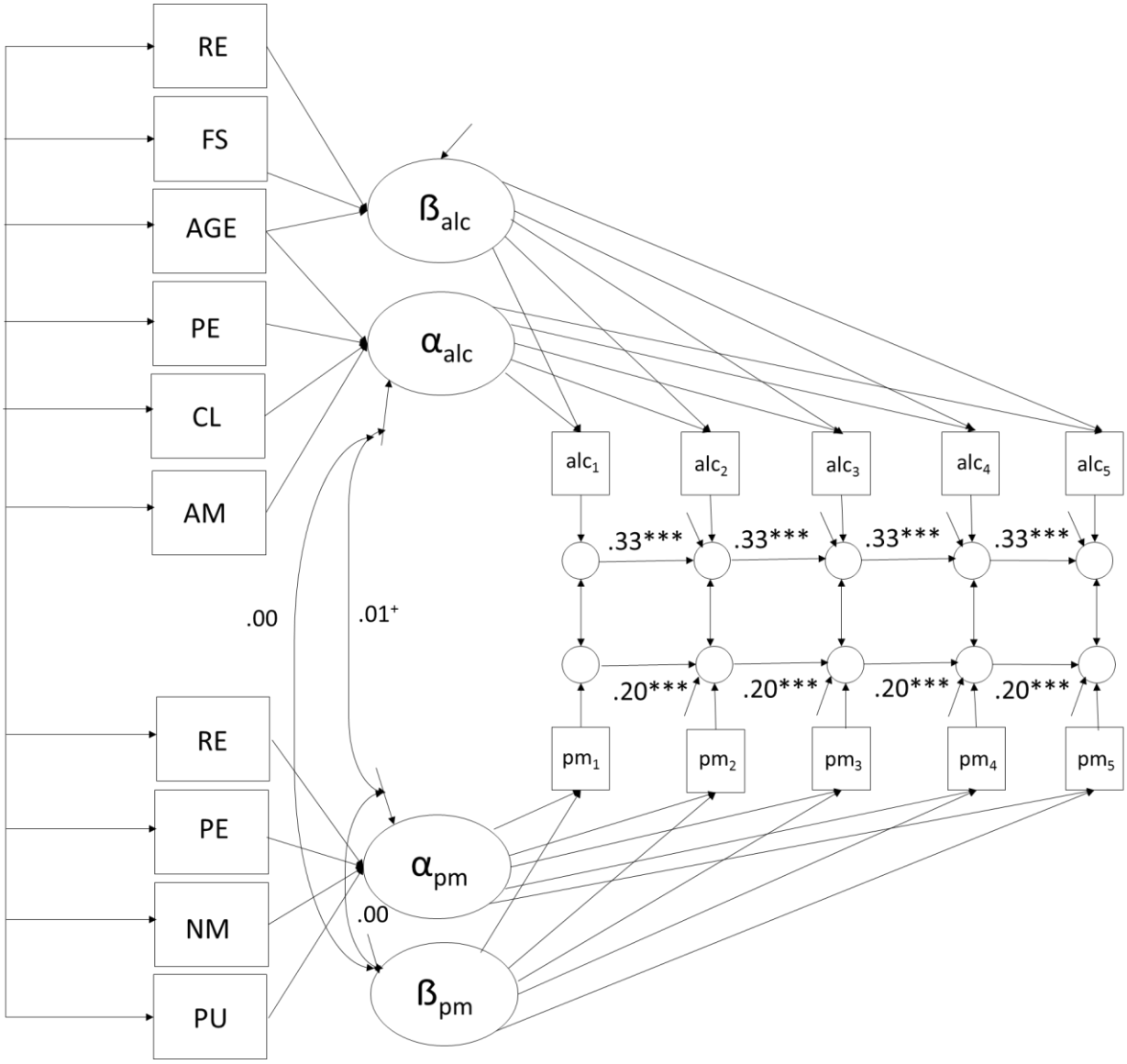
(b=.20, p<.01) were significant and equal in magnitude across time. The covariance between the latent intercept factors was positive and marginally significant (b=.01, p<.10); however the covariance between the intercept of alcohol misuse and slope of permissive messages and intercept-slope covariance for permissive messages was not significantly different from zero. Given that the prospective cross-lagged regressions were not retained in either direction, hypotheses related to the presence of reciprocal effects between permissive messages and adolescent alcohol misuse (H6 and H13) as well as to changes in the magnitude of those reciprocal effects (H20 and H27) were not supported.

Table 16: Model-building process for bivariate LCM-SR between parental permissive messages and adolescent alcohol misuse

	χ^2 (df)	Comparison Model	$\Delta\chi^2$ (df)	CFI	TLI	RMSEA
Step 2: Univariate LCM for permissive messages						
6. Random intercept	66.12 (11)			0.97	0.98	0.06
7. 6 + random linear slope	19.82 (8)	6	46.30 (3)***	0.99	0.99	0.03
8. 7 + random quadratic slope	449.78 (6)	7	--	0.69	0.49	0.21
Latent Factor	*Mean*		*Variance*			
Intercept	.19***		.06***			
Linear slope	.02***		.003**			
9. 7 + AR paths equal	13.86 (7)	7	5.96 (1)**	1.00	1.00	0.02
10. 7 + AR paths free	13.66 (6)	9	0.20 (1)	1.00	0.99	0.03
Final model step 2: 9 + covariates: Intercept: parental education, race, average negative messages, average parental alcohol use						
Step 3: Bivariate LCM-SR for permissive messages and adolescent alcohol misuse, no cross lags						
11. Residual covariances equal	142.40 (102)			0.98	0.98	0.02
12. Residual covariances free	**133.80 (99)**	**11**	**8.60 (3)****	**0.98**	**0.98**	**0.02**
Step 4- Bivariate LCM-SR for permissive messages and adolescent alcohol misuse with parent on child cross lags						
13. 12 + cross lags equal	133.41 (98)	12	.39 (1)	0.98	0.98	0.02
Prospective regressions	*Beta*	*SE*	*p-value*			
P2 on C1	.01	.01	.53			
P3 on C2	.01	.01	.53			
P4 on C3	.01	.01	.53			
P5 on C4	.01	.01	.53			
Step 5- Bivariate LCM-SR for permissive messages and adolescent alcohol misuse with child on parent cross lags						
14. 12 + cross lags equal	132.08 (98)	12	1.72(1)	0.99	0.98	0.02
Prospective regressions	*Beta*	*SE*	*p-value*			
C2 on P1	.13	.10	.19			
C3 on P2	.13	.10	.19			
C4 on P3	.13	.10	.19			
C5 on P4	.13	.10	.19			

AR= autoregressive paths; +p<.10, **p<.05,***p<.01; **Bold=final mode**

Figure 9: Final model for bivariate LCM-SR between permissive messages and adolescent alcohol misuse



+p<.10, **p<.05,***p<.01; RE=race/ethnicity; FS= family structure; PE=parental education; AM= average alcohol-specific monitoring; BC= average behavioral control; NM=average negative messages; CL=average parental closeness; PU=average parental alcohol use

Parental Negative Messages

Univariate LCM-SR for Parental Negative Messages

I first estimated a random intercept model that included only a mean and variance for the intercept factor and residual variances for each of the repeated measures of negative messages, which were allowed to vary over time (the freely-varying time-specific residuals were kept throughout the model-building process). This model fit the data well ($\chi^2(11)=82.77$; CFI=.93, TLI=.95, RMSEA=.06). Next, I added a linear slope factor by estimating a mean and variance for both the intercept and slope factor and a covariance between the two latent variables. The fit of this model to the data was excellent ($\chi^2(8)=20.70$; CFI=.99, TLI=.99, RMSEA=.03). Relative to the intercept-only model, the inclusion of the random linear slope significantly improved model fit ($\Delta\chi^2(3)=62.07$, $p<.01$). I then added a quadratic growth factor to the model, estimating a mean and variance for each factor and covariances between them all. This model could not be estimated.

The estimated means for the retained latent factors indicate the model-implied mean trajectory for the sample was characterized by an initial negative messages score of 0.83 ($p<.01$) and a significant positive linear growth factor (b=.02, $p<.01$). Thus, the average developmental trajectory of negative messages is increasing over time. In addition to these fixed effects, there was significant individual variability around the initial level of negative messages (b=.04, $p<.01$), and the rate of change in negative messages over time (b=.002, $p<.01$).

Next, I added autoregressive paths among the residuals of the repeated measures of negative messages, setting these paths to be equal over time. This model could not be estimated properly due to the linear slope factor. The variance of the slope factor in this model was not significantly different from zero (b=.02, $p<.01$), thus I restricted the variance and covariance of the linear slope factor to zero. Overall, the fit of this model to the data was excellent ($\chi^2(9)=2.30$; CFI=1.00, TLI=1.00, RMSEA=.00). The inclusion of the autoregressive paths significantly improved model fit relative to an intercept and linear slope model in which the slope variance was fixed to zero ($\Delta\chi^2(1)=38.06$, $p<.01$). Next, I allowed the autoregressive paths to vary across time, but this did not lead to significant improvement in model fit ($\Delta\chi^2(3)=1.26$, $p=.74$); therefore, I retained the model with equal autoregressive paths across time. Finally, I regressed the latent factors on the average parental socialization behavior and demographic covariates.

I retained those in which the regression parameter was significantly different from zero (intercept: average support, average behavioral control, average alcohol-specific monitoring, average permissive messages; slope: family structure).

Bivariate LCM-SR for Parental Negative Messages and Adolescent Alcohol Misuse

I then combined the univariate LCM-SRs for adolescent alcohol misuse and negative messages into a single bivariate LCM-SR. I allowed the latent intercept factors for the two constructs to covary. I allowed the time-specific residuals to covary with each other and set the covariances to be equal across Times 2-5. This model fit the data well ($\chi^2(113)=128.20$; CFI=.99, TLI=.99, RMSEA=.01). Next, I allowed the time-specific residuals to freely covary across time. Overall, this model fit the data well ($\chi^2(110)=123.95$; CFI=.99, TLI=.99, RMSEA=.01). This did not lead to a significant improvement in model fit relative to the model with equal residual covariances ($\Delta\chi^2(3)=4.25$, p=.24) and was not retained.

I then tested the inclusion of prospective relations between the constructs in multiple steps. First, I added the regression of negative messages on adolescent alcohol misuse, holding the regression of alcohol misuse on negative messages to zero. I constrained the prospective regressions to be equal across time. While the model itself fit the data well ($\chi^2(112)=128.10$; CFI=.99, TLI=.99, RMSEA=.01), the inclusion of the prospective regressions of negative messages on alcohol misuse did not significantly improve model fit relative to the bivariate model with no cross lags ($\Delta\chi^2(1)=.10$, p=.75), and thus were not retained. Next, I added the regressions of adolescent alcohol misuse on negative messages, holding the regression of negative messages on alcohol misuse to zero. Similarly, this model fit the data well ($\chi^2(112)=127.70$; CFI=.99, TLI=.99, RMSEA=.01) but did not significantly improve model fit relative to the bivariate model with no cross lags ($\Delta\chi^2(1)=.50$, p=.48) and therefore was not retained.

A summary of the model building process for the bivariate LCM-SR for negative messages and adolescent alcohol misuse is provided in Table 17. Figure 10 presents the results of the final model in which the autoregressive paths for adolescent alcohol misuse (b=.33, p<.01) and negative messages (b=.28, p<.01) are significant and equal in magnitude across time. The covariance between the latent intercept factors was positive and significant (b=.01, p<.05). Given that the prospective cross-lagged regressions were not retained in either direction, hypotheses related to the presence of reciprocal effects

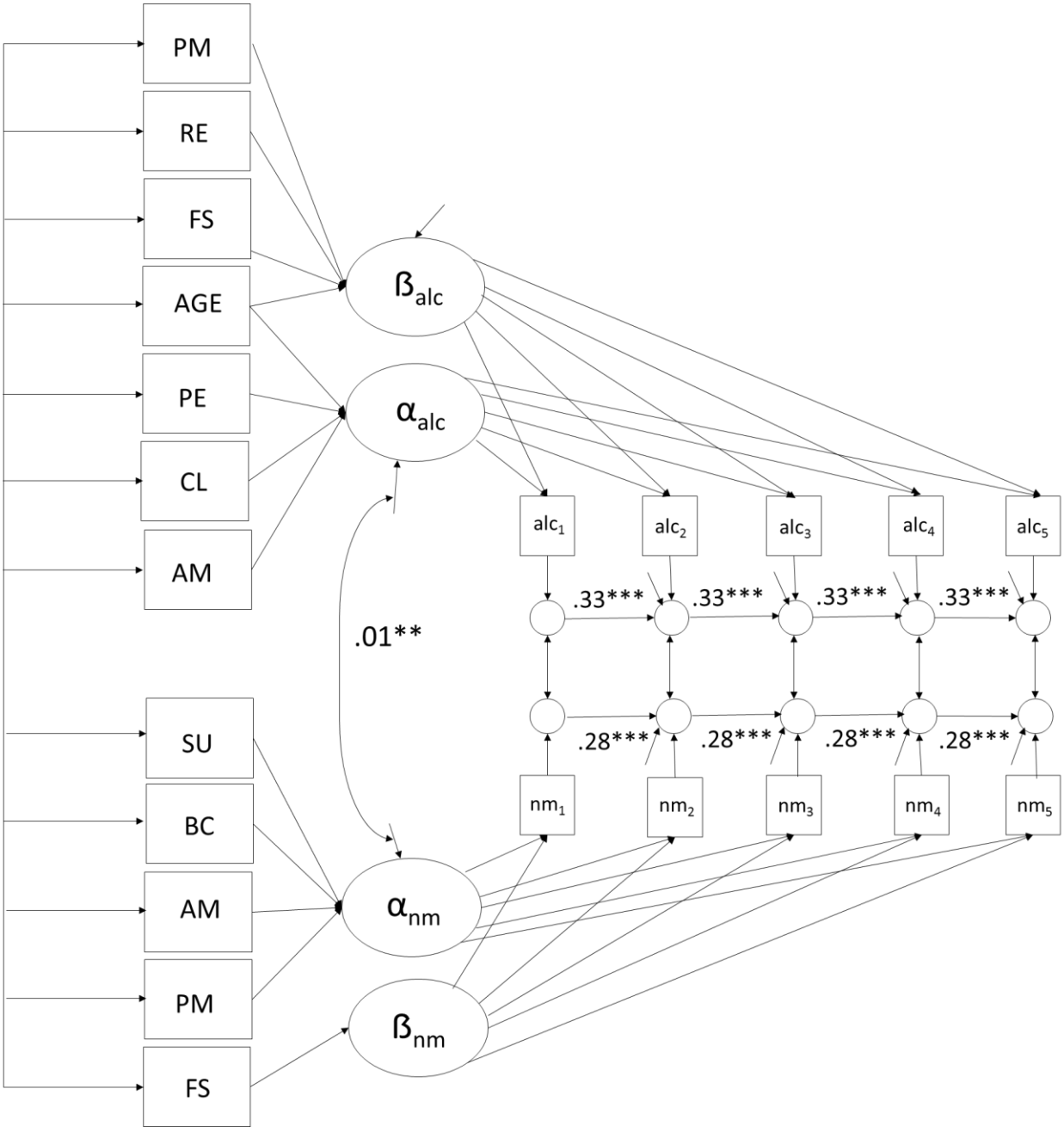
between negative messages and adolescent alcohol misuse (H7 and H14) as well as to changes in the magnitude of those reciprocal effects (H21 and H28) were not supported.

Table 17: Model-building process for bivariate LCM-SR between parental negative messages and adolescent alcohol misuse

	χ^2 (df)	Comparison Model	$\Delta\chi^2$ (df)	CFI	TLI	RMSEA
Step 2: Univariate LCM for parental negative messages						
6. Random Intercept	82.77 (11)			0.93	0.95	0.06
7. 6 + random linear slope	20.70 (8)	6	62.70 (3)**	0.99	0.99	0.03
8. 7 + Random quadratic slope	--					
Latent Factor	*Mean*		*Variance*			
Intercept	.83***		.04***			
Linear slope	.02***		.002***			
9. 7 + AR paths equal (+ fixed linear slope)	2.30 (9)	7	38.06 (1)***	1.00	1.00	0.00
10. 7 + AR paths free (+ fixed linear slope)	1.04 (6)	9	1.26 (3)	1.00	1.00	0.00
Final model Step 2: 9 + covariates Intercept: average support, average behavioral control, average parental alcohol use, average permissive messages Slope: family structure						
Step 3: Bivariate LCM-SR for parental negative messages and adolescent alcohol misuse, no cross lags						
11. Residual covariances equal	**128.20 (113)**			**0.99**	**0.99**	**0.01**
12. Residual covariances free	123.95 (110)	11	4.25 (3)	0.99	0.99	0.01
Step 4- Bivariate LCM-SR for parental negative messages and adolescent alcohol misuse with parent on child cross lags						
13. 11 + cross lags equal	128.10 (112)	11	.10 (1)	0.99	0.99	0.01
Prospective regressions	*Beta*	*SE*	*p-value*			
P2 on C1	.00	.01	.75			
P3 on C2	.00	.01	.75			
P4 on C3	.00	.01	.75			
P5 on C4	.00	.01	.75			
Step 5- Bivariate LCM-SR for parental negative messages and adolescent alcohol misuse with child on parent cross lags						
14. 11 + cross lags equal	127.70 (112)	11	.50 (1)	0.99	0.99	0.01
Prospective regressions	*Beta*	*SE*	*p-value*			
C2 on P1	.07	.10	.48			
C3 on P2	.07	.10	.48			
C4 on P3	.07	.10	.48			
C5 on P4	.07	.10	.48			

AR= autoregressive paths; +p<.10, **p<.05,***p<.01; **Bold=final model**

Figure 10: Final model for bivariate LCM-SR between negative messages and adolescent alcohol misuse



+p<.10, **p<.05,***p<.01; PM=average permissive messages; RE=race/ethnicity; FS= family structure; PE=parental education; AM= average alcohol-specific monitoring; BC= average behavioral control; NM=average negative messages; CL=average parental closeness; SU=average support

Sensitivity Analyses: Six-Month Time Lag for Prospective Relationships

I re-estimated the univariate curve for adolescent alcohol use with nine time points representing grades 6.5-10.5 in six month intervals. Covariances between residuals of each construct at the same time point could only be estimated for the times in which parental data were available (grades 6.5, 7.5, 8.5, 9.5, 10.5). The best fitting model for the univariate curve for adolescent alcohol misuse included a random intercept, a linear slope in which the variance was constrained to zero, and autoregressive paths between the repeated measures allowed to vary over time. The fit of this model to the data was moderate (χ^2 (24)=134.79; CFI=.90; TLI=.89; RMSEA=.05). The univariate curve for all parental socialization variables remained the same from previous analyses.

Parental Closeness

The final bivariate LCM-SR between parental closeness and adolescent alcohol misuse included only autoregressive paths for each univariate curve, equal residual covariances between repeated measures of the two constructs, a covariance between the intercept of parental closeness with the slope of parental closeness and the intercept of alcohol misuse, and a covariance between the intercept of alcohol misuse and the linear slope of parental closeness. This model fit the data well (χ^2 (176)=308.61; CFI=.96; TLI=.95; RMSEA=.02). Parameter estimates for the autoregressive paths between repeated measures of adolescent alcohol misuse varied over time from -.29 to .68 and were all statistically significant at p<.01 (except the path between the first and second time point for which p=.08). Estimates for the autoregressive parameters between repeated measures of parental closeness varied from -.61 to .06, none of which were statistically different from zero. No parameter estimates for the prospective paths between constructs were statistically significant. The covariance between the intercepts of the two latent factors was significant (b=-.01, p<.01), but not the covariance between the alcohol misuse intercept and slope of parental closeness (b=.00, p=.22) nor the covariance between the intercept and slope of parental closeness (b=.00, p=.63). Since prospective cross-lagged regressions were not retained in either direction, hypotheses related to the presence of reciprocal effects between parental closeness and adolescent alcohol misuse (H1 and H8) as well as to changes in the magnitude of those reciprocal effects (H15 and H22) were not supported.

Parental Support

The final bivariate LCM-SR linking parental support and adolescent alcohol misuse included autoregressive paths between repeated measures for each univariate curve, residual covariances constrained to be equal over time and a covariance between the latent intercepts of the two constructs. This model fit the data well (χ^2 (180)=309.49; CFI=.95; TLI=.94; RMSEA=.02). Parameter estimates for the autoregressive paths between repeated measures of adolescent alcohol misuse varied over time from -.30 to .68 and were all statistically significant at p<.01 (except the path between the first and second time point for which p=.08). The equal autoregressive paths between repeated measures of parental support were not significant (b=.04, p=.46). The covariance between the intercept factors of the two constructs (b=.09) was marginally significant at p<.10. No parameter estimates for the prospective paths between constructs were statistically significant; therefore, hypotheses related to the presence of reciprocal effects between parental support and adolescent alcohol misuse (H2 and H9) as well as to changes in the magnitude of those reciprocal effects (H16 and H23) were not supported.

Parental Behavioral Control

The final bivariate LCM-SR between parental behavioral control and adolescent alcohol misuse included autoregressive paths between the repeated measures of both constructs, residual covariances between repeated measures of the two constructs that freely vary over time, and a covariance between the intercepts of the two constructs. The fit of this model to the data was moderate (χ^2 (163)=395.46; CFI=.89; TLI=.88; RMSEA=.03). Parameter estimates for the autoregressive paths between repeated measures of adolescent alcohol misuse varied over time from -.33 to .68 and were all statistically significant at p<.01 (except the path between the first and second time point for which p=.06). The equal autoregressive paths between repeated measures of behavioral control were not significant (b=-.05, p=.34). The covariance between the intercept factors of the two constructs (b=-.01) was marginally significant at p<.10. No parameter estimates for the prospective paths between constructs were statistically significant; therefore, hypotheses related to the presence of reciprocal effects between behavioral control and adolescent alcohol misuse (H3 and H10) as well as to changes in the magnitude of those reciprocal effects (H17 and H24) were not supported.

Parental Alcohol Use

The final bivariate LCM-SR linking parental alcohol use and adolescent alcohol misuse included autoregressive paths between repeated measures for each univariate curve, residual covariances constrained to be equal over time, a covariance between the latent intercepts of the two constructs, and equal prospective pathways in which adolescent alcohol misuse predicts parental alcohol use at the subsequent time point. This model fit the data well (χ^2 (192)=307.83; CFI=.95; TLI=.95; RMSEA=.02). Parameter estimates for the autoregressive paths between repeated measures of adolescent alcohol misuse varied over time from -.26 to .68 and were all statistically significant at p<.01 (except the path between the first and second time point for which p=.12). Autoregressive paths between repeated measures of parental alcohol use (b=.05) were not significantly different from zero (p=.35). The covariance between the intercepts of the two constructs (b=.10) was also significantly different from zero (p<.01). Prospective paths from adolescent alcohol misuse to parental alcohol use were marginally significant (b=.26) at p<.10, therefore the hypothesis that increased adolescent alcohol use would increase parental alcohol use (H11) was moderately supported. However, hypotheses related to the influence of parental alcohol use on adolescent alcohol misuse (H4), and the changing nature of the reciprocal relations (H18 and H25) were not supported.

Parental Alcohol-Specific Monitoring

To estimate the bivariate LCM-SR between alcohol-specific monitoring and alcohol misuse, the variance of the linear slope for alcohol-specific monitoring had to be fixed to zero. The resulting bivariate model included autoregressive paths between the repeated measures of each construct, estimated residual covariances that were equal over time and covariance between the intercepts of the two constructs. The fit of this model to the data was moderate (χ^2 (179)= 335.66; CFI=.93; TLI=.92; RMSEA=.02). Parameter estimates for the autoregressive paths between repeated measures of adolescent alcohol misuse varied over time from -.29 to .68 and were all statistically significant at p<.01 (except the path between the first and second time point for which p=.09). Autoregressive paths between repeated measures of alcohol-specific monitoring (b=.01) were not significantly different from zero (p=.79). The covariance between the intercepts of the two constructs (b=.01) was significantly different from zero (p<.01). No parameter estimates for the prospective paths between constructs were statistically

significant; therefore, hypotheses related to the presence of reciprocal effects between alcohol-specific monitoring and adolescent alcohol misuse (H5 and H12) as well as to changes in the magnitude of those reciprocal effects (H19 and H26) were not supported.

Parental Permissive Messages

The final bivariate LCM-SR between permissive messages and adolescent alcohol misuse included autoregressive paths between repeated measures for each univariate curve, residual covariances between concurrent repeated measures of the two constructs that freely varied across time, a covariance between the intercept of permissive messages with the slope of permissive messages and the intercept of alcohol misuse, and a covariance between the intercept of alcohol misuse and the linear slope of permissive messages. This model fit the data well (χ^2 (161)=332.51; CFI=.95; TLI=.94; RMSEA=.03). Parameter estimates for the autoregressive paths between repeated measures of adolescent alcohol misuse varied over time from -.32 to .67 and were all statistically significant at p<.01 (except the path between the first and second time point for which p=.06). The autoregressive parameters (b=.19) between repeated measures of permissive messages were statistically different from zero (p=.02). None of the covariances between latent factors including the intercepts of the two latent factors (b=.00, p=.19), the alcohol misuse intercept and linear slope of permissive messages (b=.00, p=.47) nor the covariance between the intercept and slope of permissive messages (b=.00, p=.56) were statistically different from zero. No parameter estimates for the prospective paths between constructs were statistically significant; therefore, hypotheses related to the presence of reciprocal effects between permissive messages and adolescent alcohol misuse (H6 and H13) as well as to changes in the magnitude of those reciprocal effects (H20 and H27) were not supported.

Parental Negative Messages

The final bivariate LCM-SR linking negative messages and adolescent alcohol misuse included autoregressive paths between repeated measures for each univariate curve, residual covariances constrained to be equal over time and a covariance between the latent intercepts of the two constructs. This model fit the data well (χ^2 (179)=306.88; CFI=.95; TLI=.95; RMSEA=.02). Parameter estimates for the autoregressive paths between repeated measures of adolescent alcohol misuse varied over time from -.29 to .68 and were all statistically significant at p<.01 (except the path between the first and second time

point for which p=.09). The equal autoregressive paths between repeated measures of negative messages were also significantly different from zero (b=.28, p<.01). The covariance between the intercept factors of the two constructs (b=.00) was statistically significant at p<.05. No parameter estimates for the prospective paths between constructs were statistically significant; therefore, hypotheses related to the presence of reciprocal effects between negative messages and adolescent alcohol misuse (H7 and H14) as well as to changes in the magnitude of those reciprocal effects (H21 and H28) were not supported.

Sensitivity Analyses: Comparison of Adolescent versus Parental Report of Parental Socialization Behaviors

Perceived Parental Closeness

The final bivariate LCM-SR linking perceived parental closeness and adolescent alcohol misuse included autoregressive paths between repeated measures for each univariate curve and residual covariances constrained to be equal over time. There were also covariances between the latent intercept of alcohol misuse with the slope of closeness, and covariances between the latent intercept of closeness with the slope of closeness and the intercept of alcohol misuse. The fit of this model to the data was excellent (χ^2 (98)=129.87; CFI=.98; TLI=.98; RMSEA=.01). Parameter estimates for the autoregressive paths between repeated measures of adolescent alcohol misuse were statistically different from zero (b=.32, p<.01). The equal autoregressive paths between repeated measures of parental closeness were also significantly different from zero (b=.42, p<.01). The covariance between the intercept of alcohol misuse and slope of closeness was not statistically significant (b=-.01, p=.24), nor was the covariance between the latent intercept factors (b=.00, p=.69). The covariance between the intercept and slope of closeness was positive and significant (b=.06, p<.05). No parameter estimates for the prospective paths between constructs were statistically significant; therefore, hypotheses related to the presence of reciprocal effects between parental closeness and adolescent alcohol misuse (H1 and H8) as well as to changes in the magnitude of those reciprocal effects (H15 and H22) were not supported.

Perceived Parental Support

The final bivariate LCM-SR linking perceived parental support and adolescent alcohol misuse included autoregressive paths between repeated measures for each univariate curve and residual covariances constrained to be equal over time. There were also covariances between the latent intercept

of support with the slope of support and the intercept of alcohol misuse and between the intercept of alcohol use and slope of parental support. The model fit the data well (χ^2 (93)=135.28; CFI=.97; TLI=.96; RMSEA=.02). Parameter estimates for the autoregressive paths between repeated measures of adolescent alcohol misuse were statistically different from zero (b=.33, p<.01). The equal autoregressive paths between repeated measures of parental support were also significantly different from zero (b=.13, p<.05). The covariances between the intercept of support and both the slope of support (b=-.01, p=.70) and intercept of alcohol misuse (b=-.02, p=.17) and between the intercept of alcohol misuse and slope of closeness (b=.00, p=.51) were not statistically significant. No parameter estimates for the prospective paths between constructs were statistically significant; therefore, hypotheses related to the presence of reciprocal effects between parental support and adolescent alcohol misuse (H2 and H9) as well as to changes in the magnitude of those reciprocal effects (H16 and H23) were not supported.

Perceived Parental Behavioral Control

The final bivariate LCM-SR linking perceived parental behavioral control and adolescent alcohol misuse included autoregressive paths between repeated measures for alcohol misuse only, residual covariances constrained to be equal over time, prospective pathways in which adolescent alcohol misuse predicts parental behavioral control at the subsequent time point and six covariances between the latent factors of the two constructs. This model fit the data moderately well (χ^2 (85)=150.23; CFI=.94; TLI=.93; RMSEA=.02). Parameter estimates for the autoregressive paths between repeated measures of adolescent alcohol misuse were statistically different from zero (b=.32, p<.01). Autoregressive paths between repeated measures of behavioral control could not be estimated. There was a significant prospective regression from adolescent alcohol misuse to parental behavioral control from T3 to T4 (b=-.12, p<.01) and T4 to T5 (b=-.25, p<.01). Overall, the prospective paths from adolescent alcohol misuse to parental behavioral control decreased with each subsequent time point (lag parameter=-.13). Therefore, the hypothesis that increased adolescent alcohol misuse would lead to decreased parental behavioral control (H3) and that the magnitude of the effect of adolescent alcohol misuse on behavioral control would differ across adolescence (H24) were supported. However, hypotheses regarding the influence of behavioral control on adolescent alcohol misuse (H10 and H17) were not supported. None of the covariances between latent constructs were significant including between the intercept of behavioral

control and the linear (b=-.10, p=.52) and quadratic (b=.03, p=.59) slopes of behavioral control; between the linear and quadratic slopes of behavioral control (b=-.06, p=.22); and the intercept of alcohol misuse with the intercept (b=-.02, p=.19), linear slope (b=.00, p=.90) and quadratic slope (b=.00, p=.53) of behavioral control.

Perceived Parental Alcohol Use

The final bivariate LCM-SR linking perceived parental alcohol use and adolescent alcohol misuse included autoregressive paths between repeated measures for each univariate curve, residual covariances that varied over time and a covariance between the latent intercepts of the two constructs. This model fit the data well (χ^2 (88)=112.88; CFI=.99; TLI=.99; RMSEA=.01). Parameter estimates for the autoregressive paths between repeated measures of parental alcohol use varied over time from -1.24 to .42 and was marginally significant from T1 to T2 (p<.10), and statistically significant from T3 to T4 (p<.01) and T4 to T5 (p<.01). The equal autoregressive paths between repeated measures of alcohol misuse were also significantly different from zero (b=.32, p<.01). The covariance between the intercept factors of the two constructs was not statistically different from zero (b=.01, p=.12). No parameter estimates for the prospective paths between constructs were statistically significant; therefore, hypotheses related to the presence of reciprocal effects between parental alcohol use and adolescent alcohol misuse (H4 and H11) as well as to changes in the magnitude of those reciprocal effects (H18 and H25) were not supported.

CHAPTER 5: DISCUSSION

This study extended previous research regarding parental socialization and adolescent alcohol misuse by examining the reciprocity of behaviors between parents and adolescents across early and middle adolescence. To do so, the study explored mechanisms of influence on parenting and adolescent drinking. Bidirectional relationships were examined between adolescent alcohol misuse and seven parental socialization behaviors: three general parenting behaviors, three alcohol-specific behaviors and parental alcohol use to isolate influence effects while controlling for confounding factors.

The study integrates multiple theoretical perspectives to evaluate novel relationships between parental socialization behaviors and adolescent drinking. To complement existing research on the effects of parenting on alcohol use, I extend the research to include transactional models of development. Underlying transactional theoretical models is the assumption that individuals engage in a mutually reinforcing process with their environment, particularly other individuals closest to them. Individuals are continually developing, partly in response to their changing environment. Thus, the behavior of an individual is shaped by the behaviors of those around them, and vice versa. Transactional models of development extend previous socialization research to include bidirectional processes of influence. While there has been a call to move towards this transactional framework for understanding adolescent health risk behaviors, little previous research has specifically examined bidirectional relationships between parenting behaviors and adolescent alcohol use, and none have assessed the particularly consequential misuse of alcohol by adolescents using theoretically matched analytical techniques.

Inquiries regarding mechanisms of influence that include the nuances of socialization and transactional theories are complex, and require suitable methodological approaches. Foremost is to distinguish between sources of influence that occur between versus within individuals. These two processes reflect time-stable and time-specific relationships, respectively. This distinction provides an important delineation between influences on the study relationships that occur across time and those influences that are specific to when certain events or behaviors occur. I chose to use a latent curve model

with structured residuals to test study hypotheses for two reasons. First, this method utilizes latent factor trajectories to characterize a continual growth process reflective of the developmental nature of the behaviors and relationships examined in the study. Second, the method disentangles between and within person sources of influence to a greater extent than other methodological approaches, thus aligning the method with the theoretical basis of the study.

The first aim of this study was to determine whether prospective, reciprocal relations exist between adolescent alcohol misuse and seven parenting behaviors. The only significant finding with regard to these prospective relationships suggests that higher levels of adolescent alcohol misuse subsequently result in greater alcohol-specific monitoring behaviors by parents. The second aim of this study was to determine whether the strength and nature of reciprocal relationships between adolescent alcohol misuse and the seven parenting behaviors changed across early to middle adolescence. There was no evidence for changing reciprocal relations over time between alcohol misuse and any of the seven parenting behaviors. In addition to these main research aims, study results also highlight the extent of stability of each construct over time. Findings suggest that adolescent alcohol misuse exhibits significant stability across time; therefore, alcohol misuse at one point in time strongly influences subsequent drinking. Parental behaviors varied, however, with regard to stability as the two communication measures, permissive and negative messages, were the only constructs to demonstrate significant stability pathways. Finally, results of between-person, or interindividual processes, offer additional insights into the relationship between adolescent alcohol use and each parental socialization behavior beyond that which was hypothesized based on intraindividual processes. Interindividual results showed that the initial level of alcohol misuse was positively associated with initial levels of alcohol-specific monitoring, negative and permissive communication messages and parental alcohol use and negatively associated with parental closeness. I elaborate on each of the findings below.

Relating Study Variables via Intraindividual, Time-Specific Processes

Prospective Paths

The central hypotheses of this study are based on transactional theories of development that posit mutual influence between a person and his or her environment such that a continually changing person effects his or her context and vice versa. The prospective paths represent across-construct

influences on subsequent behavior. The prospective regressions examined in my models reflect cross-lagged effects above and beyond the effects of the stability paths and the underlying growth trajectory. Thus, this analytical strategy allows for the detection of unique cross-lagged effects, an effect that that cannot be separated using other modeling strategies such as path analyses, the dominant method used in early bidirectional research on parenting and adolescent substance use.

*Adolescent Influence on Parental Behavior***.** One significant prospective result emerged from the analyses. The finding suggests that higher levels of alcohol misuse leads to higher subsequent levels of parental monitoring of alcohol use, a relationship that remained equal in magnitude and statistically significant across all time points. This finding offers evidence of an adolescent's influence on their parent's behavior, which is the direction of influence often left out of previous research on adolescent alcohol use within the family context. One mechanism underlying this exchange may be that parents recognize a quantitative or qualitative difference in their child's behavior that raises suspicion regarding alcohol use which in turn causes the parent to specifically search for evidence of alcohol use. It should be noted that parents may or may not directly know about their child's drinking. However, there may be a collection of behaviors the adolescent exhibits that change due to his or her drinking (e.g. staying out of the house later, drop in school performance, knowledge that the child's friends are drinking alcohol) that result in parents being suspicious of their child using alcohol or other substances. The monitoring variable used in the study reflects this broader scope, measuring whether parents search for evidence of alcohol, tobacco and other drugs. Future research would benefit from more detailed questions regarding parental knowledge of child drinking and supervisory and monitoring practices specific to individual substances.

This finding contradicts hypothesized results that increases in adolescent alcohol misuse would lead parents to subsequently exhibit weaker socialization practices, including alcohol-specific monitoring. The hypothesis was based on limited previous research in which elevations in alcohol use led to decreases in later general monitoring practices[73]and alcohol-specific behavioral control.[3] Our results indicate the opposite effect for alcohol-specific monitoring: that higher levels of alcohol misuse lead to *increased* alcohol-specific monitoring. While primary prevention of alcohol use is, and should be, the central goal of practice efforts, family-based prevention programs should also prepare parents for the possibility that their teens are already drinking. Given the knowledge that early drinking has long-term

negative implications, it is imperative that prevention programs offer ways to reduce early drinking should it occur and potentially avoid the risky trajectory of behavior that often follows for those who drink alcohol at earlier ages.

Beyond alcohol-specific monitoring, no other parental socialization variables were prospectively predicted by adolescent alcohol misuse. This is in contrast to previous studies of bidirectional relationships that found increases in adolescent alcohol use led to subsequent decreases in parental attachment,[73] behavioral control[3] and monitoring[73] as well as those that found increased adolescent substance use predicted lower parental support and control.[18,19] These studies used different analytical methods than those chosen for this research, as they do not account for an underlying growth process of the constructs over time. I comment on these methodological differences and present suggestions for the lack of findings following the discussion of parental influence on adolescent behavior.

*Parental Influence on Adolescent Behavior***.** I did not find any significant prospective relations from parental behavior to adolescent alcohol misuse. These results substantiate a previous study of reciprocal effects between adolescent alcohol use and parental attachment, which found non-significant parameters linking perceived parental attachment to subsequent alcohol use.[73] Such findings suggest that the closeness of the parent-adolescent bond does not influence the development of alcohol misuse. Early bidirectional research did find full reciprocal relations between perceived parental control and support and substance use,[18] between perceived parental control and alcohol consumption, [3] and between measures of perceived parental knowledge and monitoring and alcohol use[73] as well as overall substance use;[19] however, as previously stated, these studies did not use methods that modeled underlying growth processes of the behaviors.

While our findings contradict those of early bidirectional research, this study supports recent work that demonstrates no reciprocal effects between parenting and adolescent substance use when using methods similar to those I use in this study that account for the underlying growth process of each behavior. The two methodologically similar studies found no evidence for reciprocal effects between parental knowledge and heavy episodic drinking,[20] or between parental monitoring and overall substance use.[88] Our findings therefore substantiate a limited body of evidence regarding an absence of reciprocal

relations using one-year time gaps between parental and adolescent behaviors when accounting for the developmental trajectory of each behavior.

Nevertheless, I offer several possible reasons for the lack of findings. First, it may be there is insufficient variability in the repeated measures of the parental constructs over time. One reason for the low variability, and a potential explanation for the lack of significant findings, may be due to the measurement of the parenting variables. I used parent report of all socialization variables because the three alcohol-specific variables were available only via parent report. Parental report of parenting behaviors may have led to over reporting of appropriate or socially accepted behaviors due to social desirability bias, which describes a tendency for individuals to provide an answer that would be viewed most favorably by others when completing survey questions. It may also be that as parents recall their own behaviors when responding to a question, they more easily recall behavioral choices that most align with their parenting values. Doing so would obscure reciprocal relations that occur between more negative parenting practices and adolescent alcohol misuse. It may also be that parent behavior is unaffected by youth alcohol use if the parents are unaware of their child's use. Parental knowledge of youth alcohol use, especially through disclosure by the youth themselves, is not incorporated in the parent-reported measures of their own behavior used in this study.

To address this, I conducted a sensitivity analysis using adolescent-report of the general parental socialization variables (closeness, support and behavioral control) and parental alcohol use. Child report of perceived parental behaviors is common in other studies of parenting and adolescent substance, and in some cases has been found to be more predictive of adolescent substance use than parental report of the same parenting behavior.[163] Results from the sensitivity analysis for closeness, support and parental alcohol use, however, suggest no reciprocal effects between these perceived parenting behaviors and alcohol misuse, which align with the results of the primary study analyses. Using the adolescent reported measure of behavioral control, however, I found a significant prospective relationship from adolescent alcohol misuse to subsequent general control efforts. The two final regressions, from T3 to T4 and from T4 to T5 were negative and statistically significant. This indicates that during middle adolescence, an increase in alcohol misuse by an adolescent resulted in lower subsequent perceived general control behaviors by his or her parent. This finding that perceived general parental control decreases following

elevated alcohol misuse by the teen supports previous studies of bidirectional relationships between perceived general parenting practices and adolescent substance use.[3,18] The finding for perceived general behavioral control is in the opposite direction as that found for alcohol-specific monitoring: increases in alcohol misuse resulted in *increased* parent-reported monitoring efforts specific to substance use but *decreased* adolescent-reported general behavioral control by the parent. It may be that parents shift their focus to specific behaviors as their child gets older or evidence of alcohol use appears. This potential shift is supported in that the mean values for general parenting behaviors generally declined across adolescence while the mean values for alcohol-specific behaviors increased across grades.

Second, the one-year gap between assessments may have been too long to capture adolescent and parental reactions to the other's behavior. It may be that changes in parental and adolescent behavior elicit more proximal reactions that would require closer measurement points. Even if behavior change takes longer to develop, the one-year time lag may be confounded by other contextual changes (e.g. changes in peer associations) during that time that are not captured in the analyses.

To address this limitation, I completed a sensitivity analysis assessing study relationships using a six month, rather than one-year measurement gap. This data structure was not used for the primary analysis because reciprocal relations could not be assessed within the same time frame. This is due to the design of the study in which parents were interviewed at every other wave of adolescent data collection. Nevertheless, results from this sensitivity analysis were similar to those of the primary study results. Overall, prospective relations between adolescent alcohol misuse and parental socialization behaviors were limited. The significant relationship between alcohol misuse and subsequent alcohol-specific monitoring did not hold; however adolescent alcohol misuse did result in higher ensuing parental alcohol use levels, though that result was marginally significant ($p<.10$). The lack of prospective relationships in this sensitivity analysis further suggests that these transactional relationships should be measured in close proximity to the time in which they occur.

Future research would benefit from the use of innovative methods that capture behavior in real-time. These methods, such as ecological momentary assessments and daily diary studies, enable the assessment of direct and transactional relationships within a short time frame.[164,165] This type of study design would allow for the testing of real-time dynamic familial processes relative to adolescent alcohol

misuse. Doing so would not only capture behavioral reactions and responses, but also limit confounding processes that may occur during longer measurement gaps. A related limitation of this study is that I am not able to measure whether parents know their child has been drinking. Such questions could be addressed with real-time measurement of knowledge, attitudes and behaviors.

Autoregressive Paths

The second component of the within-person model analyzed in this study is the autoregressive path between repeated measures of a construct. These are linear relationships that describe the extent to which a behavior at one time point is derived from previous iterations of that same behavior. In this way the autoregressive paths reflect the stability of a behavior over time. Importantly, the autoregressive paths must be interpreted within the context of the autoregressive latent trajectory (ALT) model, which combines the traditional autoregressive model for repeated measures with a random coefficients growth curve process. Therefore, the autoregressive path is interpreted as the influence of a behavior on a subsequent value of that behavior *beyond* the effect of the underlying growth process for that behavior.

In all models, the autoregressive path for adolescent alcohol misuse remained strongly significant (b~0.33) across time. This means that for every one unit change in child alcohol use at time T, a 0.33 increase in alcohol misuse at time T+1 is expected, net of the underlying trajectory for alcohol misuse. Every alcohol misuse autoregressive path in the model is significant which implies that an adolescent's current drinking is related to their future drinking during both early and middle adolescence. This indicates there is a strong pattern of behavior that is established during the early and middle years of adolescence, a pattern that may persist into later years of adolescence. This finding supports previous research that early drinking has persistent effects through adolescence and into young adulthood.[24,27,28,166] Such a finding is important for prevention efforts given that drinking becomes more prevalent in later years of adolescence,[110] confirming the efforts of many prevention programs to prevent and/or reduce early adolescent drinking and its subsequent negative implications for the adolescent.

Among the parental socialization behaviors, stability paths were positive and statistically significant for the two communication measures, permissive and negative messages about alcohol use. The degree to which parents talk to their child about alcohol use influences their communication about that topic at a later time. For both permissive and negative messages, the autoregressive parameter was

equal in magnitude across grades. These findings indicate that what parents say in early adolescence continues to influence their communication across the middle adolescent years. Our results are similar to previous research that found significant stability with regard to the quality and frequency of communication about alcohol across adolescence,[96,167] though these studies did not distinguish between specific messages being communicated about alcohol.

I did not find significant autoregressive pathways among repeated measures of the other socialization behaviors (closeness, support, behavioral control, parental alcohol use and alcohol-specific monitoring). The analytical method used in this study determined the trajectory (including an intercept, linear and quadratic slope) of a behavior prior to adding the autoregressive pathways. For socialization behaviors other than the two communication messages, the underlying growth process accounted for a vast majority of the model fit to the data, which was exceptional prior to adding the autoregressive paths (e.g. chi square values under 15, CFI and TLI values of .99, RMSEA values below .03). Due to this initial strong fit, the addition of the autoregressive paths did not significantly improve model fit. Our findings contrast previous studies in which significant stability paths were identified between repeated measures of general parenting behaviors, though importantly, none of these studies estimated an underlying growth process.[18,73,88]

Relating Study Variables via Interindividual, Time-Stable Processes

My hypotheses centered on the within-person components of the models I tested; that is, the time-specific relationships between each parent-adolescent dyad. Results that reflect between-person, or effects across parent-adolescent dyads, offer additional insight into the relationship between parental socialization behaviors and adolescent alcohol misuse. Between-person effects were assessed based on the significance of the covariation between the latent constructs of the two variables. These results reflect overall, time-stable relationships between alcohol misuse and parental behavior, which indicate who may be most at risk for alcohol misuse.

A significant positive covariance was found between the intercepts of alcohol misuse and negative and permissive messages, parental alcohol use and alcohol-specific monitoring. Therefore, a higher initial level of alcohol misuse is related to higher initial levels of these parental socialization behaviors, and vice versa. Findings regarding alcohol communication echo previous studies that suggest

greater communication about alcohol, particularly negative consequences, results in greater risk for initiation and escalation of alcohol use during adolescence.[99-101] It may be that an adolescents' alcohol use against their parents' advice is an act of rebellion. Next, our findings suggest that parents who have higher initial levels of alcohol use themselves also have children with higher initial levels of alcohol misuse. This positive relationship between parent and child drinking is well replicated in the literature.[11,105-110] This relationship reflects the modeling assumption of social learning theory,[60] that children learn and mimic the behaviors of their parents. Additionally, parents who drink may also hold more lenient beliefs regarding their child's use of alcohol or have greater accessibility of alcohol in the home, both of which increase an adolescent's risk of drinking.[112-114]

The finding that initial levels of adolescent alcohol misuse and alcohol-specific monitoring were positively related aligns with the time-specific relationship between the two constructs found in this study. Thus, this effect holds on average across individuals at baseline, and across time within parent-adolescent dyads. Across parent-child dyads, children who misuse alcohol more initially also have parents who monitor for their use at higher initial levels; and within specific parent-child dyads, an adolescent's alcohol misuse above their own average subsequently leads to more alcohol-specific monitoring by their parents.

A significant negative covariance was found between the intercepts of parental closeness and alcohol misuse, indicating that on average, adolescents who had higher initial levels of alcohol misuse had parents who reported lower levels of closeness towards their child. This finding supports previous research, which suggests that adolescent drinking is associated with lower levels of perceived closeness to parents.[9] Adolescents who drink alcohol may create emotional distance from their parents to hide deviant behavior such as their use of alcohol. Additionally, adolescents may strengthen the bond they have with peers who also use alcohol rather than seeking emotional support from their parents. The relationship between closeness and alcohol misuse can also be viewed in the alternative direction: parents with closer bonds to their child on average have children who on average misuse alcohol less. A closer bond with children allows parents to transmit their pro-social beliefs, such as abstaining from alcohol use, more successfully. This finding supports previous cross-sectional research that strong

parent-child bonds increase the likelihood of delayed initiation of alcohol use and lower levels of use during adolescence.[4,5,71,72]

Strengths, Limitations, and Future Directions

This study contributes to what we know regarding parental socialization and adolescent alcohol misuse due to several important strengths. First, the study utilizes a longitudinal dataset to examine the hypothesized relationships across early and middle adolescence. These are critical developmental periods given that initiation and use of alcohol at early ages puts adolescents at higher risk for detrimental short and long-term consequences. Second, this is the first study to examine bidirectional relationships between numerous parental socialization behaviors and adolescent alcohol misuse. Doing so offers insight into the mutual influence of these behaviors across adolescence. Third, the study uses rigorous analytical methods to appropriately and simultaneously model multiple effect mechanisms. Using such methods allowed me to test specific theoretical relationships and provides clearer insight into the processes that link adolescent and parent behavior. Even with such strengths, the results of this study must be interpreted within the context of several limitations, which offer insights for future research.

First, future research would benefit from multiple-informant reporting of alcohol-specific parental behaviors, including the other parent. For this study, over 90% of the parents interviewed were the mother or mother surrogate. The parenting style and specific parenting behaviors of the father may differ from the mother.[168] Investigation of socialization behaviors unique to the father, and the joint influence of multiple parenting behaviors within a family is warranted. Additionally, as previously discussed, the choice of informant has implications for the interpretation of relationships between adolescent and parental behavior. For this study, the three alcohol-specific behaviors were only available via parental report. Our findings suggest the need to further evaluate these specific behaviors beyond the general behaviors typically examined in relation to adolescent substance use. To do so, it will be important to measure alcohol-specific behaviors by child and parental report. For all socialization behaviors, the ability to assess bidirectional relationships through multiple informants would enhance our understanding of the mechanisms that underlie transactional familial processes.

Second, I tested the misuse of alcohol during adolescence due to the heightened severity of the consequences for this type of behavior. My examination of alcohol misuse during this time is important

given that adolescents who misuse alcohol at an early age are most at risk for these negative consequences, and therefore may benefit most from prevention and intervention efforts. However, this research could be extended to examine other measures of alcohol use such as initiation and frequency of use to capture additional groups of young drinkers. Additionally, while retrospective report of substance use during adolescence is common for the field, measurement that utilizes more in-depth assessments of substance use might offer greater variability of the adolescent alcohol use measure, which is a benefit for analytical techniques such as the LCM-SR that combine both growth curves and autoregressive models of time-varying repeated measures in the same modeling framework.

Finally, future research would benefit from more precise measurement of parental monitoring behaviors. Given the results of this study, parental control and monitoring practices deserve further inquiry. Previous scholars have differentiated components of monitoring to include the rules and restrictions parents place on what their child can and cannot do as well as parental knowledge of their child's activities. The source of that knowledge, whether solicited by the parent or disclosed by the child, is also an important factor to consider when measuring control and monitoring constructs.[21,92] Detailed measurement and analysis of these multiple components of parental monitoring and control would enhance our understanding of how these behaviors influence adolescent alcohol use, and vice versa.

Conclusions

In summary, I provide a rigorous examination of novel pathways that assesses bidirectional relationships between adolescent alcohol misuse and both general and alcohol-specific parental socialization behaviors. While I found limited significant results, I demonstrate that one parental behavior, alcohol-specific monitoring, is influenced by their child's behavior, the direction of influence often left out of previous socialization research. Additionally, this study corroborates growing evidence that alcohol-specific parenting behaviors differ from their more general measurements, thus indicating a need to more closely examine parental behaviors specific to alcohol use. Future research regarding family-based prevention of youth alcohol use is needed to identify effective strategies for parents to respond to their child's drinking should it occur that in turn decrease an adolescent's risk for drinking. These results provide a basis for future research to examine more dynamic transactional processes between parents and adolescents relative to alcohol use.

REFERENCES

1. Ellickson PL, McGuigan KA, Adams V, Bell RM, Hays RD. Teenagers and alcohol misuse in the United States: By any definition, it's a big problem. *Addiction.* 1996;91(10):1489-1503.

2. Foxcroft D, Ireland D, Lowe G, Breen R. Primary prevention for alcohol misuse in young people. *Cochrane DB Syst Rev.* 2002;3.

3. van der Zwaluw CS, Scholte RH, Vermulst AA, Buitelaar JK, Verkes RJ, Engels RC. Parental problem drinking, parenting, and adolescent alcohol use. *J Behav Med.* 2008;31(3):189-200.

4. Kuntsche E, van der Vorst H, Engels RC. The earlier the more? Differences in the links between age at first drink and adolescent alcohol use and related problems according to quality of parent-child relationships. *J Stud Alcohol Drugs.* 2009;70(3):346-354.

5. Sokol-Katz J, Dunham R, Zimmerman R. Family structure versus parental attachment in controlling adolescent deviant behavior: a social control model. *Adolescence.* 1997;32(125):199-215.

6. Barnes GM. The development of adolescent drinking behavior: an evaluative review of the impact of the socialization process within the family. *Adolescence.* 1977;12(48):571-591.

7. Stice E, Barrera M, Chassin L. Relation of parental support and control to adolescents' externalizing symptomatology and substance use: a longitudinal examination of curvilinear effects. *J Abnorm Child Psych.* 1993;21(6):609-629.

8. Willoughby T, Hamza C. A longitudinal examination of the bidirectional associations among perceived parenting behaviors, adolescent disclosure and problem behavior across the high school years. *J Youth Adolescence.* 2011;40(4):463-478.

9. Barnes GM, Reifman AS, Farrell MP, Dintcheff BA. The effects of parenting on the development of adolescent alcohol misuse: a six-wave latent growth model. *J Marriage Fam.* 2000;62(1):175-186.

10. Zhang L, Welte JW, Wieczorek WF. The influence of parental drinking and closeness on adolescent drinking. *J Stud Alcohol Drugs.* 1999;60(2):245-251.

11. Ary DV, Duncan TE, Duncan SC, Hops H. Adolescent problem behavior: the influence of parents and peers. *Behav Res Ther.* 1999;37(3):217-230.

12. Handley ED, Chassin L. Alcohol-specific parenting as a mechanism of parental drinking and alcohol use disorder risk on adolescent alcohol use onset. *J Stud Alcohol Drugs.* 2013:74(5):684-693.

13. Anderson KE, Lytton H, Romney DM. Mothers' interactions with normal and conduct-disordered boys: who affects whom? *Dev Psychol.* 1986:22(5), 604-609.

14. Pardini DA, Fite PJ, Burke, JD. Bidirectional associations between parenting practices and conduct problems in boys from childhood to adolescence: the moderating effect of age and African-American ethnicity. *J Abnorm Child Psych.* 2008;36(5):647-662.

15. Larsson H, Viding E, Rijsdijk FV, Plomin R. Relationships between parental negativity and childhood antisocial behavior over time: a bidirectional effects model in a longitudinal genetically informative design. *J Abnorm Child Psych.* 2008;36(5):633-645.

16. Gross HE, Shaw DS, Moilanen KL. Reciprocal associations between boys' externalizing problems and mothers' depressive symptoms. *J Abnorm Child Psych.* 2008;36(5):693-709.

17. Vuchinich S, Bank L, Patterson G. Parenting, peers, and the stability of antisocial behavior in preadolescent boys. *Dev Psychol.* 1992;28(3):510-1521.

18. Stice E, Barrera M. A longitudinal examination of the reciprocal relations between perceived parenting and adolescents' substance use and externalizing behaviors. *Dev Psychol.* 1995;31(2):322-334.

19. Coley RL., Votruba-Drzal E, Schindler HS. Trajectories of parenting processes and adolescent substance use: reciprocal effects. *J Abnorm Child Psych.* 2008;36(4):613-625.

20. Abar CC, Jackson KM, Wood M. Reciprocal relations between perceived parental knowledge and adolescent substance use and delinquency: the moderating role of parent–teen relationship quality. *Dev Psychol.* 2014;50(9):2176-2187.

21. Kerr M, Stattin H. What parents know, how they know it, and several forms of adolescent adjustment: further support for a reinterpretation of monitoring. *Dev Psychol.* 2000;36(3):366-380.

22. Kakihara F, Tilton-Weaver L. Adolescents' interpretations of parental control: differentiated by domain and types of control. *Child Dev. 2009;80*(6):1722-1738.

23. US Department of Health and Human Services. *The surgeon general's call to action to prevent and reduce underage drinking*. Rockville, MD: US Department of Health and Human Services, Office of the Surgeon General. 2007.

24. Gruber E, DiClemente R, Anderson M, Lodico M. Early drinking onset and its association with alcohol use and problem behavior in late adolescence. *Preventive Medicine.* 2006;25(3):293-300.

25. Grunbaum JA, Kann L, Kinchen S, Ross J, Hawkins J, Lowry R, Harris W, McManus T, Chyen D, Collins J. Youth risk behavior surveillance--United States, 2003. *Mortality and Morbidity Surveillance Summary.* 2003;53(2):1-96.

26. Faden VB. Trends in initiation of alcohol use in the United States 1975 to 2003. *Alcohol Clin Exp Res.* 2006;30(6):1011-1022.

27. Hawkins JD, Graham JW, Maguin E, Abbott R, Hill KG, Catalano RF. Exploring the effects of age of alcohol use initiation and psychosocial risk factors on subsequent alcohol misuse. *J Stud Alcohol.* 1997;58(3);280-290.

28. Ellickson PL, Tucker J, Klein D. Ten-year prospective study of public health problems associated with early drinking. *Pediatrics.* 2003;111(5):949-955.

29. DeWit DJ. Age at first alcohol use: A risk factor for the development of alcohol disorders. *Am J Psychiat.* 2000;157(5):745-750.

30. National Institute on Alcohol Abuse and Alcoholism. Helping patients who drink too much: A clinician's guide [Internet]. [Cited 2016 Jan 15]. Available from: http://pubs.niaaa.nih.gov/publications/Practitioner/CliniciansGuide2005/clinicians_guide.htm

31. National Institute on Alcohol Abuse and Alcoholism. Drinking levels defined [Internet]. [Cited 2016 Jan 15]. Available from: http://www.niaaa.nih.gov/alcohol-health/overview-alcohol-consumption/moderate-binge-drinking

32. Pacific Institute for Research and Evaluation. Drinking in America: myths, realities and prevention policy [Internet]. [Cited 2016 Jan 15]. Available from: http://www.lhc.ca.gov/lhc/drug/DrinkinginAmericaMosherSep26.pdf

33. Miller JW, Naimi TS, Brewer RD, Jones SE. Binge drinking and associated health risk behaviors among high school students. *Pediatrics.* 2007;119(1):76-85.

34. Hill KG, White HR, Chung I, Hawkins JD, Catalano RF. Early adult outcomes of adolescent binge drinking: person-and variable-centered analyses of binge drinking trajectories. *Alcohol Clin Exp Res.* 2000;24(6):892-901.

35. Escobedo LG, Chorba TL, Waxweiler R. Patterns of alcohol use and the risk of drinking and driving among US high school students. *Am J Public Health.* 1995;85(7):976-978.

36. Shope JT, Bingham CR. Teen driving: motor-vehicle crashes and factors that contribute. *Am J Prev Med.* 2008;35(3):S261-S271.

37. Arata CM, Stafford J, Tims MS. High school drinking and its consequences. *Adolescence.* 2003;38(151):567-579.

38. Champion HL, Foley KL, Durant RH, Hensberry R, Altman D, Wolfson M. Adolescent sexual victimization, use of alcohol and other substances, and other health risk behaviors. *J Adolescent Health.* 2004;35(4):321-328.

39. Howard DE, Qi WM. Risk procedures of adolescent girls who were victims of dating violence. *Adolescence.* 2003;38(149):1-14.

40. Reyes HL, Foshee VA, Bauer DJ, Ennett ST. Developmental associations between adolescent alcohol use and dating aggression. *J Res Adolescence. 2012;22*(3):526-541.

41. Windle M. Suicidal behaviors and alcohol use among adolescents: a developmental psychopathology perspective. *Alcohol Clin Exp Res.* 2004;28(s1):29S-37S.

42. McCarty CA, Ebel BE, Garrison MM, DiGiuseppe DL, Christakis DA, Rivara FP. Continuity of binge and harmful drinking from late adolescence to early adulthood. *Pediatrics.* 2004;114(3):714-719.

43. Oetting ER, Donnermeyer JF. Primary socialization theory: the etiology of drug use and deviance. *Subst Use Misuse.* 1998;33(4):995-1026.

44. Maccoby E, Martin J. Socialization in the context of the family: parent-child interaction. In: Mussen P, Hetherington M, editors. Handbook of child psychology: Vol. 4. Socialization, personality and social development. New York, NY: John Wiley & Sons; 1983. p. 1-101.

45. Jessor R, Graves T, Hanson R, Jessor S. Society, personality and deviant behavior- a study of a tri-ethnic community. New York, NY: Holt, Rinehart and Winston, Inc; 1968.

46. Baldwin A. Behavior and Child Development. New York, NY: Dryden Press; 1955.

47. Baldwin AL. Socialization and the parent-child relationship. *Child Dev.* 1948;19(3):127-136.

48. Baumrind D. The development of instrumental competence through socialization. In Pick A, editor. Minnesota Symposium on Child Psychology. Minneapolis, MI: University of Minnesota Press; 1973. p.3-46.

49. Baumrind D. Current patterns of parental authority. *Dev Psychol.* 1971;4(1p2), 1-103.

50. Steinberg L. We know some things: parent–adolescent relationships in retrospect and prospect. *J Res Adolescence.* 2001;11(1):1-19.

51. Steinberg L, Elmen JD, Mounts NS. Authoritative parenting, psychosocial maturity, and academic success among adolescents. *Child Dev.* 1989;60(6):1424-1436.

52. Darling N, Steinberg L. Parenting style as context: an integrative model. *Psychol Bull.* 1993;113(3):487-496.

53. Maccoby EE. The role of parents in the socialization of children: an historical overview. *Dev Psychol.* 1992;28(6):1006-1017.

54. Bell RQ. A reinterpretation of the direction of effects in studies of socialization. *Psychol Review.* 1968;75(2):81-95.

55. Mischel W. Toward a cognitive social learning reconceptualization of personality. *Psychol Review.* 1973;80(4):252-283.

56. Kuczynski L, Marshall S, Schell K. Value socialization in a bidirectional context. In: Grusec, JE, Kuczynski L, editors. Parenting and the Internalization of Values: A Handbook of Contemporary Theory. New York, NY: Wiley & Sons, Inc; 1997. p.23-50.

57. Sameroff, A. Transactional models in early social relations. *Hum Dev.* 1975;18(12):65-78.

58. Sameroff AJ, MacKenzie M. Research strategies for capturing transactional models of development: the limits of the possible. *Dev Psychopathol.* 2003;15(03):613-640.

59. Lerner RM, Spanier GB. A dynamic interactional view of child and family development. In: Lerner RM, Spanier GB, editors. Child influences on marital and family interaction: a life-span perspective. San Francisco: Academic Press; 1978. p.1-22.

60. Bandura, A. Social learning theory. Englewood Cliffs, NJ: Prentice-Hall; 1977.

61. Cairns R. Social interactional methods: An introduction. In Cairns R, editor. The analysis of social interactions: methods, issues and illustrations. Hillsdale, NJ: Erlbaum; 1979.

62. Patterson GR. Performance models for antisocial boys. *Am Psychol.* 1986;41(4):432-444.

63. Patterson GR, Dishion TJ, Bank L. Family interaction: a process model of deviancy training. *Aggressive Behav.* 1984;10(3):253-267.

64. Patterson GR. Coercive family processes. Eugene, OR: Castalia; 1982.

65. Patterson GR, DeBaryshe BD, Ramsey E. A developmental perspective on antisocial behavior. *American Psychol.* 1989;44:329-335.

66. Hirschi T. Causes of delinquency. Berkeley, CA: University of California Press; 1969.

67. Petraitis J, Flay B, Miller T. Reviewing theories of adolescent substance use: organizing pieces in the puzzle. *Psychol Bull.* 1995;117(1):67-86.

68. Elliott D, Huiziga D, Ageton S. Explaining delinquency and drug use. Beverly Hills, CA: Sage Publications; 1985.

69. Grusec JE, Goodnow JJ, Kuczynski L. New directions in analyses of parenting contributions to children's acquisition of values. *Child Dev.* 2000;71(1):205-211.

70. Chassin L, Handley ED. Parents and families as contexts for the development of substance use and substance use disorders. *Psychol Addict Behav.* 2006;20(2):135-137.

71. Oxford ML, Harachi TW, Catalano RF, Abbott RD. Preadolescent predictors of substance initiation: a test of both the direct and mediated effect. *Am J Drug Alcohol Ab.* 2001;27(4):599-616.

72. Tyler KA, Stone RT, Bersani B. Examining the changing influence of predictors on adolescent alcohol misuse. *J Child Adoles Subst.* 2007;16(2):95-114.

73. van der Vorst H, Engels RC, Meeus W, Deković M. Parental attachment, parental control, and early development of alcohol use: a longitudinal study. *Psychol Addict Behav.* 2006;20(2):107-116.

74. Bell NJ, Forthun LF, Sun S. Attachment, adolescent competencies, and substance use: developmental considerations in the study of risk behaviors. *Subst Use Misuse.* 2000;35(9):1177-1206.

75. Buist KL, Deković M, Meeus W, van Aken MA. The reciprocal relationship between early adolescent attachment and internalizing and externalizing problem behaviour. *J Adolescence.* 2004;27(3):251266.

76. Barnes GM, Farrell MP. Parental support and control as predictors of adolescent drinking, delinquency, and related problem behaviors. *J Marriage Fam.* 1992;54(4):763-776.

77. Rollins R, Thomas D. Parental support, power, and control techniques in the socialization of children. In: Burr W, Hill R, Nye I, Reiss I, editors. Contemporary theories about the family (1st Edition). New York, NY: Free Press; 1979. p. 317-364.

78. Barnes GM, Farrell MP, Cairns A. Parental socialization factors and adolescent drinking behaviors. *J Marriage Fam.* 1986;48(1):27-36.

79. Windle M, Miller-Tutzauer C, Domenico D. Alcohol use, suicidal behavior, and risky activities among adolescents. *J Res Adolescence.* 1992;2(4):317-330.

80. Anderson AR, Henry CS. Family system characteristics and parental behaviors as predictors of adolescent substance use. *Adolescence.* 1994;29(114):405-420.

81. Baumrind D. The influence of parenting style on adolescent competence and substance use. *The J Early Adolescence.* 1991;11(1):56-95.

82. Dishion TJ, Loeber R. Adolescent marijuana and alcohol use: the role of parents and peers revisited. *Am J Drug Alcohol Ab.* 1985;11(1-2):11-25.

83. Akers RL, Krohn MD, Lanza-Kaduce L, Radosevich M. Social learning and deviant behavior: a specific test of a general theory. *Am Sociol Rev.* 1979;44(4):636-655.

84. van der Zwaluw CS, Scholte RJ, Vermulst AA, Buitellar JK, Verkes RJ, Engels RC. Parental problem drinking, parenting, and adolescent alcohol use. *J Behav Med.* 2008;31(3):189-200.

85. Stavrinides P, Georgiou S, Demetriou A. Longitudinal associations between adolescent alcohol use and parents' sources of knowledge. *Brit J Dev Psychol.* 2010;28(3):643-655.

86. Van Zundert RMP, van der Vorst H, Vermulst AA, Engels RC. Pathways to alcohol use among Dutch students in regular education and education for adolescents with behavioral problems: the role of parental alcohol use, general parenting practices, and alcohol-specific parenting practices. *J Fam Psychol.* 2006;20(3):456-467.

87. Laird RD, Pettit GS, Bates JE, Dodge KA. Parents' monitoring-relevant knowledge and adolescents' delinquent behavior: evidence of correlated developmental changes and reciprocal influences. *Child Dev.* 2003;74(3):752-768.

88. Elkins SR, Fite PJ, Moore TM, Lochman JE, Wells KC. Bidirectional effects of parenting and youth substance use during the transition to middle and high school. *Psychol Addict Behav.* 2014;28(2):475-486.

89. Kerr M, Stattin H, Burk WJ. A reinterpretation of parental monitoring in longitudinal perspective. *J Res Adolescence.* 2010;20(1):39-64.

90. Jackson C, Henriksen L, Dickinson D. Alcohol-specific socialization, parenting behaviors and alcohol use by children. *J Stud Alcohol Drugs.* 1999;60(3):362-367.

91. van der Vorst H, Engels RC, Meeus W, Deković M, van Leeuwe J. The role of alcohol-specific socialization in adolescents' drinking behaviour. *Addiction.* 2005;100(10):1464-1476.

92. Stattin H, Kerr M. Parental monitoring: a reinterpretation. *Child Dev.* 2000;71(4):1072-1085.

93. van der Vorst H, Burk WJ, Engels RC. Do parents and best friends influence the normative increase in adolescents' alcohol use at home and outside the home? *J Stud Alcohol Drugs.* 2010;71(1):105-114.

94. van der Vorst H, Engels RC, Deković M, Meeus W, Vermulst AA. Alcohol-specific rules, personality and adolescents' alcohol use: a longitudinal person-environment study. *Addiction.* 2007;102(7):1064-1075.

95. van der Vorst H, Engels RC, Meeus W, Deković M. The impact of alcohol-specific rules, parental norms about early drinking and parental alcohol use on adolescents' drinking behavior. *J Child Psychol Psych.* 2006;47(12):1299-1306.

96. Koning IM, van den Eijnden Regina JM, Verdurmen JE, Engels RC, Vollebergh WA. Developmental alcohol-specific parenting profiles in adolescence and their relationships with adolescents' alcohol use. *J Youth Adolescence.* 2012;41(11):1502-1511.

97. Mares SH, Lichtwarck-Aschoff A, Engels RC. Alcohol-specific parenting, adolescent alcohol use and the mediating effect of adolescent alcohol-related cognitions. *Psychol Health.* 2013;28(7):833-848.

98. van den Eijnden R, van de Mheen D, Vermulst A, Vet R. Alcohol-specific parenting and adolescents' alcohol-related problems: the interacting role of alcohol availability at home and parental rules. *J Stud Alcohol Drugs.* 2011;72(3):408-417.

99. Ennett ST, Bauman KE, Foshee VA, Pemberton M, Hicks KA. Parent-child communication about adolescent tobacco and alcohol use: what do parents say and does it affect youth behavior? *Journal Marriage Fam.* 2001;63(1):48-62.

100. Andrews JA, Hops H, Ary D, Tildesley E, Harris J. (1993). Parental influence on early adolescent substance use specific and nonspecific effects. *J Early Adolescence.* 1993;13(3):285-310.

101. Spijkerman R, van den Eijnden RJ, Huiberts A. Socioeconomic differences in alcohol-specific parenting practices and adolescents' drinking patterns. *Eur Addict Res.* 2008;14(1):26-37.

102. Reimuller A, Hussong A, Ennett ST. The influence of alcohol-specific communication on adolescent alcohol use and alcohol-related consequences. *Prev Sci.* 2011;12(4):389-400.

103. Zehe JM, Colder CR. A latent growth curve analysis of alcohol-use specific parenting and adolescent alcohol use. *Addictive Behav.* 2014;39(12):1701-1705.

104. Ary DV, Tildesley E, Hops H, Andrews J. The influence of parent, sibling, and peer modeling and attitudes on adolescent use of alcohol. *Sub Use Misuse.* 1993;28(9):853-880.

105. Alati R, Baker P, Betts KS, Connor JP, Little K, Sanson A, Olsson C. The role of parental alcohol use, parental discipline and antisocial behaviour on adolescent drinking trajectories. *Drug Alcohol Depend.* 2014;134:178-184.

106. Peterson PL, Hawkins JD, Abbott RD, Catalano RF. Disentangling the effects of parental drinking, family management, and parental alcohol norms on current drinking by black and white adolescents. *J Res Adolescence.* 1994;4(2):203-227.

107. Chassin L, Rogosch F, Barrera M. Substance use and symptomatology among adolescent children of alcoholics. *J Abnorm Psychol.* 1991;100(4):449-463.

108. van der Vorst H, Vermulst AA, Meeus WHJ, Deković M, Engels RC. Identification and prediction of drinking trajectories in early and mid-adolescence. *J Clin Child Adolesc.* 2009;38(3):329-341.

109. White HR, Johnson V, Buyske S. Parental modeling and parenting behavior effects on offspring alcohol and cigarette use: a growth curve analysis. *J Subst Abuse.* 2000;12(3):287-310.

110. Duncan SC, Duncan TE, Strycker LA. Alcohol use from ages 9 to 16: a cohort-sequential latent growth model. *Drug Alcohol Depend.* 2006;81(1):71-81.

111. Rüütel E, Sisask M, Värnik A, Värnik P, Carli V, Wasserman C, et al. Alcohol consumption patterns among adolescents are related to family structure and exposure to drunkenness within the family: results from the SEYLE project. *Int J Environ Res and Publ Health.* 2014;11(12):12700-12715.

112. Vermeulen-Smit E, Koning IM, Verdurmen JE, van der Vorst H, Engels RC, Vollebergh WA. The influence of paternal and maternal drinking patterns within two-partner families on the initiation and development of adolescent drinking. *Addict Behav.* 2012;37(11):1248-1256.

113. Latendresse SJ, Rose RJ, Viken RJ, Pulkkinen L, Kaprio J, Dick DM. Parenting mechanisms in links between parents' and adolescents' alcohol use behaviors. *Alcohol Clin Exp Res.* 2008;32(2):322-330.

114. Komro KA, Maldonado-Molina MM, Tobler AL, Bonds JR, Muller KE. Effects of home access and availability of alcohol on young adolescents' alcohol use. *Addiction.* 2007;102(10):1597-1608.

115. Steinberg L, Morris AS. Adolescent development. *Journal of Cognitive Education and Psychology.* 2001;2(1):55-87.

116. Oudekerk B, Allen J, Hessel E, Molloy L. The cascading development of autonomy and relatedness from adolescence to adulthood. *Child Dev.* 2014;82(2):472-485.

117. Silk JS, Morris AS, Kanaya T, Steinberg L. Psychological control and autonomy granting: opposite ends of a continuum or distinct constructs? *J Res Adolescence.* 2003;13(1):113-128.

118. Caplan R. Person-environment fit: past, present, and future. In: Cooper C, editor. Stress research. New York, NY: Wiley & Sons, Inc; 1983. p. 35-78.

119. Caplan RD, Harrison RV. Person-environment fit theory: some history, recent developments, and future directions. *J Soc Issues.* 1993;49(4):253-275.

120. Eccles JS, Buchanan CM, Flanagan C, Fuligni A, Midgley C, Yee D. Control versus autonomy during early adolescence. *J Soc Issues.* 1991;47(4):53-68.

121. Grotevant HD, Cooper CR. Individuality and connectedness in adolescent development. In: Skoe E, von der Lippe A, editors. Personality Development in Adolescence: A Cross National and Life Span Perspective. London: Rutledge Press; 1998. p.3-37.

122. Grotevant HD. Adolescent development in family contexts. In: Damon W, Eisenberg N, editors. Handbook of Child Psychology. Hoboken, NJ: Wiley & Sons; 1998. pp.1097-1149.

123. Grotevant HD, Cooper CR. Individuation in family relationships. *Hum Dev.* 1986;29(2):82-100.

124. Steinberg L. Reciprocal relation between parent-child distance and pubertal maturation. *Dev Psychol.* 1988;24(1):122-128.

125. Collins WA, Harris ML, Susman A. Parenting during middle childhood. In: Bornstein MH, editor. Handbook of parenting, Vol 1: Children and parenting. Hillside, NJ: Lawrence Erlbaum Associates; 1995. p. 65-89.

126. Collins W, Laursen B. Parent-adolescent relationships and influences. In: Lerner R, Steinberg L, editors. *Handbook of adolescent psychology* (2nd Edition). Hoboken, NJ: John Wiley & Sons, Inc; 2004. p. 331-362.

127. Steinberg L, Silk JS. Parenting adolescents. In: Bornstein MH, editor. Handbook of parenting, Vol 1: Children and parenting. Hillside, NJ: Lawrence Erlbaum Associates; 2002. p. 65-89.

128. Smetana J, Crean HF, Campione-Barr N. Adolescents' and parents' changing conceptions of parental authority. *New Dir Child Adoles Dev.* 2005;2005(108):31-46.

129. Smetana JG. Adolescents' and parents' conceptions of parental authority. *Child Dev.* 1988;65(4):321-335.

130. Smetana JG. Concepts of self and social convention: adolescents' and parents' reasoning about hypothetical and actual family conflicts. Minnesota Symposia on Child. Minneapolis, MI: University of Minnesota Press; 1988. p. 79-122.

131. Laursen B, Williams VA. Perceptions of interdependence and closeness in family and peer relationships among adolescents with and without romantic partners. *New Dir Child Adoles Dev.* 1997;1997(78):3-20.

132. Allen JP, Land D. Attachment in adolescence. In: Cassidy J, Shaver P, editors. Handbook of Attachment: Theory, Research, and Clinical Applications. New York: Guilford Press; 1999. pp. 319-335.

133. Berndt TJ. Developmental changes in conformity to peers and parents. *Dev Psychol.* 1979;15(6):608-616.

134. Berndt TJ. Obtaining support form friends during childhood and adolescence. In: Belle D, editor. Children's Social Networks and Supports. Hoboken, NJ: John Wiley & Sons, Inc; 1990. p.308-331.

135. Savin-Williams RC, Berndt TJ. Friendship and peer relations. In: Belle D, editor. Children's Social Networks and Supports. Hoboken, NJ: John Wiley & Sons, Inc.; 1990. p.207-237.

136. Jessor R, Jessor S. Problem behavior and psychosocial development: A longitudinal study of youth. New York: Academic Press; 1977.

137. Windle M. Parental, sibling, and peer influences on adolescent substance use and alcohol problems. *Appl Dev Sci.* 2000;4(2):98-110.

138. Curran PJ, Stice E, Chassin L. The relation between adolescent alcohol use and peer alcohol use: a longitudinal random coefficients model. *J Consult Clin Psych.* 1997;65(1):130-140.

139. Larson RW, Richards MH, Moneta G, Holmbeck G, Duckett E. Changes in adolescents' daily interactions with their families from ages 10 to 18: Disengagement and transformation. *Dev Psychol.* 1996;32(4):744-754.

140. Barnes GM. Impact of the family on adolescent drinking patterns. In: Collins R, Leonard K, Searles J, editors. Impact of the family on adolescent drinking patterns. Guildford Press: London; 1990. p. 137-161.

141. Barnes G, Windle M. Family factors in adolescent alcohol and drug abuse. *Pediatrics.* 1987;14(12):13-18.

142. Fisher L, Bauman K. Influence and selection in the friend adolescent relationships: Findings from studies of adolescent smoking and drinking. *J Appl Social Psychol.* 1988;18(4):289-314.

143. Spijkerman R, van den Eijnden R, Overbeek G, Engels RC. The impact of peer and parental norms and behavior on adolescent drinking: the role of drinker prototypes. *Psychol Health.* 2007;22(1):729.

144. Nash SG, McQueen A, Bray J. Pathways to adolescent alcohol use: family environment, peer influence, and parental expectations. *J Adolescent Health.* 2005;37(1):19-28.

145. Ennett ST, Foshee VA., Bauman KE, Hussong A, Cai L, Reyes HL, Farris R, Hipp J, Durant R. The social ecology of adolescent alcohol misuse. *Child Dev.* 2008;79(6):1777-1791.

146. Thissen D, Nelson L, Rosa K, McLeod LD. Item response theory for items scored in more than two categories. In: Thissen D, Wainer H, editors. Test scoring. Hillsdale, NJ: Lawrence Erlbaum Associates; 2001. p. 141-186.

147. Rubin DB. Inference and missing data. *Biometrika.* 1976;63(3):581-592.

148. Heitjan DF, Basu S. Distinguishing "missing at random" and "missing completely at random". *The Am Stat.* 1996;50(3):207-213.

149. Duncan SC, Duncan TE. Modeling incomplete longitudinal substance use data using latent variable growth curve methodology. *Multivar Behav Res.* 1994;29(4):313-338.

150. Miyazaki Y, Raudenbush S. Tests for linkage of multiple cohorts in an accelerated longitudinal design. *Psychol Methods.* 2000;5(1):44-63.

151. Raudenbush S, Bryk A. Hierarchical linear models: Applications and data analysis (2nd Edition). Thousand Oaks, CA: Sage Publications; 2002.

152. Curran PJ, Bauer DJ. The disaggregation of within-person and between-person effects in longitudinal models of change. *Annu Rev Psychol.* 2011;62:583-619.

153. Curran PJ, Willoughby MT. Implications of latent trajectory models for the study of developmental psychopathology. *Dev Psychopathol.* 2003;15(3):581-612.

154. Hussong AM, Cai L, Curran PJ, Flora DB, Chassin L, Zucker RA. Disaggregating the distal, proximal, and time-varying effects of parent alcoholism on children's internalizing symptoms. *J Abnorm Child Psych.* 2008;36(3):335-346.

155. Hoffman L, Stawski RS. Persons as contexts: evaluating between-person and within-person effects in longitudinal analysis. *Res Hum Dev.* 2009;6(2-3):97-120.

156. Curran PJ, Howard AL, Bainter SA, Lane ST, McGinley JS. The separation of between-person and within-person components of individual change over time: a latent curve model with structured residuals. *J Consult Clin Psychol.* 2013;82(5):879-894.

157. Bollen KA, Curran PJ. Autoregressive latent trajectory (ALT) models a synthesis of two traditions. *Sociol Method Res.* 2004;32(3):336-383.

158. Bollen KA, Curran PJ. Latent curve models: A structural equations perspective. Hoboken, NJ: John Wiley & Sons, Inc; 2006.

159. Rindskopf D. Using phantom and imaginary latent variables to parameterize constraints in linear structural models. *Psychometrika.* 1984;49(1):37-47.

160. Macho S, Ledermann T. Estimating, testing, and comparing specific effects in structural equation models: the phantom model approach. *Psychol Methods.* 2011;16(1):34-43.

161. Engels RC, van der Vorst H, Deković M, Meeus W. Correspondence in collateral and self-reports on alcohol consumption: a within family analysis. *Addict Behav.* 2007;32(5):1016-1030.

162. Fisher SL, Bucholz KK, Reich W, Fox L, Kuperman S, Kramer J, et al. Teenagers are right—parents do not know much: an analysis of adolescent–parent agreement on reports of adolescent substance use, abuse, and dependence. *Alcohol Clin Exp Res.* 2006;30(10):1699-1710.

163. Latendresse SJ, Rose RJ, Viken RJ, Pulkkinen L, Kaprio J, Dick DM. Parental socialization and adolescents' alcohol use behaviors: predictive disparities in parents' versus adolescents' perceptions of the parenting environment. *J Clin Child Adolesc.* 2009;38(2):232-244.

164. Hussong AM, Hicks RE, Levy SA, Curran PJ. Specifying the relations between affect and heavy alcohol use among young adults. *J Abnorm Psychol.* 2001;110(3):449-461.

165. Mohr CD, Armeli S, Tennen H, Carney MA, Affleck G, Hromi A. Daily interpersonal experiences, context, and alcohol consumption: crying in your beer and toasting good times. *J Pers Soc Psychol.* 2001;80(3):489-500.

166. Griffin KW, Botvin GJ, Epstein JA, Doyle MM, Diaz T. Psychosocial and behavioral factors in early adolescence as predictors of heavy drinking among high school seniors. *J Stud Alcohol.* 2000;61(4):603-606.

167. van der Vorst H, Burk WJ, Engels RC. The role of parental alcohol-specific communication in early adolescents' alcohol use. *Drug Alcohol Depen.* 2010;111(3):183-190.

168. Simons LG, Conger RD. Linking mother–father differences in parenting to a typology of family parenting styles and adolescent outcomes. *J Fam Issues.* 2007;28(2):212-241.