Parent and peer influences reconsidered: the convoy of social support model of adolescent substance use

Michael John Cleveland
Iowa State University

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Parent and peer influences reconsidered: The convoy of social support model of adolescent substance use

by

Michael John Cleveland

A dissertation submitted to the graduate faculty
in partial fulfillment of the requirements for the degree of
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Program of Study Committee:
Jacques Lempers, Major Professor
Frederick Gibbons
Frederick Lorenz
Peter Martin
Ron Werner-Wilson

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For the Major Program
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CHAPTER 1. INTRODUCTION

Several decades of research have examined adolescent substance use from a wide range of theoretical and methodological perspectives. While much has been learned about the risk and protective factors that influence adolescent substance use behaviors (see Hawkins, Catalano, & Miller, 1992 for review), there still remain many issues that lack complete consensus. One of the most enduring debates among adolescent researchers involves the relative importance of parents and peers on adolescent risk behaviors—especially adolescent substance use. That is, it remains unsettled whether parents or peers are the primary determinants of adolescents' use of alcohol, tobacco, and other drugs.

The current study is an attempt to clarify the relative importance of parental and peer influence by using latent growth methodology (LGM). Longitudinal data will be used to test hypotheses concerning the changes in parental and peer influence over a 6-year period in a panel of rural adolescents (mean age of 14 years at first assessment). LGM allows for the estimation of individual differences in the developmental aspects of parental and peer influence. Therefore, it offers an innovate approach to this unsettled topic. Drawing on the assumptions of the social convoy model of social support (Antonucci, 1985; Kahn & Antonucci, 1980), a multivariate latent growth curve model is proposed in which the relative contributions of parental and peer influences on adolescent substance use are predicted by both the magnitude and change of social support (provision of aid, affection, and affirmation) the adolescent receives from each of these two primary interpersonal relationships. Furthermore, the multivariate model (see Figure 6) suggests that each source of influence has an independent effect on adolescent substance use.
2.1. Parent and Peer Influences

2.1.1. The Hydraulic Model Of Social Influence

The conventional wisdom regarding parental and peer influences on adolescent behavior is reflected in the belief that adolescence marks a developmental stage when a decline in parental influence is matched by an increase in the influence of friends or peers. This exchange occurs as adolescents begin to look beyond their family for sources of emotional support and normative values, turning instead to close-age friends and peers (Buhrmester, 1996; Furman & Buhrmester, 1985; 1992). An important assumption behind this belief is that the absolute level of social influence remains constant throughout adolescence—a proposition termed the hydraulic model to reflect the dynamic yet unvarying amount of influence available (Kandel & Lesser, 1972).

A perusal of theory and research on adolescent substance use confers considerable support for the hydraulic model of influence, as many authors have concluded that parental influence predominates at younger ages while peers are more influential for older adolescents (Berndt, 1979, Glynn, 1981; Krosnick & Judd, 1982). As an example, one recent study investigated the extent to which personality, family, and peer factors were related to young adult tobacco use in a sample of 976 children and their mothers (Brook, Whiteman, Czeisler, Shapiro, & Cohen, 1997). Brook et al. studied developmental changes in these predictors by dividing the sample into two groups: younger (less than 5 years old at time 1) and older (between 5 and 10 years old at first data collection). These two groups were followed over a period of seventeen years and three subsequent collections of data. Thus, the sample ranged from age 18 to 28 years at the final data collection. Parent influence was measured by child
and mother reports of several parenting measures (e.g., maternal affection, discipline, and conflict) as well as by the mother's report of smoking behavior. Child and mother reports were used to measure peer influence within several domains including deviancy, academic achievement, and use of tobacco and other substances. Regression analyses conducted separately for the two age cohorts revealed significant differences between the two groups in terms of childhood and adolescent predictors of adult tobacco use. Specifically, when controlling for adolescent personality measures, peer group risk factors were related to adult smoking only in the older age group, leading the authors to conclude that the magnitude of peer influence gains in importance between early and later adolescence (Brook et al., 1997).

2.1.2. Methodological Issues

While there seems to be general support for the hydraulic model, recent research has suggested that methodological weaknesses have resulted in inaccurate estimations of parental influence vis-à-vis friend influence (Aseltine, 1995; Bauman & Ennett, 1996; Kandel, 1996). Kandel (1996) proposes that this miscalculation results from two factors: overestimation of peer influences and failure to consider parental contributions to peer effects. Peer influence may be exaggerated in a number of situations. For instance, reliance on cross-sectional data may confound selection and socialization effects such that the researcher is unable to determine if similarity between friends is due to individuals of like-mind seeking each other out (birds of a feather flocking together) or friends influencing each other's behavior (as in peer pressure).

Overestimation of peer effects may also occur when adolescents' perceptions of peer behaviors or attitudes are used rather than self-reports. In this case, similarities in friends' behavior may actually reflect attribution and projection by the participant. Kandel (1996)
suggests that correlations based on targets’ perceptions of friends’ behaviors may be two to three times higher than those estimated from the friends’ self-reported data. The study by Brook et al. (1997) cited above underscores the ubiquity of this limitation—although some control for reporter bias was accomplished by using child and mother reports of parent and peer influences, no mention of the potential limitations of children’s reporting on peer behaviors is included in the authors’ discussion of the study’s limitations.

Noting the same methodological issues as Kandel (1996), Aseltine (1995) used a prospective design to examine the relative importance of parental and peer influences on adolescent delinquency and marijuana use. This study incorporated actual self-reports of friends’ behaviors in structural models that also tested for reciprocal associations between the target adolescent and the linked best friend. Although Aseltine (1995) concluded that parental influences were secondary to peer influences, he reported that the effects attributed to peer influence in this study were significantly lower than those typically reported in the literature. Furthermore, the estimates of parental influences were generally much more robust than what has generally been found in other studies. Aseltine (1995) found that both attachment to mom and parental monitoring predicted lower levels of adolescent delinquency while parental monitoring was also associated with decreases in adolescent marijuana use.

Furthermore, there is both cross-sectional (Wang, Fitzhugh, Westerfield, & Eddy, 1995) as well as longitudinal (Bauman, Carver, & Gleiter, 2001; Chassin, Presson, Sherman, Montello, & McGrew, 1986) evidence that the respective levels of parental and friend influence do not vary across adolescence. One of the first studies to reach this conclusion investigated age-related variation in the magnitude of parent and peer influences on adolescent cigarette use (Chassin et al., 1986). The authors of this longitudinal study
concluded that a variety of both parental and peer factors were significant predictors of transitions in smoking status among the adolescents (e.g., from never smoker to initial trier, from experimenter to regular user). Most importantly, Chassin and her colleagues pointed out that conclusions regarding age-related differences in the magnitude of parental and peer influences depended on whether cross-sectional or longitudinal analyses were employed. Cross-sectional analyses showed age-related increases in the magnitude of peer influence and a decrease in parental influence. However, longitudinal analyses revealed no age-related differences. The levels of parent and peer influences remained constant in strength across a sample ranging from 6th to 11th grades. Although this study is limited to only smoking behaviors, it represents a significant departure from the “virtual unanimity among theorists” who maintain that parent influences decrease, and peer influences increase, across adolescence (Krosnick & Judd, 1982).

Noting that the results of Chassin et al. (1986) may be limited to a local sample, Bauman et al. (2001) employed data drawn from a nationally representative sample of middle and high school students to examine progressions in adolescent smoking behaviors (nonsmoker, experimental smoker, occasional smoker, and frequent smoker). Contrary to their expectations, the authors (using both cross-sectional and longitudinal analyses) found no support for the hypothesis that parental influence (parent cigarette use) weakens and peer influence (peer cigarette use) increases as adolescents become older. However, these findings, like those of Chassin et al. (1986) are limited in that they may not generalize to adolescent risk behaviors other than cigarette smoking. Furthermore, parent and peer influences were represented in this study solely by parent and peer use of cigarettes. Finally, this study was also limited by the short, one-year time interval between the two waves.
Therefore, as the authors suggest, additional research is needed to assess the validity of the popular conceptualization of parental and peer influences on adolescent behavior.

Taken together, the above studies suggest that the conventional wisdom about parent and peer influences may not be entirely accurate. Although few in number, these well-designed, longitudinal studies provide support for the notion that parent and peer influences do not significantly vary across early to late adolescence. These findings, however, must be viewed with a certain amount of caution. As mentioned above, although these studies included samples from a wide age range of adolescents (6th-graders to 12th-graders), both were limited by the fact that the follow-up interviews were conducted after only one year. Thus, they offer a somewhat truncated picture of the developmental changes that take place over this time. A more complete picture of developmental changes in the magnitude of parent and peer influences on adolescent risk behaviors requires that cohort and historical time effects be controlled by following the same panel over a substantially longer time period. It is also important to note that the majority of studies that have examined variation in parent and peer influences have been limited primarily to only smoking behaviors. Very little research has been conducted to determine the relative importance of parent and peer influence for other adolescent risk behaviors or other types of substance use.

2.1.3. Findings from the Oregon Research Institute

The work of researchers at the Oregon Research Institute stands as an important exception to some of the limitations mentioned above. This research group has been at the vanguard of scientists taking advantage of recent developments in statistical techniques, such as latent growth models, which allow researchers to model developmental trends and examine intra- and inter-individual differences in the patterns and etiology of adolescent
substance use. Two studies within this group (Duncan, Duncan, & Hops, 1994; Duncan, Tildesley, Duncan, & Hops, 1995) are particularly pertinent to the discussion of age-related changes in parent and peer influences. Duncan et al. (1994) found that family influence (family cohesion) and peer influence (peer encouragement) were both significant predictors of adolescent alcohol use, though in opposite directions. That is, family influence was negatively related to adolescent alcohol use whereas peer influence was positively related to adolescent use. In addition, the largest effects of family influence were found in the oldest adolescent group, which was 15 years old at the first wave of data collected over a total of four years. Changes in peer influence, on the other hand, were most influential for the 12- and 13-year-old cohorts. In their discussion of these results, the authors note that although peer influences may play a role in adolescent alcohol use throughout adolescence, family influence may be curvilinear, bottoming out during early adolescence and subsequently increasing during the post pubertal periods.

Using the same sample, Duncan et al. (1995) expanded on their earlier work by examining latent growth models for alcohol, cigarette, and marijuana use. This study employed an associative growth curve model to determine if family cohesiveness and peer encouragement would have a common influence across the three different substances and across time. Support for a common influence across substances was found and together with strong correlations between initial levels and developmental trajectories of the individual substances, the results suggest that similar patterns of use exist among the three substances. As seen in the earlier study, the results also indicated that both family and peer influences had significant, though opposing, effects on initial levels of substance use. Furthermore, this study also replicated the finding that changes in peer encouragement were predictive of
initial levels and slopes of substance use over time.

This finding, however, highlights an important limitation of both of these studies—the use of change scores for both of the social influence factors introduces "concurrency into the design and potential confounds as to probable cause and effect" (Duncan et al., 1995, p. 1657). Noting this concern, the authors conclude that long-term longitudinal data are necessary to fully explore the reciprocal processes between the adolescent and his/her social environment. Although this study, along with others from the Oregon group and other researchers, has helped elucidate these complex relationships, it is difficult to not agree with their assessment that "the nature of the influence of family and peers on adolescent substance use, and particularly on the development of substance use, is far from clear" (Duncan et al., 1995, p. 1647-1648). On one hand, there is evidence that the magnitude of neither parental and peer influence varies across the adolescent period, while other research has found support that parent influence is curvilinear--bottoming out during the transition into adolescence when peer influence is at its apex.

2.2. Theoretical Framework

In order to help make sense of these contradictory findings some sort of schematic is necessary. One recent attempt to integrate diverse factors that contribute to adolescent substance use classifies theoretical causes of adolescent substance use along the two dimensions of type and level of influence (Petraitis, Flay, & Miller, 1995). According to this framework, three types of influence can be found among existing adolescent substance use theories (social or interpersonal, cultural or attitudinal, and intrapersonal), which operate at three distinct levels of influence (proximate, distal, and ultimate). Among these dimensions, the authors note that the preponderance of theories have focused on the social or
interpersonal influences, with less attention paid to cultural and intrapersonal types of influence. Furthermore, social influences have been studied at all three levels of influence: normative beliefs at the proximal level, attachment to role models at the distal level, and characteristics of such role models at the ultimate level of influence. Because it is also within this type of influence (social/interpersonal) that parent and peer influences reside, the remaining discussion will be limited to this area. As such, the following is not meant to be a comprehensive review of theories regarding the etiology of adolescent substance use, but rather a limited examination of the most widely used approaches to the complex pattern of relations between adolescents, their parents, and their peers.

2.2.1. Differential Association Theory

Most early investigations of social influence and substance use in the sociological literature approached the topic by subsuming substance use within general sociological theories of deviance and crime. Of these sociological perspectives, Sutherland’s *differential association theory* (1947; Sutherland & Cressey, 1980) was among the first to focus on family and peer influences of adolescent delinquency. One of the primary tenets of the differential association theory is that criminal or delinquent behavior is learned. Moreover, according to the differential association theory, delinquent behavior is learned through interactions with other people—especially through intimate social groups such as family, school, and peers. Thus, both prosocial and deviant behaviors are products of socialization—they differ only by the values espoused by the socialization agents. It is certainly possible that families (i.e., parents) can support antisocial behaviors—and that peers can promote prosocial acts. However, it is often assumed within this framework that parents and peer groups represent antagonistic forces, with peers luring the young person into a world of
delinquent behavior against a backdrop of conventional norms in the family.

Based on these propositions, differential association theory implies that peers are the primary sources of interpersonal influence on adolescent risk behavior. Furthermore, within this framework, parental influence on adolescent delinquency is limited to the indirect role that parents play in limiting their child's access to deviant peer groups (Glynn, 1981). Support for differential association theory is provided by studies that have found that peer socialization factors are the strongest predictors of adolescent substance use (e.g., Oetting & Beauvais, 1986, 1987; White, Johnson, & Horwitz, 1986). However, differential association theory suffers from some serious weaknesses. Primarily, differential association theory fails to explain why adolescents who associate with deviant persons do not become deviant themselves. Along this same line, differential association theory employs a certain circular reasoning in that it explains deviance solely through contact with deviant friends. This begs the question of how the "first deviant" came to be. Other researchers (e.g., Whitbeck, 1999) have noted that family process factors play a central role in determining associations with deviant peers, pointing out another weakness of differential association theory.

2.2.2. Social Control Theory

Contrasting the differential association theory is the social control theory of Travis Hirschi (1969; Gottfredson & Hirschi, 1990). The principal claim of this sociological perspective is that deviant behavior is the result of weak or insufficient bonds to conventional society. Thus, social control theory presumes that humans are by nature antisocial and delinquent. From this premise, control theorists ask a fundamentally different question than differential association theorists. Rather than asking what makes people commit delinquent acts, the important question is why most people do not commit crimes (Leighninger &
Popple, 1996). This perspective posits that young people form bonds with important societal institutions—most notably, family, school, and religion, which inhibit antisocial behavior by promoting conformity to group norms. From this proposition, control theorists maintain that adolescents without strong attachment to these and other conventional groups are likely to commit delinquent acts. Those with strong bonds to family, school, and/or religious institutions, on the other hand, are expected to be at less risk for delinquent behavior.

Unlike differential association theory, which places greater emphasis on peer influence, social control theory suggests that parents are the most important interpersonal influence on adolescent risk behavior. That is, according to this view, parents should have direct and independent effects on adolescent behavior, irrespective of the youth's involvement with delinquent friends (Glynn, 1981). Social control theory has also prompted a considerable number of research studies meant to explain adolescent substance use. Support for this view is provided by evidence that substance use is associated with weak bonds to family and school and strong attachment to deviant peers (Elliott, Huizinga, & Ageton, 1985; Jessor & Jessor, 1977). Other researchers have found that adolescent substance use is associated with disruptions in the family environment (Denton & Kampfe, 1994; Rhodes & Jason, 1990). However, social control theory seems to suffer from the opposite weaknesses of differential association theory mentioned above. That is, while differential association theory promotes the primacy of peer relationships, social control theory fails to account for the impact of deviant peers (Elliott et al., 1985). Critics have also pointed out that social control theory does not adequately identify the conditions that might lead to weak bonds or attachment to society (Elliott et al., 1985).
2.2.3. Social Learning Theory

In response to the weaknesses of the above two perspectives, much of the recent research conducted on adolescent risk behaviors has incorporated the basic tenets of both differential association theory and control theory with elements from psychological perspectives. Most notable among these approaches are social learning theory (Akers, 1998) and social cognitive learning theory (Bandura, 1986). Both of these theories share many assumptions with differential association theory above, such as the belief that adolescents acquire knowledge about deviant behaviors primarily through associations with other people. Thus, these theories underscore the importance of influential role models in adolescent behavior. However, the social learning theories extend differential association theory by also emphasizing the mediating role that cognitive beliefs play between role models and adolescent risk behavior (Petraitis et al., 1995).

For instance, one of the key concepts in Bandura’s (1986) social cognitive learning theory is self-efficacy, defined as beliefs a person holds that s/he can successfully perform a certain behavior. Such beliefs can play a critical role in both adolescent experimentation with, and abstention from, risk behaviors by providing the individual with both use and refusal skills, respectively (Bandura, 1982). There is substantial evidence that self-efficacy beliefs influence adolescent risk behaviors (Barkin, Smith, & DuRant, 2002; Carvajal, Evans, Nash, & Getz, 2002; Cohen & Fromme, 2002; Li, Pentz, & Chou, 2002; Wills, Gibbons, Gerrard, & Brody, 2000). Support for social learning theories is also provided by evidence that substance use by significant others predicts adolescents’ use of alcohol, tobacco, and other drugs. Many researchers have found that friends’ substance use is consistently found among the strongest predictors of adolescent substance use (Ary & Biglan, 1988; Aseltine,
1995; Blanton, Gibbons, Gerrard, Conger, & Smith, 1997; Garneir & Stein, 2002; Reifman, Barnes, Dintcheff, Farrell, & Uhteg, 1998). Notwithstanding this relationship, other authors have noted that parents' use of substances (especially alcohol and tobacco) is also associated with their children's drug use (Engels, Knibbe, de Vries, Drop, & van Breukulen, 1999; Wickrama, Conger, Wallace, & Elder, 1999).

These respective modeling influences are at the crux of the debate regarding the relative influence of parents and friends on adolescent substance use. That is, there is evidence that both sources of influence are significant predictors of adolescent substance use. Though many researchers have expanded on social learning theory—notably by also hypothesizing indirect effects of parent behaviors on adolescent substance use through control mechanisms (e.g., Ary, Duncan, Duncan, & Hops, 1999; Duncan et al, 1994; Dishion, Capaldi, Spracklen, & Li, 1995)—the bulk of recent empirical research concerning the relationships between families, peers, and adolescents has been guided by the tenets of this theory (Vega & Gil, 1998).

Social learning theory is, however, also not without its weaknesses. Most notably, this perspective assumes that an adolescent's behavior is determined in large part by his or her respective attachments to significant role models, but does not adequately explain how one role model may come to predominate over another. Thus, the theory does not account for individual differences regarding whom the adolescent uses as a role model—parents, peers, both, or neither. Furthermore, social learning theory fails to adequately explain the processes that are involved in the development of these important attachment relationships.

The theoretical approaches described above seem to offer an incomplete picture of the complex patterns of adolescent social relationships and their influence on adolescent risk
behaviors. While social learning theory comes the closest to bridging the gap between macro- and micro-level processes, it falls short of offering a comprehensive account of the interaction between the individual and his or her social environment in its failure to explain why some significant others are more “significant.” Furthermore, none of these theories makes explicit mention of developmental changes that may occur in the adolescent’s social relationships. It seems necessary, then, to move beyond the bounds of these traditional theories of the study of interpersonal factors and adolescent risk behavior. What is needed is a theoretical perspective that provides a more comprehensive picture of adolescents and their social relationships.

2.3. The Ecological Systems Theory

This type of holistic approach can be found in the ecological systems theory developed by Bronfenbrenner (1989). According to the ecological systems theory, a person resides within an environment of a series of nested systems that interact reciprocally and synergistically with the developing person. Specifically, Bronfenbrenner (1989) identifies a hierarchy of four interacting systems that range from the most proximal to the most remote. Beginning with the most proximal and extending outward, these four systems are labeled the micro-, meso-, exo-, and macrosystems.

The *microsystem* is described as the most proximal and immediate level of environment that involves the child as an active participant in activities, roles, and interpersonal relationships. Examples of microsystems may include the home, school, or peer group. Extending outward, the next level of the environment is the *mesosystem*, where two or more microsystems interact to create synergistic effects. Bronfenbrenner proposes that what happens in one microsystem influences and interacts with another microsystem; thus,
individuals may play different roles within the mesosystem—son or daughter in the family, friend in the peer group, boyfriend or girlfriend in a relationship, or student while in school. This is the level at which an adolescent may, for example, be faced with conflicting pressures from parent and peer influences.

The exosystem includes the larger community setting in which the individual lives. Compared to the micro- and macrosystems, the exosystem exerts a more distal influence, indirectly affecting processes within one or more of the more immediate settings. For example, demands at the workplace may require that a parent work overtime, thus affecting the ability of the parent to adequately monitor their children's after-school activities. This lack of supervision may, in turn, facilitate the child's experiences with alcohol or other drugs.

The most distal of the systems is the macrosystem, the overarching system of cultural values and belief systems that operates to offer differential resources, opportunities, and lifestyles to the individual. The macrosystem includes such distinctions as social class, ethnicity, and subculture and is reflected in cultural norms and social customs. Research at this level might include studies that examine how substance use patterns and risk factors may differ across ethnic and cultural groups (e.g., Wallace & Muroff, 2002).

Bronfenbrenner also emphasizes the importance of the chronosystem, which represents the influence of time on individual development in three respects. First, developmental time refers to ontogenetic passage of time as the individual matures and grows older. The second aspect of time is called cohort time, referring to shared experiences that result from entering into a certain experience with others. Finally, historical time includes events that occur in history that affect all individuals, regardless of age or birth cohort.
Within these five systems, Bronfenbrenner (1989) notes that four factors are interacting: person, process, context, and time. Person factors determine how the individual interacts with the four systems and may include personal characteristics or proclivities. Person factors are often represented by biological or psychological attributes such as age, gender, or personality type. Process factors represent the psychological or social experiences that "drive" development and comprise the core of the ecological systems approach. Examples of process factors may include parenting style and mother-child interactions. The third types of factors are called context factors, which include physical, socioemotional, and mental settings in which behavior occurs. Thus, the ecological model emphasizes the importance of the various circumstances and settings in which the child resides. Finally, time factors are also included in the ecological model, with consideration of all three effects of time—developmental, cohort, and historical.

The ecological perspective stresses the interaction among all four of these factors at each of the various settings described above—an ambitious task for any research project to undertake. However, Bronfenbrenner himself notes that the purpose of this theory is heuristic: "The aim was not to test hypotheses, but to generate them. Even more broadly, the goal was to develop a theoretical framework that could provide both structure and direction for the systematic study of organism-environment interaction in processes of human development" (1989, p. 230). Thus, operationalization of the ecological systems approach may be best accomplished by more precise research models that illuminate developmental processes (Bronfenbrenner, 1989).

2.4. The Social Convoy Model

Many authors (e.g., Levitt, 2000) have suggested that a full understanding of family
and peer relationships can be best accomplished by viewing such relationships as part of a broader social system, consistent with principles of the ecological systems theory. Such an approach is offered in the convoy of social support model (Antonucci, 1985; Kahn & Antonucci, 1980). The convoy of social support model (social convoy model) emphasizes the interpersonal interactions across various social systems (such as parents, siblings, and peers) that vary over the lifespan. Therefore, the social support convoy approach seems well-suited to test hypotheses concerning the two most proximal systems described above—the microsystem and the macrosystem—as well as those involving the influence of the chronosystem.

The primary tenet of the social convoy model is that throughout the life course, interpersonal relationships provide aid, affection, and affirmation (social support) that contribute to one’s well-being (Antonucci, 1985; Kahn & Antonucci, 1980). Thus, each individual has a personal network of family, friends, and others who exchange social support. The term convoy is used to imply that this social support network is dynamic—as each person moves through the life cycle, some members of the network will remain constant while others may be gained or lost. According to the social convoy model, as people’s needs and circumstances change, so does the amount and type of social support exchanged (Kahn & Antonucci, 1980).

In accord with ecological systems theory, Kahn and Antonucci (1980) describe a social support convoy as a social network that is determined jointly by the interaction of the characteristics of the individual and the properties of the environment. More specifically, this interaction between personal and situational factors influences both the structure and functions of the social support convoy (Antonucci, 1985). The convoy structure is described
as the network composition and refers to the size of the network as well as its stability and complexity. Conceptually and methodologically, convoy structure has most often been represented as a series of concentric circles, representing the degree of social support offered to the individual. The persons who provide the highest levels of support to the individual are located within the innermost circle and often include close family members. Those persons who provide less support to the individual are found in the outer circles of the convoy structure. Convoy functions include the actual support (i.e., aid, affection, and affirmation) that is exchanged by members of the convoy.

The social convoy model also suggests that the influence of time, particularly from a life course perspective, is important to the understanding of social support (Kahn & Antonucci, 1980). The authors point out that as people move through the life course, their circumstances may change, resulting in concomitant changes in their needs for, and ability to provide, support: “the form and amount of social support appropriate at a given time and place depend on...changing needs and circumstances: there is no single lifelong recipe” (Kahn & Antonucci, 1980). This emphasis on timing highlights the need to consider important transitions that occur throughout the life course such as starting school, developing at puberty, or becoming a parent.

2.4.1. Development and the Social Convoy Model

Drawing heavily on the theories of attachment, the social convoy model proposes clear theoretical hypotheses concerning the development of interpersonal relationships across the life course (Levitt, 1991). Antonucci (1985) describes this development as a dynamic process of evolving social networks. First, beginning with the attachment to primary caregivers in the early stages of the lifespan, she notes that individuals develop various
interpersonal relationships that will form the foundation of their support convoy. Therefore, it is expected that the nature of these relationships will also change as the child develops and matures. For example, as the child’s cognitive abilities develop, he or she will likely begin to take a more active role in eliciting support from caregivers. Thus, a child’s ability to use language, which can be used to ask for specific types of support, is just one example of a key developmental transition that alters the interaction between parent and child. Along with the changing nature of support relationships, it is important to keep in mind that the boundaries of the child’s social support network are shifting as well. Thus, Antonucci (1985) also points out that new members may be added to the convoy at the same time that other members may be lost. Normative life transitions, such as those discussed above, may present plentiful opportunities for movement into or out of the social support convoy.

Although most of the research on the social convoy model has been conducted with adult samples (with particular attention to transitions that occur during mid- and late-adulthood), there is reason to expect that it can be successfully applied to earlier stages of the lifespan. One of the first studies to expand the focus of the social convoy model to children and adolescents was conducted by Levitt, Guacci-Franco, and Levitt (1993). This cross-sectional study examined age variations in convoy structure and function, as well as age-related differences in the relations between convoy support measures and indicators of well-being (general self-concept and teacher ratings of sociability and mood) in a sample of 333 children between the ages of 7 and 14 years old. In general, this study provided evidence that social convoys develop across the transition from childhood to early adolescence, including an emergent role of peers as a source of support in adolescence. Significant age differences were found in both convoy structure and function such that by age 10, early adolescents
included more friends in their support convoy (although primarily in the outer circles of the convoy structure) and reported receiving more support from these friends compared to the younger children. Importantly, relatively few age-related changes were found in the adolescents' inner-circle composition and function. This continuity suggests that although friends played an increasing role in providing support during the transition into adolescence, close family members (i.e., parents) remained the primary sources of support.

2.4.2. Social Support and Adolescent Outcomes

Considerable research has shown that as children move into adolescence, they spend an increasing amount of time with friends and peers relative to the amount of time spent with parents and other family members (Hartup, 1983; Larson & Richards, 1991). Furthermore, other research has demonstrated that support from peers increases over adolescence, usually peaking in the mid-adolescent period (Cauce, Reid, Landesman, & Gonzales, 1990; Furman & Buhrmester, 1992; Helsen, Vollebergh, & Meeus, 2000; Scholte, van Lieshout, & van Aken, 2001). However, there is also evidence that parents remain influential throughout this transitional period and that many adolescents and their parents maintain a rather good relationship (Franco & Levitt, 1998; Munsch & Blyth, 1993; van Wel, Linssen, & Abma, 2000; van Wel, ter Bogt, & Raaijmakers, 2002). One study, however, found that although mothers and fathers were rated as important sources of instrumental support and affection among a sample of adolescents (age range: 11-19 years), the adolescents also perceived these parental relationships as highly conflictual (Lempers & Clark-Lempers, 1992).

Two studies by Furman and Buhrmester (1985, 1992) are noteworthy in their investigation of the relative influence of parental and peer support and also warrant particular attention. In the earlier study (Furman & Buhrmester, 1985), 5th- and 6th-grade adolescents
rated mothers and fathers as the most prominent providers of affection, enhancement of
worth, and instrumental aid compared to grandparents, siblings, and friends. The adolescents
also rated these parental relationships as more important and satisfying than the other types
of relationships.

A subsequent cross-sectional study explored developmental changes in supportive
relationships in a sample of adolescents from four stages of adolescence (Furman &
Buhrmester, 1992). Results of this study indicated that 4<sup>th</sup>-graders rated parents as the most
active providers of social support, 7<sup>th</sup>-graders rated both parents and same-sex friends as
important sources of support, 10<sup>th</sup>-graders rated same-sex friends slightly higher than parents,
and college students ranked mothers, same-sex friends, and romantic partners about equally
in providing support. Thus, these results suggest opposite patterns of change in the support
provided by parent and same-sex friends across pre-, early, mid-, and late adolescence:
parental support declined to its lowest point at the same time that support from same-sex
friends peaked.

A number of studies have also shown that social support, in general, influences an
array of positive and negative adolescent outcomes. For instance, general measures of social
support have been associated with psychological well-being (Scholte et al., 2001), self-
esteeem (DuBois et al., 2002; Franco & Levitt, 1998; Moran & DuBoies, 2002), and academic
achievement (Levitt, Guacci, Franco, & Levitt, 1994). Many studies have also shown that
family support has a negative influence on problem behaviors including delinquency and

There is also a substantial body of research that has examined the differential effects
of family and friend support. Barrera and Li (1996) presented results from 20 studies that
have incorporated both family and peer support as predictors of a variety of mental health outcomes, including both internalizing and externalizing symptoms. From this review, the authors conclude that of the two, support from family (most often parents) was the stronger predictor, particularly in relation to depression and externalizing symptoms (such as substance use). Furthermore, Barrera and Li (1996) noted that several studies showed that family and peer support systems have opposite effects on adolescent distress and problem behavior (e.g., Barrera & Garrison-Jones, 1992; Cauce, Felner, Primavera, & Ginter, 1982; Cauce, Hannan, & Sargeant, 1992; Chassin et al., 1986; Wills & Vaughan, 1989).

Wills, Mariani, and Filer (1996) presented an overview of their research program in the same handbook as Barrera and Li (1996) and reached much the same conclusions: (1) the effect of parental support on adolescent substance use is greater than that of peer support, (2) parental support has important buffering effects in that it interacts with negative life events and friends’ reported substance use, and (3) the support of peers is positively related to adolescent substance use. In discussing the implications of these findings, Wills et al. (1996) point out that the protective role of parents, though seemingly self-evident, runs counter to the widely-held conception that adolescent substance use occurs primarily because of peer pressure. In contrast to these “peer-centered” theories, Wills et al. (1996) offer several explanations why parental support is an important factor in determining adolescent problem behaviors. First, their research has shown that parental support has extensive effects on adolescents’ coping skills as well as on deviance-prone attributes, which may increase affiliations with substance-using peers. Second, parental support has been shown to contribute to the development of self-regulation ability—skills which play a determining role in adolescent adaptation and achievement vs. maladjustment.
Wills et al. (1996) note the consistency of these results with attachment theories that emphasize the development of working models of social relationships within the context of early-childhood interactions with parents (e.g., Sarason, Pierce, & Sarason, 1990; Sarason et al., 1991). This consistency suggests that the link between adolescents’ perceptions of supportive relationships and early attachment relationships may clarify how parental support contributes to positive adolescent outcomes. Specifically, this work indicates that the ability of adolescents to draw on the supportive functions of their social support network contributes to positive and healthy adolescent outcomes (Wills et al., 1996).

### 2.4.3. Advantages of the Social Convoy Model Approach

This conclusion that adolescents with positive perceptions of their social relationships are more likely to utilize these sources of support and therefore, are more likely to experience positive outcomes, underscores the advantages of using the social convoy model to explore the relative influence of parents and peers. The implication of this conclusion is clear: a full appreciation of the nature of adolescents’ relationships with parents and other providers of social support is essential to understanding the distinctive role that these two sources of support may play in influencing adolescent risk behaviors. The social convoy model offers a unique framework from which these social relationships can be studied.

In keeping with Bronfenbrenner’s emphasis on studying interacting systems, a primary advantage of the social convoy model is the fact that it explicitly proposes a reciprocal relation between the individual and his or her social environment. That is, the individual can be an active agent in determining the structure of his or her support network. Unlike the theories described earlier (differential association, social control, and social learning), which present the adolescent as a passive recipient of parent or peer influences, the
social convoy model introduces agency into the equation by acknowledging the reciprocal nature of these relationships.

Another advantage to studying the relative influence of parents and peers with the social convoy model is the fact that it offers testable hypotheses about why some relationships are more influential than others. As noted above, traditional explanations of deviance are limited in this respect. However, according to the social convoy model, the relative importance of one’s sources of social support is determined by their respective placement within the convoy structure. Although close family members such as parents are likely to be placed in the innermost circle, the social convoy model predicts that others may replace these persons, depending on one’s circumstances. These exchanges may be especially apparent during normative life transitions such as when a child enters early adolescence. This dynamic view of social influence reveals another advantage of the social convoy model: its ability to account for both stability and change throughout time. Most developmental researchers agree that this is a fundamental assumption of the lifespan development approach to human development.

2.5. Focus of Present Research

The current study is an attempt to integrate the findings from the social support literature, in general, and the social convoy model, in particular, with the disparate conclusions drawn from studies of adolescent risk behaviors. To the best of the author’s knowledge, the social convoy model has not been previously used to test hypotheses about adolescent substance use. The work is intended, therefore, as a first step toward gaining a more complete understanding of the interpersonal forces that are involved in this complex process.
Two distinct constructs will be employed to measure the relative importance of parents and peers within a social convoy model: (1) adolescents' perceptions of social support received from parents and friends, and (2) their assessments of the importance of parents' and friends' beliefs and attitudes in their substance use-related decisions. These two aspects are intended to capture the distinct processes involved in the relation between provision of support and adolescent risk behaviors. The first element, social support received from parents and friends, represents the placement of parents and friends in the adolescents' social convoy networks. That is, the relative placement of each in the adolescents' inner circle of support corresponds to the level of support the adolescents' report receiving from the respective source. The second construct, assessment of importance of parents' and friends' beliefs, represents the extent to which adolescents draw on the supportive functions of their social support network. This construct was described above as a hypothesized mediator between support functions and adolescent outcomes (Wills et al., 1996).

Because the social convoy model emphasizes the dynamic nature of social influences across the lifespan, this study utilizes latent growth methods to estimate growth parameters (initial levels and slopes) for parent and peer support as well as parental and peer influences on adolescents' decisions to use alcohol, tobacco, and marijuana. In addition, a multivariate latent growth model will be tested in this study. The multivariate model suggests that growth parameters of social support from parents and peers predict growth parameters of parental and peer influence, respectively, and that each source of influence has an independent effect on adolescent substance use. Presently, there is no theoretical or empirical consensus whether it is best to study separate models of alcohol, tobacco, and marijuana use or if these substances can be combined into an overall index of use. Therefore, this study will take a
conservative approach by estimating separate models to help determine if the patterns of alcohol, cigarette, and marijuana use are similar. These analyses will help determine if a combined substance use measure is warranted.

2.6. Hypotheses

2.6.1. Social Support

One of the key assumptions of the social convoy model is that changes in convoy structure may occur, especially during developmental transitions such as adolescence. Specifically, the social convoy model proposes that the boundaries of an individual’s inner circle of support are fluid and that persons in this inner circle may change, allowing for developmental changes in the relative importance of some network members. Most research has shown that close family members often occupy this innermost level of influence, but there is also evidence that beginning in early adolescence, friends are often also included as important sources of support (Levitt et al., 1993). Therefore, it is expected that while the level of parental support will decrease linearly across the adolescent period, support from friends will show a positive linear trend, increasing during this time.

2.6.2. Social influence

The increase in friend support during adolescence is consistent with other research, which has shown that adolescents’ perceptions of friendships also change during this time, from simple companionship to more intimate and self-disclosing relationships (Youniss & Smollar, 1985). There is also research that has shown that friends and parents may both be considered as important sources of social influence on adolescents during mid- to late adolescence (e.g., Furman & Buhrmester, 1985; 1992; Lempers & Clark-Lempers, 1992). This research has suggested that the level of parent influence (which often acts as a
protective buffer against risk behaviors) will exceed peer influence (which is often a risk factor for adolescent risk behaviors) in early adolescence but across the transitions into mid- and late adolescence, the levels of these two sources of influence will become increasingly similar.

Levitt et al. (1993) discuss the changing nature of friendships in the context of social support and propose that the increasing support from friends relative to parents during adolescence may “provide a bridge” toward the adolescents’ independence from the family. Reflecting this emerging autonomy, it is likely that adolescents will begin to turn toward others in their inner circle of support for guidance in their decision-making processes.

Therefore, it is expected that the level of parent influence on adolescents’ substance use decisions will be highest at early adolescence, decreasing through mid- to late adolescence. Conversely, it is expected that the level of friend influence on this decision-making process, lowest in early adolescence, will increase during the transitions across adolescence.

2.6.3. Relations Among Support, Influence, and Substance Use

The research cited above has also shown that parental support and peer support seem to act in opposite ways in predicting adolescent substance use—parental support is negatively related to use and friend support is positively related (Wills et al., 1996). A multivariate latent growth curve model will be used in this study to examine these relationships. Specifically, this multivariate latent growth curve model outlines a mediational process to describe the relationship between social support and adolescent substance use.

First, as outlined in the social convoy model, the level of social support provided by network members is reflected in their placement in the convoy model. As described above, the emergence of friends in the inner circle of the convoy may reflect an increasing tendency
of the adolescent to turn to friends, rather than parents, as sources of advice, particularly in such domains as substance use. *Therefore, it is expected that growth parameters of the two sources of support will predict growth parameters of each of the respective sources of influence.* That is, *high levels of, and changes in, parental and friend support will predict high levels of, and changes in, parental and peer influence, respectively.*

Furthermore, research has shown that parental support and friend support seem to have opposing effects on adolescent substance use, with support from parents decreasing the likelihood that adolescent will be involved with alcohol, tobacco, or other drugs while friend support is linked to increasing levels of use. The multivariate latent growth curve model used in the proposed study hypothesizes that this relationship between social support and adolescent substance use is mediated by the respective influence on adolescents’ decision-making these two sources of support provide.

Thus, the hypothesized model predicts that parental influence and peer influence will have independent and opposite effects on growth parameters of adolescent substance use. *That is, it is expected that high initial levels of parent influence will be related to lower initial levels of adolescent substance use. Similarly, increasing rates of change in parental influence will be associated with decreasing trajectories of adolescent substance use.* Opposite patterns will be expected for the relations between the growth parameters of peer influence and adolescent substance use. *Thus, high initial levels of friend influence are hypothesized to predict elevated initial levels of adolescent substance use, whereas increasing rates of change in friend influence will predict increasing trajectories of adolescent use.*
CHAPTER 3. RESEARCH DESIGN

3.1. Methods Used

3.1.1. Participants

The original sample in this longitudinal study was a panel of 500 adolescents (256 females), and their parents (490 mothers and 440 fathers) and siblings, from 50 rural counties in Iowa recruited for a longitudinal study of adolescent health risks (see Blanton et al., 1997; Gerrard, Gibbons, Benthin, & Hessling, 1996; and Gibbons, Gerrard, & McCoy, 1995 for descriptions of original analyses on this sample). At first assessment, half of the sample was 13 years old and half was 15 years old. There were virtually no minority students in the sample. Although the panel was followed over a period of eight years, a significant drop in the retention rate occurred beginning at the seventh wave. As shown in Table 1, year-to-year retention rates across the first six waves were all near or greater than 90%. However, less than 75% of the participants were retained at waves seven and eight. Therefore, the current study used data from the first six waves of the study, representing more than 75% of the original sample (N = 381). Attrition analyses, described in detail later, were conducted to determine if there were significant differences between participants in the final sample and those not included in the current study. Analyses were also conducted to determine if imputation of missing data was warranted in order to take advantage of the full sample.

Table 1
Attrition Rates Across The Eight Waves

<table>
<thead>
<tr>
<th></th>
<th>W1</th>
<th>W2</th>
<th>W3</th>
<th>W4</th>
<th>W5</th>
<th>W6</th>
<th>W7</th>
<th>W8</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average age (years)</td>
<td>14</td>
<td>15</td>
<td>16</td>
<td>17</td>
<td>18</td>
<td>19</td>
<td>20</td>
<td>21</td>
</tr>
<tr>
<td>N included in the sample</td>
<td>500</td>
<td>476</td>
<td>463</td>
<td>411</td>
<td>407</td>
<td>381</td>
<td>281</td>
<td>200</td>
</tr>
<tr>
<td>Percent of original sample</td>
<td>--</td>
<td>95.2</td>
<td>92.6</td>
<td>82.2</td>
<td>81.4</td>
<td>76.2</td>
<td>56.2</td>
<td>40.0</td>
</tr>
<tr>
<td>Retention rate of previous wave</td>
<td>--</td>
<td>95.2</td>
<td>97.3</td>
<td>88.8</td>
<td>99.0</td>
<td>93.6</td>
<td>73.8</td>
<td>71.2</td>
</tr>
</tbody>
</table>
3.1.2. Procedures

The families' participation was solicited through mailings to the parents of all 8th- and 10th-graders in randomly chosen rural public schools throughout the state of Iowa. Criteria for participation in the study included that the target adolescent have a sibling within two years of age, and willingness of the sibling and the custodial parent(s) to participate in the study. Of the families that met the criteria, 73% agreed to participate.

For the first three waves of data collection, all questionnaires were administered in the families' homes by a trained interviewer. After presenting instructions and obtaining informed consent, the interviewer asked the adolescents (and other family members) to complete the questionnaire in private. Anonymity was stressed and all family members were reminded several times that they were not to discuss each other's responses at any time. Beginning with the fourth wave, some adolescents had moved out of the home; therefore at subsequent waves, these adolescents were contacted by mail. Of those remaining in the sample, 249 (61%) lived at home at T5 and 92 (24%) at T6. Families were paid $50.00 for their participation at T1 and T2, and $55.00 at subsequent waves of data collection. Adolescents who had moved out of their parents' homes were paid $35.00 for participation. The interval between all waves was approximately one year.

3.1.3. Measures

Demographic variables. Two indicators of the parents' socioeconomic status were available from the mother and father interviews: total annual household income and highest level of education. Annual household income was assessed at the second wave of data collection using the question, “Taking into consideration all sources of income, what was the total income of your family household before taxes in [previous year]?.” Parents responded
individually to this item on an 18-point scale (anchors: 1 = $4,999 or less, 18 = $85,000 or more). A combined measure of household income was created by taking the mean of both parents' responses only if both parents answered the question. If one of the parents failed to respond to the question, the other parent's response was used as a proxy for household income. Combined and proxy measures of household income were then recoded into a 5-point scale: 1 = less than $20,000; 2 = $20,000 – $40,000; 3 = $40,000 – $60,000; 4 = $60,000 – $80,000; 5 = greater than $80,000. Table 2 provides summary details of all of the measures.

The parents' highest level of education was assessed at T1 by having the parents individually circle a number corresponding to the highest year of completed education. The values 1-12 indicated grade of school while values 13, 14, 15, 16, and 17+ were used to indicate post-secondary education. Separate indicators of mothers' and fathers' level of education were created by recoding each measure into a 4-point scale: 1 = only grade school; 2 = some high school; 3 = high school diploma; 4 = at least some college.

Parent substance use. Self-reported frequencies of alcohol use were also available from the parents at the first wave of data collection. The parents responded individually to the question, "How often do you currently drink alcohol?" on a 5-point scale: 1 = never, 2 = occasionally (e.g., social occasions), 3 = up to 1 drink per day, 4 = 1 or 2 drinks per day, 5 = frequently (more than 2 drinks per day).

Parental support. Adolescents' perceptions of parental support were assessed at T1 to T3, using the extended version of the Interpersonal Support Evaluation List (ISEL) (Cohen, Mermelstein, Kamarck, & Hoberman, 1985) as a base to assess emotional and instrumental support functions in the context of the adolescents' relationships with parents. At the first
wave, the items were preceded by an introduction worded as follows: "Here are some questions about who you talk to when you have problems or when you need advice. First, we'd like to know how you feel about talking to your mother or father, or the relative you talk to most." The introduction was modified at the second and third waves as follows:

"Think about the adult in your home who you feel closest to. We'd like to know how you feel about talking to this person." The introduction was followed by twelve items (T1 and T2) or six items (T3), assessed at T1 on a 4-point scale (1 = not at all true, 2 = a little true, 3 = somewhat true, 4 = very true) and a 5-point scale at T2 and T3 (anchors: 1 = not at all true, 5 = very true). Responses for the latter two waves were recoded to 4-point scales by collapsing the second and third-lowest categories. To maintain consistency across waves, a summary score of parental support using the mean of the six-item subscale available across all three waves will be used in these analyses. Reliability was assessed for the six-item summary scores at each wave (T1: a = .85, T2: a = .89, T3: a = .85). The following is a list of the six items used in the creation of the support subscale:

I feel that I can trust [my parent/that person] as someone to talk to.  
When I feel bad about something, [my parent/that person] will listen.  
If I talk to [my parent/that person] I think they try to understand how I feel.  
If I need to know something about the world (like how things work) I can ask [my parent/that person] about it.  
If I have a problem with my health I think I can talk to [my parents/that person] about it.  
If I'm having a problem with a friend, [my parent/that person] would have advice about what to do.

Friend support. Adolescents' perceptions of friend support were measured with items also derived from the ISEL, parallel to those for parental support. The following statement introduced these items: "Now we'd like to know how you feel about talking to a friend, when you have a problem or need advice. Answer for the friend (or friends) you talk to most." The same statements used for parent support (12 items at T1 and T2, 6 items at T3) followed this introduction, with "friend(s)" replacing "parent," using the same 4-point or 5-point scales.
Responses for the two latter waves were recoded to 4-point scales by collapsing the second and third-lowest categories. Again, a summary score of friend support using the mean of the six-item subscale was used. Reliability coefficients were calculated for the six-item summary scores at each wave (T1: α = .84, T2: α = .86, T3: α = .89).

**Parent and friend influence—alcohol use.** Single items at each wave were used to measure adolescents' assessments of the likelihood that both parents' and friends' attitudes and beliefs would influence their own drinking behavior. Following the prompt, “Describe the following factors in terms of how much they are likely to influence your drinking behavior,” the adolescents were asked to use a 7-point scale (1 = “not at all” to 7 = “very much”) to indicate agreement with “Your [friends'/parents’] attitudes and beliefs about drinking.”

For parental influence to use alcohol, all 15 possible correlations (6 + 5 / 2) across the six waves of data were significant at the .01 level. These correlations ranged from .21 (T1 – T5) to .56 (T2 – T3). The average interitem correlation across the six waves was .40. Similarly, 13 of the 15 possible correlations between the 6 friend influence on alcohol use items were significant at the .05 level, ranging from .11 (T2 – T5) to .50 (T4 – T5). The correlation between the T1 and T4 friend influence to use alcohol items was marginally significant (r = .09, p = .07) and the correlation between T1 and T5 items was not significant (r = .08, p = .11). The average interitem correlation for the friend influence to use alcohol items was .27.

**Parent and friend influence—tobacco use.** Single questions at the six waves were also used to measure adolescents' assessments of the likelihood that parents' and friends' attitudes and beliefs would influence their smoking behavior. For these items, the prompt “Please
indicate how much each of the following factors would influence whether or not you will smoke” was followed by the statements “Your [friends’/parents’] attitudes and beliefs about smoking.” Responses to both questions were made on 7-point scales from 1 = “not at all” to 7 = “very much.” All of the interitem correlations among the parental influence to use tobacco were significant at the .01 level and ranged between .21 (T1 – T5) to .54 (T4 – T5). The average interitem correlation for the parental influence to use tobacco items was .38. For friend influence to use tobacco, 14 of the 15 possible correlations were significant at the .05 level, ranging from .11 (T1 – T6) to .41 (T5 – T6). The correlation between the T3 and T4 measures of friend influence on tobacco use was not significant (r = .06, p = .26). The average interitem correlation for the friend influence to use tobacco items was .25.

Adolescent drinking. Single items were used at each wave to assess the frequency of drinking: “How many times in the last X months have you had a whole drink of alcohol (for example, a bottle of beer, a glass of wine, or a whole mixed drink?),” followed by a 5-point scale (1 = “never,” 2 = “once or twice,” 3 = “a few times,” 4 = “more than a few times (up to once a week),” 5 = “regularly (at least two or three times a week).” The time frame changed from 3 months at T1-T4 to 12 months at T5-T6. All across-time intercorrelations among the alcohol use measures were significant at the .01 level, ranging from .20 (T1 – T6) to .74 (T4 – T5). The average interitem correlation for the alcohol use items was .46.

Adolescent smoking. Single items were also used at each wave to assess the frequency of smoking: “What is the most that you have ever smoked cigarettes?” followed by a 6-point scale (1 = “never,” 2 = “tried once or twice,” 3 = “tried a few times,” 4 = “I have smoked a few times every month),” 5 = “I have smoked several times a week,” 6 = “I have smoked every day”). The smoking items were recoded into 5-point scales at all waves by collapsing
the fourth and fifth categories ("a few times a month" and "several times a week") into one. All across-time intercorrelations among the cigarette use measures were significant at the .01 level, ranging from .37 (T1 – T5) to .86 (T5 – T6). The average interitem correlation for the cigarette use items was .63.

Adolescent marijuana use. Marijuana use was assessed at five of the six waves with the following items: "How many times have you used marijuana?" (T1-T3) and "In the past 12 months, how many times have you used marijuana?" (T5-T6), followed by 5-point scales (1 = “never,” 2 = “once or twice,” 3 = “a few times,” 4 = “more than a few times,” 5 = “regularly (once a week or more).”) Marijuana use was not assessed at the fourth wave of data collection. Seven of the possible ten across-time intercorrelations (5 + 4/2) among the marijuana use measures were significant at the .05 level, ranging from .12 (T2 – T5) to .60 (T5 – T6). Correlations between T1 and T3, T4, and T5 marijuana use were not significant. The average interitem correlation for the marijuana use items was .24.

Table 2
Description of Study Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Scale</th>
<th>Source of Information</th>
<th>Waves Measured</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parents’ combined income</td>
<td>1 to 5</td>
<td>Mother &amp;/or Father</td>
<td>2</td>
</tr>
<tr>
<td>Mother’s education</td>
<td>1 to 4</td>
<td>Mother</td>
<td>1</td>
</tr>
<tr>
<td>Father’s education</td>
<td>1 to 4</td>
<td>Father</td>
<td>1</td>
</tr>
<tr>
<td>Mother’s alcohol use</td>
<td>1 to 5</td>
<td>Mother</td>
<td>1</td>
</tr>
<tr>
<td>Father’s alcohol use</td>
<td>1 to 5</td>
<td>Father</td>
<td>1</td>
</tr>
<tr>
<td>Parent Support</td>
<td>1 to 4</td>
<td>Target</td>
<td>1 to 3</td>
</tr>
<tr>
<td>Friend Support</td>
<td>1 to 4</td>
<td>Target</td>
<td>1 to 3</td>
</tr>
<tr>
<td>Parent Influence-Alcohol Use</td>
<td>1 to 7</td>
<td>Target</td>
<td>1 to 6</td>
</tr>
<tr>
<td>Friend Influence-Alcohol Use</td>
<td>1 to 7</td>
<td>Target</td>
<td>1 to 6</td>
</tr>
<tr>
<td>Parent Influence-Cigarette Use</td>
<td>1 to 7</td>
<td>Target</td>
<td>1 to 6</td>
</tr>
<tr>
<td>Friend Influence-Cigarette Use</td>
<td>1 to 7</td>
<td>Target</td>
<td>1 to 6</td>
</tr>
<tr>
<td>Target Alcohol Use</td>
<td>1 to 5</td>
<td>Target</td>
<td>1 to 6</td>
</tr>
<tr>
<td>Target Cigarette Use</td>
<td>1 to 5</td>
<td>Target</td>
<td>1 to 6</td>
</tr>
<tr>
<td>Target Marijuana Use</td>
<td>1 to 5</td>
<td>Target</td>
<td>1 to 3, 5 to 6</td>
</tr>
</tbody>
</table>
CHAPTER 4. DATA ANALYSIS

4.1. Studying Change Over Time

The primary research question addressed in this study concerns the developmental changes associated with the relative importance of parents and friends on adolescents' substance use behaviors. In order to demonstrate change over time, a longitudinal research design is required (Duncan, Duncan, Strycker, Li, & Alpert, 1999). The traditional approach to studying change over time has been the autoregressive (or residual change) model (Figure 1). This model measures the autoregressive effect (the effect of the measure at Time 1 on the measure of the same variable at Time 2). However, Duncan et al. (1999) summarize the limitations of this traditional approach by listing several shortcomings inherent in these models.

Figure 1. Representation of the autoregressive model.
First, typical autoregressive models are inadequate, because they are concerned solely with variances and covariances of the independent variables. However, sample means are often also of considerable interest to researchers. The second limitation of autoregressive models is that they are not able to generalize beyond more than two points in time, even in multiwave panel studies. In such studies, it is necessary to focus only on change scores between any two points in time (i.e., residualized change). The third limitation of autoregressive models concerns the fact that by controlling for initial levels of the behavior, these models are limited only to predictors that predict change in the rank order of the observations over time. However, Duncan et al. (1999) point out that in certain phenomena (e.g., those that are monotonically stable), the rank order remains the same, even though significant changes at the individual or group level may occur. Furthermore, residualized change score models are limited by the fact that the amount of change may often be a function of the individuals’ initial status (Collins, 1996).

These limitations of autoregressive models are reiterated by several other authors who advance the notion that the analysis of change (i.e., development over time) cannot be conducted using the same methods that are used in other types of research (e.g., Collins, 1996; Rogosa, Brandt, & Zimowski, 1982). Specifically, developmental models are needed that are capable of describing an individual’s developmental trajectory as well as capturing individual differences in these trajectories over time (Duncan et al., 1999). Furthermore, developmental models need to be able to accommodate predictors of individual differences and, at the same time, capture the developmental changes of the group. Finally, more advanced techniques are required in which the relationship between initial status and rate of change can be estimated (Collins, 1996). Latent growth curve methodology satisfies all of
these criteria and therefore represents a valuable tool in modeling development by identifying predictors and correlates of change.

4.2. The Basic Latent Growth Curve

The basic latent growth curve model, depicted in Figure 2, comprises two latent factors, with the repeated measures of the construct over time as the indicators. Conceptually, this model can be viewed as a confirmatory factor-analytic model, with the latent factors representing chronometric common factors of individual differences over time (McArdle, 1988). The first latent factor defines the intercept of the growth curve in which the factor loadings of the repeated measures are set to 1.0, which represents the starting point of the growth curve at Time 1. The second latent factor defines the slope of the growth curve and represents the rate of change of the trajectory over time. The means of these latent intercept and slope factors represent the group growth parameters and are overall measures of the intercept and slope for all participants. The variances of the latent factor reflect the variation among individuals around the overall group growth parameters. The estimation of variability in individual changes over time makes this a random coefficients model.

Growth curve methodology can be thought of as consisting of two stages. In the first stage, a regression curve, not necessarily linear, is fit to the repeated measures of each individual in the sample. In the second stage, the parameters for an individual’s curve become the focus of the analysis rather than the original measures (Duncan et al., 1998). Latent growth curve models define change over time in terms of unobserved latent factors and thus fit into the general structural equation modeling framework (Stoolmiller, Duncan, Bank, & Patterson, 1993; Willett & Sayer, 1994).
Figure 2. Representation of the latent growth curve model.
CHAPTER 5. RESULTS

This section will present the findings of the research. Summary statistics for all variables will be presented first, followed by the results of repeated measures multivariate analyses of variance (MANOVA) tests employed to detect group differences among dependent variables. Next, a series of MANOVA analyses will be used to track the trajectories of participants with varying levels of social support (from parents and friends) at Time 1 across time for the other variables. Several analyses will then be conducted to determine if a combined measure of substance use is warranted. These analyses will include pairwise intercorrelations among the use measures as well as the parent and friend influence items, separate univariate growth curve models for each of the three substances, an associative model, and a comparison of the factor-of-curves and curve-of-factors models.

Next, the results of the attrition analysis and model-based missing data analyses will be presented. These results were conducted to determine if imputation of missing data was warranted. The results of data imputation using the EM algorithm available in SPSS MVA are presented next. The resulting EM imputed covariance matrix was then used to test the hypotheses of the study. Specifically, univariate growth curves were used to test hypotheses concerning the developmental changes in parental support and influence as well as friend support influence. A series of nested models were then tested in a hierarchical manner to determine the relations among the growth parameters of support, influence, and adolescent substance use. Finally, multi-sample analyses were used to test for group differences between males and females and between the older and younger age groups.
5.1. Data Summarization

Table 3 provides label names and descriptive statistics of all variables in the study. Measures of mothers’ and fathers’ alcohol use are presented first, followed by three indicators of SES: parents’ combined income, and mothers’ and fathers’ education level. The repeated measures of support (times 1 -3), influence (times 1-6), and use (times 1 -6) are listed next. Mean levels of parent and friend support did not differ at Time 1; however, paired samples t-tests indicated that friend support exceeded parent support at both Times 2 ($t = -4.350, p < .001$) and Time 3 ($t = -2.338, p < .05$). The reported influence of both parents and friends on adolescents’ decisions to use alcohol and cigarettes were highest at Time 1 and lowest at Time 6. Paired samples t-tests indicated that for both types of substances and at all waves, the level of parent influence was greater than that of friend influence (all $p$s < .001). The reported use of all three substances was lowest at the first wave and highest at Time 6.

Values of univariate skewness and kurtosis are also included in Table 3. To satisfy the assumption of univariate normality, Kline (1998) suggests that absolute values greater than 3.0 and 10.0 are indicative of extreme skewness and kurtosis, respectively. Using these criteria, the only items that seem to offer extreme violations of the normality assumptions are the first three measures of marijuana use. Because estimation techniques employed by latent growth curve modeling, such as maximum likelihood, assume multivariate normality, the data were also examined for normality using PRELIS 2.3 (Joreskog & Sorbom, 1999). PRELIS provides a measure of multivariate normality, commonly referred to as Mardia’s statistic of kurtosis (Mardia’s PK; Mardia, 1970), which equaled 1.14. Although there is no standard cutoff for this index, Bentler (1998) recommends that multivariate normality can be assumed if this value is less than 3.00.
Table 3
Descriptive Statistics for all Study Variables

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<th>Variable</th>
<th>Label</th>
<th>Min.</th>
<th>Max.</th>
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<th>SD</th>
<th>Skewness</th>
<th>Kurtosis</th>
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<td>Meduc</td>
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<td>2.11</td>
<td>-.05</td>
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<td>3.69</td>
<td>2.10</td>
<td>.05</td>
<td>-1.36</td>
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</tr>
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<td>2.55</td>
<td>1.80</td>
<td>.83</td>
<td>-1.55</td>
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</table>
As seen in Table 4, there were increases in the percentage of regular users of both alcohol and cigarettes across the six waves of data collection. By Time 6, most of the respondents reported at least trying alcohol (89.9%) or cigarettes (71.2%). The respondents reported very little marijuana use at the first three waves, with slight increases in the percentage of regular users at Time 5 (2.0%) and Time 6 (2.4%). More than a quarter of the respondents (26.1%) reported at least experimental use of marijuana at Time 6, though most of these reported using marijuana only once or twice (9.8%) or a few times (8.2%).

These substance use rates are comparable to those found in national norm studies such as the Monitoring the Future (MTF) survey (Johnston, O'Malley, & Bachman, 2003). For instance, at Time 1 (1991) when the average age was 14 years old, 28.1% of the
adolescents in this study reported at least some use of alcohol during the previous 3-month period. This rate is similar to the 30-day prevalence rate for 8th-graders (in 1991) reported in the MTF study, which was 25.1%. The 12-month prevalence rate of Time 5 alcohol use in this sample was 82.8%, slightly higher than the rate for 12th-graders in the MTF study in 1995 (73.7%). For cigarette use, the lifetime prevalence rates for this sample was 31.5% at Time 1 and 64.6% at Time 5. Again, these rates are comparable to the MTF study, which showed lifetime prevalence rates of 44.0% for 8th-graders in 1991 and 64.2% for 12th-graders in 1995. Marijuana use in this sample was lower than rates in the MTF study. While only 1.2% reported any use of marijuana at Time 1, and 17.9% at Time 5, the MTF study reports prevalence rates of 6.2% for 8th-graders in 1991 and 34.7% for 12th-graders in 1995.

Table 4  
*Percent of Respondents Reporting Different Levels of Substance Use*

<table>
<thead>
<tr>
<th>DV</th>
<th>Never</th>
<th>Once/twice</th>
<th>Few times</th>
<th>&gt; Few times</th>
<th>Regularly</th>
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<tbody>
<tr>
<td><strong>Target Alcohol Use</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time 1</td>
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<td>13.3</td>
<td>10.2</td>
<td>3.2</td>
<td>1.4</td>
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<tr>
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<td>16.8</td>
<td>8.4</td>
<td>1.5</td>
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<td>20.3</td>
<td>13.4</td>
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<td>13.7</td>
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5.2. Group Differences

5.2.1. Time, Gender, and Age Differences

Descriptive statistics were also calculated separately for the two age groups and for male and female participants. Repeated measures three-way multivariate analyses of variance (MANOVA) were used to test for mean differences across time for the two groups—gender and age—as well as for the interaction of gender and age. MANOVA is a technique used for assessing group differences across multiple dependent variables simultaneously. MANOVA is preferred over multiple one-way ANOVAs, because it controls for the experiment-wide error rate. That is, the use of multiple one-way ANOVAs raises the probability of a Type I error, resulting in the possibility of erroneous effects. Additionally, MANOVA is the preferred method when significant multicollinearity exists among dependent variables. In this case, MANOVA may detect combined differences not found in separate univariate tests and thus provide more statistical power.

Results of the repeated measures three-way MANOVAs are reported in Table 5. Significant gender differences were found for both parental support \( (F[1, 425] = 9.45, p = .002) \) and friend support \( (F[1, 426] = 107.31, p < .001) \). The main effect of age was also significant for several of the outcome variables: friend support \( (F[1, 426] = 10.04, p = .002) \), friends' influence on cigarette use \( (F[1, 313] = 5.69, p = .018) \), and reported use of alcohol \( (F[1, 310] = 24.59, p < .001) \) and cigarettes \( (F[1, 303] = 6.14, p = .014) \). Significant main effects for time were found for all of the outcome variables except friend support, which was marginally significant: \( (F[2, 852] = 2.54, p = .053) \).
Table 5
Time x Gender x Age Repeated Measures MANOVA Results

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<th>Interaction Effects</th>
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Note. Values represent F-test values. Inf = Influence; G x A = Gender by Age interaction effect; G x T = Gender by Time interaction effect; A x T = Age by Time interaction effect; G x A x T = Gender by Age by Time interaction effect. *p < .10; †p < .05; **p < .01; ***p < .001.

Significant interaction effects were also found for several of the outcome variables, indicating that mean values of these measures differed across time for the groups (male/female or younger/older age). In higher-order designs such as this, interaction effects are best examined in descending order, beginning with the highest-order interaction (Pedhazur & Schmelkin, 1991). The first step in this analytic approach examines the second-order interaction (i.e., gender x age x time). If this interaction is significant, Pedhazur and Schmelkin recommend that simple interaction effects next be considered. In other words, interactions between two of the factors are examined within each level of the third factor. As seen in the table, four second-order interactions were significant (for parent influence to use alcohol, friend influence to use cigarettes, target alcohol use, and target cigarette use).
Therefore, Table 6 presents means and standard deviations for male and female participants in each of the two age groups.

Examination of the mean levels of parental influence to use alcohol reveals that although all four groups reported similar levels at T1, younger males reported an increase in parental influence between T1 and T3, after which this influence declined. In contrast, the other three groups reported a less steep decline across the six waves. A somewhat similar pattern was found in the targets’ reported influence from friends to use cigarettes. In this case, younger males reported an increase across the first four waves and then a sharp decrease in friend influence. The other groups reported a more general decline in friend influence over time.

The second-order interactions for alcohol and cigarette use were also significant. Examination of mean levels of alcohol use among the four groups indicates that although all of the groups reported an increase in use across time, the reported use of alcohol at T1-T5 was greater among the older participants. However, by Time 6, the reported use of alcohol was greater for males than females, regardless of age. For cigarette use, the males and females in the older age group reported nearly identical trends across the six waves with both groups increasing across time at the same rate. Among the younger adolescents, however, males and females did not report similar trends, resulting in a disordinal interaction. That is, the reported use of cigarettes was higher for younger males than younger females at the first two waves, but beginning at T3, the females reported higher levels of cigarette use. In fact, females in the younger age group reported the highest level of cigarette use at T6 among all four groups.
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Table 6
Means And Standard Deviations, By Age And Gender
5.2.2. Time 1 Social Support Differences

Repeated measures MANOVAs were also used to test for mean differences on the variables across time for Time 1 levels of parent and friend support. To accomplish this, standardized scores for parent and friend support were calculated. Cutoff values of +/- 1 SD were then used to create three groups based on level of Time 1 support: low, medium, and high. Tables 7 and 8 display the means and SDs of all variables for the three parent and friend support groups, as well as results of the repeated measures MANOVAs. The three parental support groups differed in the mean level of friend support \((F [2, 410] = 14.21, p < .001)\) and of parental influence to use alcohol \((F [2, 295] = 27.52, p < .001)\) and cigarettes \((F [2, 299] = 17.00, p < .001)\). The interaction of level of Time 1 parental support and time was significant only for the repeated measures of parent support \((F [4, 852] = 25.54, p < .001)\).

Examination of these means reveals that the two extreme groups at Time 1 appeared to regress to the mean, though highest levels of parent support at Time 3 were reported by the high Time 1 group and, similarly, lowest levels were found in the low Time 1 group.

Only two outcome variables showed significant means differences among the three groups of friend support: parent support \((F [2, 418] = 23.84, p < .001)\) and friend support itself \((F [2, 427 = 188.76, p < .001]; \text{see Table 8})\). Again, the significant interaction of level of Time 1 friend support and time on repeated measures of friend support \((F [4, 854] = 23.40, p < .001)\) revealed a regression to the mean pattern. A significant level of time 1 friend support by time interaction was also found for the repeated measures of cigarette use. Interpretation of this interaction effect was less clear than the regression to the mean patterns above, but it appears that although the three groups started out at about the same level of cigarette use, the rate of increase was higher for both of the extreme groups.
Table 7
Means And Standard Deviations Of All Variables For The Three Parent Support Groups

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Note. PS x T = Parent Support by Time interaction effect; F-values in italics indicate p < .05.
Table 8
Means And Standard Deviations Of All Variables For The Three Friend Support Groups

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Note. FS x T = Friend Support by Time interaction effect. F-values in italics indicate p < .05.
5.3. Combined Substance Use

There is evidence that adolescents' use of alcohol, cigarettes, and other drugs are intercorrelated (Duncan, Duncan, Biglan, & Ary, 1998) and that developmental trajectories of their use are also positively related (e.g., Duncan & Duncan, 1996; Duncan et al., 1998; Duncan et al., 1995). These authors suggest, therefore, that growth parameters for alcohol, cigarette, and marijuana use can be adequately modeled by a higher order substance use construct. However, methods to examine multivariate relations among developmental functions such as these are not well established (Duncan et al., 1998). To determine if such a combined measure of substance use was warranted in this study, a series of analyses were conducted. The first step in this series was to determine if alcohol use, cigarette use, and marijuana use were significantly correlated within each wave. These analyses included the correlations among alcohol, cigarette, and marijuana use, as well as between the parental and friend influence to use alcohol and cigarettes items. Next, average interitem correlations across the six waves of data collection were calculated to determine if different combinations of substances (e.g., alcohol and cigarettes compared to alcohol and marijuana) exhibited similar patterns of correlations. Similar interitem correlations within and across time would suggest that the different combinations could be combined into an overall measure of substance use at each wave.

However, the intercorrelations can only provide evidence that the substances are related at a certain point in time. Further evidence is needed to determine if the different substances have similar developmental trajectories. Therefore, results of separate univariate growth curve models for each of the three substances will be presented next. These analyses will allow us to determine if the latent growth curve of the three substances "tell the same
story." That is, a variety of comparisons can be made to determine if the substances follow the same pattern of growth over the six waves. First, overall goodness of fit indices will be compared to determine how well the specified model fits the observed data for each of the three substances. Departures from linear growth can then be determined by examining the slope factor loadings for each of the models. Finally, the means and variances associated with the latent growth factors (intercept and slope) will also be compared. Similar mean levels for the intercept factor will indicate that the total sample reported similar initial levels of use across the substances. Similar mean levels of the slope factor will suggest that the rate of change across substances is similar for the total sample.

Although the separate univariate growth curves will allow for comparison of growth across the different substances, a more precise metric of their covariance can be determined in a multivariate analysis (Duncan et al., 1998). This approach is based on McArdle (1988) and uses latent growth curve modeling to test both first-order and second-order multivariate growth models. The first-order model is termed an associative LGM and allows for the comparison of correlations among pairs of developmental parameters. The second-order models include two alternative methods to examine correlations among growth parameters and are termed the factor-of-curves model and the curve-of-factors model. Fitting the associative model allows for the estimation of means, variances, and covariances for the growth curve parameters (intercepts and slopes) for all three substances at once. In essence, the associative model combines the separate univariate growth curves into one larger model. Relationships among the intercepts and slopes for each of the three substances can be compared in the associative model to indicate whether or not initial levels and change over time in the use of alcohol, cigarettes, and marijuana are related.
The factor-of-curves approach (Figure 3) can be used to examine whether a higher-order factor adequately described relations among lower-order developmental functions, such as intercept and slope. In this model, growth curves were applied to each substance separately to obtain individual intercepts and slopes in the first-order LGM. The second-order common factors (intercept and slope) were then used to describe individual differences among the first-order growth parameters. To specify this model, the covariances among the first-order latent growth curves were fixed at zero. Factor loadings for the higher-order structure were restricted to be equal over time for each substance, using the parameters of alcohol use as reference scaling points, to impose a form of factorial invariance necessary to ensure the same units of scaling for the second-order factor scores (Duncan, et al., 1999). Thus, Figure 3 shows that the factor loadings between the first- and second-order factors are restricted to be equal over time for cigarette ($L_a$) and for marijuana use ($L_b$).

The final method used to analyze the multivariate relationships among alcohol, cigarette, and marijuana use is the curve-of-factors model, depicted in Figure 4. This method involved fitting a higher-order growth curve to factor scores representing common factor scores for the three substances at each time period. Thus, the observed variables, measured at each time point, were factor analyzed to produce a higher-order common intercept and common slope (Duncan et al., 1998). Error terms for each variable across time were allowed to covary to improve the goodness of fit of the model (Duncan et al., 1998). In order to ensure mathematical identification of the model, the curve-of-factors LGM required that loadings for alcohol ($L_a$) and marijuana use ($L_b$) were equal over time (see McArdle, 1988; Meredith & Tisak, 1982; Nesselroade, 1983).
Figure 3. Representation of the hierarchical factor-of-curves LGM.
Figure 4. Representation of the hierarchical curve-of-factors LGM.
5.3.1. Correlations

The interitem correlations among alcohol, cigarette, and marijuana use, and parent and friend influence on alcohol and cigarette use are reported in Table 9. As seen in the table, the interitem correlations between alcohol and cigarette use were modest and remained quite stable across the six waves of data collection, with an average correlation of 0.54. In contrast, intercorrelations between marijuana and alcohol and cigarette use were weak, averaging only .27 across six waves. The correlations between cigarette and marijuana use showed the most variation across the six waves, ranging from .26 (Time 2) to .44 (Time 5). The average interitem correlation between cigarette and marijuana use was .34. Interitem correlations between the targets' report of interpersonal influence to use alcohol and cigarettes were modest for both parent and friend influence. The average interitem correlation across waves was 0.50 for parent influence and 0.44 for friend influence. Measures of parental and friend influences on marijuana use were not available at any wave and could not be compared to the measures of interpersonal influence to use the other two substances.

Table 9

<table>
<thead>
<tr>
<th>Substance Use</th>
<th>Influence to Use Alcohol</th>
<th>Influence to Use Cigarettes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Alcohol-Cigarette</td>
<td>Alcohol-Marijuana</td>
</tr>
<tr>
<td>Wave 1</td>
<td>.56***</td>
<td>.18***</td>
</tr>
<tr>
<td>Wave 2</td>
<td>.56***</td>
<td>.27***</td>
</tr>
<tr>
<td>Wave 3</td>
<td>.60***</td>
<td>.29***</td>
</tr>
<tr>
<td>Wave 4</td>
<td>.57***</td>
<td>n/a</td>
</tr>
<tr>
<td>Wave 5</td>
<td>.53***</td>
<td>.30***</td>
</tr>
<tr>
<td>Wave 6</td>
<td>.42***</td>
<td>.31***</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td>.54</td>
<td>.27</td>
</tr>
</tbody>
</table>

*Note. *** p < .001.*
5.3.2. Univariate Growth Curve Models

Next, separate univariate growth curve models were tested for each of the three substances to track individual trajectories of alcohol, cigarette, and marijuana use across the six waves using maximum likelihood estimation procedures available in LISREL 8 (Joreskog & Sorbom, 1996). These models provide measures of the average initial status and growth of substance use for the total sample as well as indications of variability in these measures among the total sample. Because of the need for complete data for all variables in latent growth curve modeling, listwise deletion was used in these initial latent growth curve models. Discussion of other methods to handle missing values will be presented later. Fixed parameter specifications of linear growth were specified for all three models. In all models, error terms at adjacent points in time were allowed to covary (e.g., between Time 1 and Time 2, Time 2 and Time 3, etc.). Modification indexes with values greater than 10 were used to identify additional significant correlated error terms.

Fit indices for these three models are reported in Table 10. The Normed Fit Index (NFI), Non-normed Fit Index (NNFI), Comparative Fit Index (CFI) and \( \chi^2 \)-test statistic are all commonly used to determine the fit of the hypothesized model of interest to the actual observed data. The \( \chi^2 \)-test statistic provides a measure of the overall fit of the model, with nonsignificant results indicating a good fit. However, because the \( \chi^2 \)-test statistic is highly sensitive to sample size, values for the NFI, NNFI, and CFI are often used as better indicators of fit. For all three, values greater than .90 indicate that the model does an adequate job of representing the observed data. Examination of Table 10 indicates that the latent growth models for all three substances appear to fit the data reasonably well.
Table 10
*Fit Indices for Separate Use Models*

<table>
<thead>
<tr>
<th></th>
<th>NFI</th>
<th>NNFI</th>
<th>CFI</th>
<th>( \chi^2 )</th>
<th>N</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol Use</td>
<td>.92</td>
<td>.90</td>
<td>.93</td>
<td>72.76</td>
<td>314</td>
<td>10</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Cigarette Use</td>
<td>.98</td>
<td>.98</td>
<td>.99</td>
<td>27.81</td>
<td>307</td>
<td>10</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Marijuana Use</td>
<td>.93</td>
<td>.90</td>
<td>.94</td>
<td>37.63</td>
<td>331</td>
<td>6</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

However, although the goodness of fit indices for the alcohol, cigarette, and marijuana use growth curve models were similar, comparison of means and variances of the growth parameters (Table 11) reveals different patterns of growth among alcohol, cigarette, and marijuana use. Specifically, estimated values for the initial levels and rates of change were more similar for alcohol and cigarette use compared to the marijuana use model. That is, initial levels of marijuana use were lower than those for alcohol and cigarette use, and the rate of increase in marijuana use was much less than that for either alcohol or cigarette use. Similarly, significant variation in initial level was found only in the alcohol and cigarette use models.

Another similar pattern can be found in the estimated covariance of intercept and slope among the three substances. For alcohol and cigarette use, a significant negative covariance indicated that those individuals who started out at lowest levels of initial use reported the greatest increase in use across the six waves. The covariance between initial status and slope of marijuana use, however, was not significant. Thus, there was no relation between the adolescents' Time 1 marijuana use and their subsequent change in marijuana use across the six waves.
Table 11
Means and Variances of Growth Parameters for Separate Use Models

<table>
<thead>
<tr>
<th></th>
<th>Intercept</th>
<th>Slope</th>
<th>Covariance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M_i$</td>
<td>$t$</td>
<td>$D_i$</td>
</tr>
<tr>
<td>Alcohol Use</td>
<td>1.43</td>
<td>28.58</td>
<td>.63</td>
</tr>
<tr>
<td>Cigarette Use</td>
<td>1.38</td>
<td>30.71</td>
<td>.69</td>
</tr>
<tr>
<td>Marijuana Use</td>
<td>1.01</td>
<td>86.09</td>
<td>.00</td>
</tr>
</tbody>
</table>

5.3.3. The Associative Model

To further examine the relationships among alcohol, cigarette, and marijuana use, an associative latent growth model was estimated next. For pragmatic reasons, the EQS structural equation program (Bentler, 1995) was used for the associative and higher order models (see Appendices I-III for program controls). This model allows for the simultaneous estimation of means, variances, and covariances for the growth curve parameters for all three substances. Model fitting procedures indicated a less than ideal fit of the associative model to the observed data: $\chi^2 (126, N = 300) = 809.38, p < .001; \text{NFI} = .76, \text{NNFI} = .77, \text{CFI} = .79$.

Means and variances of the growth curve parameters are reported in Table 12. These results display much the same pattern as seen in the separate univariate growth curve models above, though with a few exceptions. As seen in the univariate models, the average initial levels of alcohol and cigarette use were higher than the average initial level of marijuana use. These results suggest that younger adolescents are more likely to use alcohol or cigarettes compared to marijuana. Furthermore, the average slopes for alcohol and cigarette use are more than four times the average rate of change reported for marijuana use, suggesting that as a whole, the sample reported higher increases in alcohol and cigarette use across time.
compared to changes in marijuana use. A significant correlation between initial level and slope was found only for alcohol use in the associative model. This indicates that adolescents who reported lower levels of alcohol use at the first wave reported the greatest increase in alcohol use across the time of data collection. No association was found between initial status and slope for either cigarette or marijuana use in the associative model. The significant values for variances of the initial status and slope for all three substances indicated that substantial individual differences were found among the sample for these growth parameters.

Table 12
*Means and Variances of Growth Parameters for the Associative Model*

<table>
<thead>
<tr>
<th>Substrate</th>
<th>Intercept</th>
<th>Intercept</th>
<th>Slope</th>
<th>Slope</th>
<th>Covariance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M_i</td>
<td>t</td>
<td>D_i</td>
<td>t</td>
<td></td>
</tr>
<tr>
<td>Alcohol Use</td>
<td>1.39</td>
<td>.02</td>
<td>26.56</td>
<td>.54</td>
<td>9.23</td>
</tr>
<tr>
<td>Cigarette Use</td>
<td>1.41</td>
<td>.00</td>
<td>28.85</td>
<td>.60</td>
<td>10.07</td>
</tr>
<tr>
<td>Marijuana Use</td>
<td>1.00</td>
<td>.00</td>
<td>316.65</td>
<td>.00</td>
<td>12.23</td>
</tr>
</tbody>
</table>

The relationships among the intercepts and slopes of alcohol, cigarette, and marijuana use are presented in Table 13. In general, these results show stronger correlations among the alcohol and cigarette use growth parameters compared to those between marijuana use and the other two substances. For example, the correlation between the initial levels of alcohol and cigarette use was .776. This suggests that adolescents who reported high initial levels of alcohol use were also likely to report high initial levels of cigarette use. In contrast, the correlation between initial levels of marijuana use and alcohol or use was not significant and the correlation between initial levels of marijuana use and cigarette use, though significant, was only .125. The highest correlation between rates of change was also found between
alcohol and cigarette use (.567). The correlation between tobacco and marijuana use slopes was slightly less (.466) while the correlation between change in alcohol use and change in marijuana use was only .274.

Table 13

**Standardized Covariances among Growth Parameters for Alcohol, Tobacco, and Marijuana Use in Associative Model**

<table>
<thead>
<tr>
<th></th>
<th>Alcohol</th>
<th>Tobacco</th>
<th>Marijuana</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Int</td>
<td>Slope</td>
<td>Int</td>
</tr>
<tr>
<td>Alcohol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>1.000</td>
<td>-.359***</td>
<td>1.000</td>
</tr>
<tr>
<td>Slope</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tobacco</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>.776***</td>
<td>-.362***</td>
<td>1.000</td>
</tr>
<tr>
<td>Slope</td>
<td>.051</td>
<td>.567***</td>
<td>-.083</td>
</tr>
<tr>
<td>Marijuana</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>-.043</td>
<td>-.017</td>
<td>.125*</td>
</tr>
<tr>
<td>Slope</td>
<td>.299***</td>
<td>.274**</td>
<td>.288***</td>
</tr>
</tbody>
</table>

Note. *p < .05; **p < .01; ***p < .001.

5.3.4. The Factor-Of-Curves Model

The next step in the analyses was to fit the two higher-order models. Although it is not possible to statistically compare these two second-order models, Duncan et al. (1999) suggest that it is useful to fit both alternative models and compare them in the context of various common model parameters. Goodness of fit indices for the factor-of-curves model indicated that a higher-order common factor representation of alcohol, cigarette, and marijuana use may not be appropriate: \( \chi^2 (136, N = 300) = 922.44, p < .001; \) NFI = .73, NNFI = .76, CFI = .76. Although significant mean levels were found for the common intercept \( (M_i = 1.41, t = 28.77) \) and slope \( (M_s = .27, t = 15.53) \), neither of the growth
parameters for marijuana use was significant. Significant variation in the common intercept 
($D_i = .37, t = 6.55$) and slope ($D_s = .03, t = 6.97$) were also found.

5.3.5. The Curve-Of-Factors Model

Model fitting procedures resulted in the following indices of fit for the curve-of-
factors model: $\chi^2 (89, N = 300) = 335.20, p < .001; \text{NFI} = .90, \text{NNFI} = .88, \text{CFI} = .92$.
Parameter estimates of the curve-of-factors model indicated significant mean levels for the 
common intercept ($M_i = 1.39, t = 30.01$) and slope ($M_s = .27, t = 16.60$). Significant 
individual variation was also found for the common intercept ($D_i = .59, t = 9.11$) and slope 
($D_s = .05, t = 5.86$) in the curve-of-factors model.

5.3.6. Adequacy Of Combined Measures Of Substance Use

The results of the several different types of analyses reported above seem to support 
the notion that the growth parameters for alcohol and cigarette use are similar, yet each 
different from the use of marijuana. For example, the zero-order correlations between alcohol 
use and cigarette use at each wave were consistently greater than the correlations between 
marijuana and alcohol use as well as the correlations between marijuana and cigarette use. 
Modest correlations were also found when comparing the reported influence of both parents 
and friends on adolescents’ decisions to use alcohol and cigarettes.

Further evidence that a combined measure of alcohol and cigarette use (and excluding 
marijuana use) was warranted can be found in the results of the univariate as well as first and 
second-order combined latent growth curve models. In all of the models, the average initial 
levels and rates of change of alcohol and cigarette use were similar and higher than average 
levels and slopes of marijuana use. These results suggest that adolescents in this sample
followed a similar pattern of alcohol and cigarette use that starts at a higher level and escalates at a higher rate than that of marijuana use.

Finally, the results of the factor-of-curves and curve-of-factors models offer additional support for considering a combined measure of alcohol and cigarette use. The goodness of fit indices for the factor-of-curves were all well below the .90 level, which indicated that a higher-order common substance use factor was not justified for this sample. These results suggest that the higher order models do a poor job of representing the population parameters. That is, the restrictions imposed by these higher order models do not reflect the actual data. Though the fit indices for the curve-of-factors model were higher than the factor-of-curves model, none were substantially higher than the generally accepted cutoff value of .90. Again, these indicate that a 3-factor higher-order substance use factor was generally not warranted for this sample. Thus, combined measures of alcohol and cigarette use, and of parent and friend influence to use alcohol and cigarettes were used in further analyses.

5.4. Attrition Analyses

A common concern in most longitudinal studies involves the issue of subject attrition across the years of the study (Duncan & Duncan, 1995). One common method of dealing with missing data is to simply exclude any case that has missing data for any of the variables in the analysis. This method is known as listwise deletion, casewise deletion, or complete case analysis (Allison, 2001). However, a major disadvantage of using listwise deletion is that it often excludes a sizable proportion of the original sample, particularly in multivariate studies (Allison, 2001). Another disadvantage of listwise deletion is that this approach assumes that the data are missing completely at random (MCAR), a questionable assumption,
which, if violated, may lead to inaccurate estimates and large sample bias (Duncan & Duncan, 1995; also see Muthen, Kaplan, & Hollis, 1987).

5.4.1. Attrition Groups

To deal with this issue, Duncan and Duncan (1994) describe a procedure based on Muthen et al. (1987) that adjusts for biases due to systematic differences between subgroups, such as respondents and nonrespondents. This procedure, which defines a model for the partially missing data, allows the researcher to evaluate underlying model assumptions such as whether the data are indeed MCAR. By taking the incompleteness of the data into account, the model-based approach to missing data makes use of all potentially useful data and provides large sample estimates of the variance and covariance components of the model tested.

Muthen et al. (1987) have formulated an extension of the general latent growth curve model that includes partially missing data. This procedure involves first dividing the sample into subgroups that represent the different patterns of missing data and then placing equality constraints across the missing data groups to obtain unbiased and consistent estimates for each measure of interest. Table 14 provides the observed means and standard deviations by attrition group, representing various stages of completeness, at each time point for the two primary measures of interest—alcohol and cigarette use. Thus, for alcohol use, group 1 \((N = 314)\) represents participants with complete data across all six waves, group 2 \((N = 44)\) is those with alcohol use data at only the first five waves, group 3 \((N = 13)\) for the first four waves, and so on. Also included in each set of attrition groups is a group labeled “other.” This group represents those participants who dropped out yet returned to the study at a later point in time.
### Table 14
**Means and Standard Deviations for Alcohol and Cigarette Use by Attrition Group**

<table>
<thead>
<tr>
<th>Group</th>
<th>Year 1</th>
<th>Year 2</th>
<th>Year 3</th>
<th>Year 4</th>
<th>Year 5</th>
<th>Year 6</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Alcohol Use</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>1.45</td>
<td>.89</td>
<td>1.76</td>
<td>1.08</td>
<td>1.90</td>
<td>1.16</td>
</tr>
<tr>
<td>2</td>
<td>1.64</td>
<td>.99</td>
<td>1.71</td>
<td>1.00</td>
<td>2.11</td>
<td>1.30</td>
</tr>
<tr>
<td>3</td>
<td>1.08</td>
<td>.28</td>
<td>1.69</td>
<td>1.03</td>
<td>2.23</td>
<td>1.24</td>
</tr>
<tr>
<td>4</td>
<td>1.68</td>
<td>1.11</td>
<td>1.84</td>
<td>1.02</td>
<td>2.21</td>
<td>1.44</td>
</tr>
<tr>
<td>5</td>
<td>1.64</td>
<td>.92</td>
<td>2.27</td>
<td>1.35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>1.29</td>
<td>.83</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>other</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| Cigarette Use | | | | | | | | | | | | | |
| 1     | 1.38 | .79  | 1.68 | 1.02 | 1.97 | 1.24 | 2.26 | 1.37 | 2.44 | 1.43 | 2.72 | 1.45 | 307 |
| 2     | 1.86 | 1.10 | 2.17 | 1.10 | 2.33 | 1.24 | 2.67 | 1.51 | 3.29 | 1.54 |        |        | 42  |
| 3     | 1.50 | .80  | 2.08 | 1.51 | 2.25 | 1.49 | 2.83 | 1.95 |        |        |        |        | 12  |
| 4     | 1.63 | 1.12 | 1.79 | 1.27 | 2.58 | 1.74 |        |        |        |        |        |        | 19  |
| 5     | 2.27 | 1.49 | 2.46 | 1.57 |        |        |        |        |        |        |        |        | 11  |
| 6     | 1.85 | 1.35 |        |        |        |        |        |        |        |        |        |        | 13  |
| other |        |        |        |        |        |        |        |        |        |        |        |        | 96  |

### 5.4.2. Chi-Square Analyses

As a first step in determining if there were significant differences among the different groups, each of the attrition group variables was recoded into a trichotomous variable that distinguished between those who were present at all six waves, those with consecutive participation across fewer than six waves, and those who dropped out and later returned. Chi-square analyses were then conducted by cross-tabulating the trichotomous attrition group variables with several demographic variables, including target gender, age, parent income, mother and father education, and mother and father alcohol use. These results indicated that target females were more likely than target males to have complete data across all six waves of data collection for both substances (all \( \chi^2 [df = 2] > 16.00, ps < .001 \)). Missing data patterns for both substances also differed according to the level of the fathers' education.
Participants whose father was at least a high school graduate were more likely to have complete data across all six waves, with the highest participation among those with fathers who attended at least some college (all $\chi^2$ [df = 6] > 24.00, ps < .001).

5.4.3. Patterns Of Missing Data

Proceeding as outlined by Duncan and Duncan (1994), a model-based approach was used next to determine the missing-data mechanism of the data used in this study. Three different missing-data mechanisms (or patterns) are possible (Allison, 2001). In the simple, bivariate case, when the pattern of missing data on response variable Y is independent of both Y and an independent variable, X, the data are missing completely at random (MCAR), meaning that the missing data occurred by chance. In this situation, respondents with missing data can be treated as distinct random samples from the same population and the missing data pattern is considered ignorable.

Alternatively, when the missingness of values for Y is dependent on values of observed variable X, the data are considered missing at random (MAR). For example, suppose some participants report their gender but do not respond to an item concerning their use of alcohol. If the probability that alcohol use is missing varies according to the participant's gender, but does not vary according to the level of alcohol use within each gender, the missing data pattern is MAR. This means that the probability of missing data on alcohol use depends on gender, but within each gender, the probability of missing alcohol use data was unrelated to the level of alcohol use. In other words, even though males may have been more likely than females to have not reported their level of alcohol use, those who did respond to the alcohol use item reported the same distribution of values as females. When missing data are MAR, the values of Y can be considered a random sample of the sampled
values defined by the values of $X$ (Duncan & Duncan, 1995). In this case, the missing-data mechanism is also ignorable.

The third missing-data pattern occurs when the distribution of the missing data depends on values for the dependent variable $Y$. In this situation, the respondents are systematically different from the nonrespondents and the missing-data pattern is not ignorable. For example, when adolescents fail to report high levels of alcohol use for no particular reason, the mechanism related to the nonresponse is unobserved and indeterminable. In these situations, the missing data mechanism must be modeled in order to obtain good estimates of the parameters of interest (Allison, 2001). One such model-based approach to missing data that incorporates a latent growth curve model is described by Duncan et al. (1999).

5.4.4. Model-Based Approach To Missing Data

The model-based approach to handling missing data consists of the simultaneous analysis of multiple groups representing the various patterns of complete data available. Duncan et al. (1999) outline this strategy and their method is used as the basis for the following. The first step divides the sample into separate subgroups with distinct missing data patterns (see Table 14 on p. 66) and then expands structural equation modeling to include means, or regression intercepts to estimate a latent growth curve model. A multiple-group analysis is then conducted by placing equality constraints across the missing-data groups to obtain unbiased and consistent estimates. These equality constraints are then used to test both an unrestricted $H_1$ hypothesis and a restricted $H_0$ hypothesis.

The unrestricted, or saturated, $H_1$ model tests whether the data are MCAR. The variances, covariances, and means of the observed variables are the parameters of interest in
this step of the analysis. The $H_I$ hypothesis tests the equality of the moment matrices representing the across-group equality constraints for the variances, covariances, and means of the observed (Y) variables. A non-significant chi-square value and high goodness-of-fit indices (e.g., NFI > .90) indicate that MCAR cannot be rejected for the different subsamples. Under this condition, the different subsamples can be regarded as random samples from the same population and all corresponding parameters should be equal across the groups. In other words, if the $H_I$ hypothesis is not rejected, the attrition groups listed in Table 14 would be considered random sub-samples of the entire sample.

The restricted $H_0$ model represents the hypothesized model of interest (a latent growth curve in this case), assumed to be invariant across the different groups. This model involves the test of equality of the moment matrices reflecting equality constraints for regression coefficients, variances, and covariances of the independent (X) variables, and regression intercepts, or means, of the dependent (Y) variables. The difference in chi-square values for the two models provides a test of the hypothesized model. If this difference is non-significant, based on the difference in degrees of freedom, and the missing data mechanism is MAR, correct maximum likelihood (ML) estimates are obtained from the $H_0$ model. Duncan et al (1999) note that even if one cannot assume MAR, this multi-sample approach is likely to reduce biases inherent in listwise deletion, commonly used in SEM analyses.

Tests of the $H_I$ and $H_0$ hypotheses were conducted separately for each of the two substances—alcohol and cigarettes. For the alcohol use models, the data in group 1 ($N = 314$) represents the complete data available for the analysis; in other words, these are the data that are obtained from listwise deletion of missing data. As seen in Table 14 (p. 66), if data for five data points were used instead of six, an additional 44 participants would be included.
(group 2). If four data points were used, the 13 participants in group 3 would be included, and so on for the rest of the attrition groups. Similarly, the data contained in group 1 ($N = 307$) of the cigarette use models represents the complete data. The covariance matrices for each of the attrition groups are included in Appendix IV.

Previous authors have noted the limitations of the LISREL program in estimating models with incomplete data. Specifically, in multisample analyses, because LISREL requires the same number of variables in each sample, "special tricks" are necessary to specify the model (Allison & Hauser, 1991). These special techniques are not necessary in the EQS program; therefore, all missing data models were estimated using EQS (Bentler, 1995; see Appendices V and VI). For groups with incomplete data, values of "0" were used in the covariance matrices and vectors of means and standard deviations to allow for the simultaneous estimation of the latent growth curve models across the groups.

Table 15 presents the fit indices for the $H_I$ and $H_0$ models, as well as the listwise models from above. Results of the $\chi^2$ difference tests for $H_I$ versus $H_0$ are also presented. For the unrestricted ($H_I$) alcohol use missing data model, the $\chi^2$-test statistic equaled 77.08 with 48 degrees of freedom. Though this value is significant ($p < .005$), which indicates a less than adequate fit to the observed data, the $\chi^2$-test statistic is sensitive to large sample sizes. Therefore, it is useful to also examine other indices of fit such as the NFI, NNFI, and CFI to determine if the hypothesized model adequately represented the relationships among the observed variables. The goodness of fit indices for the alcohol use MCAR model ($H_I$) are all greater than .90, suggesting that the model provides an adequate fit to the data.

Similarly, the $\chi^2$-test statistic for the unrestricted ($H_I$) cigarette use missing data model was significant: $\chi^2 (48, N = 391) = 154.63, p < .001$. However, the goodness of fit
indices (NFI, NNFI, CFI) again were all greater than .90, which indicated the $H_1$ model was
tenable for this data set. However, because it was found earlier that complete data for both
alcohol and cigarette use were more likely to occur among females and participants with
more highly educated fathers, the missing patterns cannot be treated as MCAR for either of
the two substances. Rather, based on these results it is more reasonable to make the
assumption that the missing data are MAR, or at least ignorable.

Table 15
Fit Indices for Alcohol and Cigarette Use Missing Data Models

<table>
<thead>
<tr>
<th></th>
<th>NFI</th>
<th>NNFI</th>
<th>CFI</th>
<th>$\chi^2$</th>
<th>$N$</th>
<th>df</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol Use $^a$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$H_1$ (MCAR)</td>
<td>.93</td>
<td>.98</td>
<td>.97</td>
<td>77.08</td>
<td>401</td>
<td>48</td>
<td>.005</td>
</tr>
<tr>
<td>$H_0$</td>
<td>.79</td>
<td>.91</td>
<td>.84</td>
<td>223.49</td>
<td>401</td>
<td>60</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Listwise model</td>
<td>.84</td>
<td>.81</td>
<td>.85</td>
<td>150.38</td>
<td>314</td>
<td>12</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Cigarette Use $^b$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$H_1$ (MCAR)</td>
<td>.91</td>
<td>.96</td>
<td>.94</td>
<td>154.63</td>
<td>391</td>
<td>48</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>$H_0$</td>
<td>.84</td>
<td>.93</td>
<td>.87</td>
<td>280.31</td>
<td>391</td>
<td>60</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Listwise model</td>
<td>.92</td>
<td>.90</td>
<td>.92</td>
<td>132.69</td>
<td>307</td>
<td>12</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

$a$ Difference test for alcohol use: $\chi^2(12, N = 401) = 146.41, p < .001$.

$b$ Difference test for cigarette use: $\chi^2(12, N = 391) = 125.68, p < .001$.

Under the assumption of an ignorable missing data pattern (e.g., MAR), the $\chi^2$
difference test provides a correct test of the hypothesized model of interest (Duncan et al.,
1999). If the missing data mechanism is ignorable, and the test of $H_0$ against $H_1$ does not lead
to rejection (i.e., a non-significant $\chi^2$ difference), correct maximum likelihood estimates of
model parameters are obtained in the $H_0$ model. However, even when the missing data
mechanism is non-ignorable, this multi-sample method is likely to reduce the biases inherent
in complete-case (i.e., listwise deletion) analyses (Duncan et al., 1999). The results of both
\( \chi^2 \) difference tests seem to fall into this situation. Though both tests resulted in significant \( \chi^2 \) differences, based on the degrees of freedom, the maximum likelihood estimates obtained in the \( H_0 \) model (utilizing all available data) are not expected to exhibit large sample biases.

5.5. Data Imputation

Based on the results of the missing-data analyses, it was determined that imputation of missing data was justified. To accomplish this, combined measures of alcohol and cigarette use were created, as well as combined measures of parent and friend influence to use the two substances. Next, SPSS Missing Values Analysis (MVA) was used to impute missing values using the expectation-maximization (EM) algorithm. The EM algorithm (Dempster, Laird, & Rubin, 1977) is a general approach for computing maximum likelihood estimates of parameters from incomplete data. This approach consists of two iterative steps: (1) replace missing values with estimated values and estimate the model parameters (expectation, or \( E \)-step), and (2) maximize the fitting function to estimate the unknown parameters as if the missing data were observed (maximization, or \( M \)-step). The EM algorithm converges when estimates for the means and covariance matrix do not change from one iteration to the next; this produces a single complete data set with all missing values replaced with imputed estimates from the algorithm. Table 16 provides descriptive statistics for all variables provided by complete case analysis, listwise deletion, and EM imputation.
Table 16
Descriptive Statistics For Complete Data, Listwise Deletion, And Imputed With EM

<table>
<thead>
<tr>
<th>Variable</th>
<th>Complete data</th>
<th>Listwise Deletion (N=230)</th>
<th>EM Imputation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (N)</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Maluse</td>
<td>1.82 (489)</td>
<td>.58</td>
<td>1.86</td>
</tr>
<tr>
<td>Faluse</td>
<td>2.16 (437)</td>
<td>.92</td>
<td>2.08</td>
</tr>
<tr>
<td>Meduc</td>
<td>3.54 (488)</td>
<td>.56</td>
<td>3.59</td>
</tr>
<tr>
<td>Feduc</td>
<td>3.53 (439)</td>
<td>.61</td>
<td>3.65</td>
</tr>
<tr>
<td>Income</td>
<td>2.37 (473)</td>
<td>.96</td>
<td>2.54</td>
</tr>
<tr>
<td>Parent Support</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time 1</td>
<td>3.10 (473)</td>
<td>.69</td>
<td>3.21</td>
</tr>
<tr>
<td>Time 2</td>
<td>2.88 (477)</td>
<td>.74</td>
<td>2.98</td>
</tr>
<tr>
<td>Time 3</td>
<td>3.01 (460)</td>
<td>.72</td>
<td>3.14</td>
</tr>
<tr>
<td>Friend Support</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time 1</td>
<td>3.12 (483)</td>
<td>.66</td>
<td>3.17</td>
</tr>
<tr>
<td>Time 2</td>
<td>3.04 (474)</td>
<td>.67</td>
<td>3.09</td>
</tr>
<tr>
<td>Time 3</td>
<td>3.10 (452)</td>
<td>.70</td>
<td>3.16</td>
</tr>
<tr>
<td>Parent Influence</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time 1</td>
<td>4.50 (493)</td>
<td>1.79</td>
<td>4.73</td>
</tr>
<tr>
<td>Time 2</td>
<td>4.35 (475)</td>
<td>1.84</td>
<td>4.56</td>
</tr>
<tr>
<td>Time 3</td>
<td>4.28 (463)</td>
<td>1.87</td>
<td>4.43</td>
</tr>
<tr>
<td>Time 4</td>
<td>4.03 (411)</td>
<td>1.88</td>
<td>4.26</td>
</tr>
<tr>
<td>Time 5</td>
<td>3.94 (407)</td>
<td>1.79</td>
<td>4.15</td>
</tr>
<tr>
<td>Time 6</td>
<td>3.59 (379)</td>
<td>1.78</td>
<td>3.79</td>
</tr>
<tr>
<td>Friend Influence</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time 1</td>
<td>3.37 (492)</td>
<td>1.66</td>
<td>3.33</td>
</tr>
<tr>
<td>Time 2</td>
<td>3.37 (475)</td>
<td>1.66</td>
<td>3.33</td>
</tr>
<tr>
<td>Time 3</td>
<td>3.38 (463)</td>
<td>1.63</td>
<td>3.47</td>
</tr>
<tr>
<td>Time 4</td>
<td>3.26 (411)</td>
<td>1.61</td>
<td>3.31</td>
</tr>
<tr>
<td>Time 5</td>
<td>3.12 (407)</td>
<td>1.50</td>
<td>3.21</td>
</tr>
<tr>
<td>Time 6</td>
<td>2.78 (379)</td>
<td>1.59</td>
<td>2.98</td>
</tr>
<tr>
<td>Alc/Cig Use</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time 1</td>
<td>1.51 (485)</td>
<td>.82</td>
<td>1.38</td>
</tr>
<tr>
<td>Time 2</td>
<td>1.80 (474)</td>
<td>.98</td>
<td>1.68</td>
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<tr>
<td>Time 3</td>
<td>2.05 (463)</td>
<td>1.13</td>
<td>1.85</td>
</tr>
<tr>
<td>Time 4</td>
<td>2.53 (407)</td>
<td>1.26</td>
<td>2.41</td>
</tr>
<tr>
<td>Time 5</td>
<td>2.83 (407)</td>
<td>1.21</td>
<td>2.65</td>
</tr>
<tr>
<td>Time 6</td>
<td>3.11 (376)</td>
<td>1.15</td>
<td>2.97</td>
</tr>
</tbody>
</table>
5.6. Hypothesis Testing

5.6.1. Parental and Friend Support

*Hypothesis One:* the level of parental support will decrease linearly across the adolescent period.

*Hypothesis Two:* support from friends will show a positive linear trend, increasing during this time.

The first step in the hypothesis testing analyses consisted of tests for the presence of change in parent and friend support over the first three years of the study. Participants were between the ages of 13 years to 17 years during this time. To test these hypotheses, two separate univariate growth curves (one for parent support and one for friend support) were estimated using the imputed covariance matrix in the LISREL 8.3 (Joreskog & Sorbom, 2001) structural equation modeling software program, with sample size set to $N = 230$. Although there remains debate about the most appropriate sample size to use when imputed data are used in structural models, this approach yields conservative estimates for model parameters (since standard error values decrease as sample size increase). Thus, this approach reduces the likelihood of committing a Type I error.

*Parental support.* A two-factor LGC model was estimated for the three repeated measures of parent support at Waves 1, 2, and 3. This model was found to provide a less than ideal fit to the data, $\chi^2 (1, N = 230) = 21.47, p < .001$; NFI = .88, NNFI = .66, CFI = .89, RMSEA = .29. Table 17 presents the estimates of growth parameters for the parent support LGC model. The average initial level of parental support at Wave 1 ($M_t$) was 3.05; the significant value for the variance of intercept ($D_I = .34, t = 5.33$) indicated that significant variation existed among the adolescents in their reported level of parent support at Wave 1. A significant negative mean for the slope factor ($M_s = -.05, t = -2.19$) indicated that the total
sample reported decreases in parental support across these three waves, and variation in the slope factor was not significant. The correlation between the intercept and slope factors was not significant.

**Friend support.** A second two-factor LGC model was also estimated for the three repeated measures of friend support across the first three waves. This model was found to fit the data very well, $\chi^2 (1, N = 230) = 3.05, p = .081; \text{NFI} = .98, \text{NNFI} = .96, \text{CFI} = .99, \text{RMSEA} = .09$. The estimates of growth parameters for the friend support LGC model are also presented in Table 17. As seen, the average initial level of parental support at Wave 1 ($M_1 = 3.10, t = 73.18$) was significant as was the variance of the intercept ($D_1 = .28, t = 5.16$). This indicated that significant variation existed among the adolescents in their reported level of friend support at Wave 1. Neither parameters concerning change in friend support (mean or variance) were significant. This indicated that the overall group did not report changes in the level of friend support nor was there significant variation among the individuals in the change in support from friends. The correlation between the intercept and slope factors for friend support was also not significant.

### Table 17
*Means and Variances for Univariate Latent Growth Curve Models using Imputed Covariance Matrices*

<table>
<thead>
<tr>
<th></th>
<th>Intercept</th>
<th>Slope</th>
<th>Covariance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M_1$</td>
<td>$t$</td>
<td>$D_1$</td>
</tr>
<tr>
<td>Parent Support</td>
<td>3.05</td>
<td>67.45</td>
<td>.34</td>
</tr>
<tr>
<td>Friend Support</td>
<td>3.10</td>
<td>73.18</td>
<td>.28</td>
</tr>
<tr>
<td>Parent Influence</td>
<td>4.54</td>
<td>41.58</td>
<td>1.90</td>
</tr>
<tr>
<td>Friend Influence</td>
<td>3.49</td>
<td>37.10</td>
<td>.97</td>
</tr>
</tbody>
</table>
5.6.2. Parental and Friend Influence

Hypothesis Three: the level of parent influence on adolescents' substance use decisions will be highest at early adolescence, decreasing through mid- to late adolescence.

Hypothesis Four: the level of friend influence on this decision-making process, lowest in early adolescence, will increase during the transitions across early, mid, and late-adolescence.

The second set of hypotheses was also tested using univariate growth curve models. Because these two models used data from all six waves of the data collection, they represent patterns of change for interpersonal influence on substance use decisions across the age range of 13 years to 20 years. As before, the imputed covariance matrices from the EM algorithm were used, setting the sample size at $N = 230$.

Parent influence. A two-factor LGC model was estimated for the six combined measures of parental influence to use alcohol and cigarettes. This model was found to fit the observed data reasonably well, $\chi^2 (16, N = 230) = 48.10, p < .001; \text{NFI} = .91, \text{NNFI} = .95, \text{CFI} = .94, \text{RMSEA} = .10$. Examination of Lagrangian multiplier (LM) tests for this model indicated that three of the error covariances for the observed variables should be estimated in the model; therefore, the original model was respecified, freeing these covariances, and estimated a second time. The revised model fit the observed data well, $\chi^2 (13, N = 230) = 21.86, p = .06; \text{NFI} = .96, \text{NNFI} = .98, \text{CFI} = .98, \text{RMSEA} = .06$. Because the chi-square difference between these two models was significant ($\Delta \chi^2 (3) = 26.24, p < .001$), the revised model was deemed the more appropriate of the two. Estimates of growth parameters for the parent influence model are presented in Table 17 (p. 75). The average initial level of parental influence at Wave 1 ($M_I$) was 4.54 and significant ($t = 41.58$). The mean value for the slope of parental influence was negative and significant ($M_s = -.20, t = -7.47$), which indicated that
the adolescents as a whole reported a decline in the amount of parental influence across the six waves. Significant variance components for both the intercept ($D_0 = 1.90, t = 6.94$) and the slope ($D_1 = .09, t = 5.03$) factors provided evidence that there were significant individual differences in both initial levels and change in parental influence over time. The significant negative covariance between the intercept and slope factors ($\text{Cov}_{\delta} = -.18, t = -3.04$) indicated an inverse relation between initial levels of parental influence and change in influence over time. Thus, adolescents with the highest levels of parent influence at Time 1 tended to report less decline in this influence across time.

*Friend influence.* Another two-factor LGC model was estimated for the six repeated measures of the combined index of friend influence to use alcohol and cigarettes. As in the LCG model for parent influence, the initial model was found to provide an adequate fit to the data, $\chi^2 (16, N = 230) = 45.28, p < .001$; NFI = .85. NNFI = .91, CFI = .90 RMSEA = .09, but LM tests suggested that the fit could be improved by freeing two error covariances. The revised model for friend influence fit the observed data well, $\chi^2 (14, N = 230) = 26.68, p = .02$; NFI = .91. NNFI = .95, CFI = .96, RMSEA = .06. Again, the chi-square difference between these two models was significant ($\Delta \chi^2 (2) = 19.04, p < .001$) and the revised model was used. Estimates of growth parameters for the friend influence model are presented in Table 17. The average initial level of friend influence at Wave 1 ($M_0 = 3.49, t = 37.10$) was significant, as was the mean value for the slope ($M_1 = -.12, t = -4.70$), which indicated that the adolescents as a whole reported a decline in their friend influence over time. Significant variance components for both the intercept ($D_0 = .97, t = 4.61$) and the slope ($D_1 = .07, t = 4.26$) factors indicated that there were significant individual differences in both initial levels and change in friend influence over time. The significant negative covariance between the
intercept and slope factors (Cov\textsubscript{ts} = -1.2, t = -2.41) indicated an inverse relation between initial levels of friend influence and change in influence over time. Thus, adolescents with highest levels of friend influence at Time 1 tended to report less decline in this influence.

5.6.3. Relations Among Support, Influence, and Substance Use

Hypothesis five: growth parameters of the two sources of support will predict growth parameters of each of the respective sources of influence. That is, high levels of parental and friend support will predict high levels of parental and peer influence, respectively.

Hypothesis six: high initial levels of parent influence will be related to lower initial levels of adolescent substance use. Similarly, increasing rates of change in parental influence will be associated with decreasing trajectories of adolescent substance use.

Hypothesis seven: high initial levels of friend influence are hypothesized to predict elevated initial levels of adolescent substance use, whereas increasing rates of change in friend influence will predict increasing trajectories of adolescent use.

Nested models. In order to establish mediation, a nested models approach was used to test the relationships between social support, social influence, and substance use, controlling for the covariates (mother’s alcohol use, father’s alcohol use, and SES). Figure 5 displays the series of hierarchically related multivariate models used for this approach. The first model is labeled “Baseline” and is used to estimate direct paths from each of the covariates to the growth parameters (intercept and slope) of substance use. All other paths in the baseline model were constrained to zero. Next, Model A retains the paths from the Baseline model and adds direct paths from the social support growth parameter to the substance use intercept and slope. Thus, Model A represents a less restricted model than the baseline model because four paths are freed. In a sequential fashion, Model B adds four more estimated paths from the social influence growth parameters to the substance use growth parameters. The final step in the nested model approach is Model C, which estimates the six hypothesized paths from the social support parameters to the social influence parameters.
Baseline Model:
Covariates $\rightarrow$ Use

Model A: adding Support $\rightarrow$ Use

Figure 5. Nested Models.
Figure 5. (Continued).

Model B: Adding Influence $\rightarrow$ Use

Model C: Adding Support $\rightarrow$ Influence
Baseline model. Fitting the baseline model to the data resulted in a less than ideal fit, \( \chi^2(367, N = 230) = 533.95, p < .001; \) NFI = .82. NNFI = .93, CFI = .94 RMSEA = .05. Of the six estimated paths from the covariates to the substance use growth parameters, only two were significant or approached significance. The measure of SES had a significant negative effect on the adolescents' initial levels of substance use (\( \beta = -.28, t = -2.75, p = .007 \)). The direct path from mothers' use of alcohol to the adolescents' substance use slope approached significance (\( \beta = .13, t = 1.68, p = .09 \)).

Model A. Model A added direct paths from the social support parameters to the substance use parameters. This model resulted in the following fit indices, \( \chi^2(363, N = 230) = 516.32, p < .001; \) NFI = .83. NNFI = .93, CFI = .94 RMSEA = .04. Results indicated that the parental support intercept was negatively related to the initial level of adolescent substance use (\( \beta = -.28, t = -3.53, p < .001 \)), and the friend support intercept was positively related to the substance use intercept (\( \beta = .19, t = 2.35, p = .020 \)). Neither the parent support slope nor the friend support slope was found to be a significant predictor of the adolescents' rates of change (parent: \( \beta = -.19, t = -1.73, p = .086 \); friend: \( \beta = .15, t = 1.34, p = .182 \)).

Model B. Model B was estimated in the next step, which added direct paths from the social influence parameters to the substance use parameters. This model resulted in the following fit indices, \( \chi^2(359, N = 230) = 486.15, p < .001; \) NFI = .84. NNFI = .94, CFI = .95 RMSEA = .04. Tests of individual paths revealed that the intercept of the parent influence was negatively related to the substance use intercept (\( \beta = -.40, t = -5.19, p < .001 \)) and the friend influence intercept was positively related to the substance use intercept (\( \beta = .21, t = 2.49, p = .014 \)). Furthermore, positive change in parental influence was related to a decrease
in change in adolescent substance use ($\beta = -.22$, $t = -2.44$, $p = .016$). The path from the friend influence slope to adolescent use slope was not significant ($\beta = .04$, $t = .50$, $p = .62$).

**Model C.** The final model in the nested model approach, Model C, adds six additional parameters to the previous step—from the two support intercepts to the respective influence intercepts and slopes and the two support slopes to the respective influence slopes. This model provided an adequate fit to the data, $\chi^2 (353, N = 230) = 418.81$, $p = .009$; NFI = .86, NNFI = .97, CFI = .97 RMSEA = .03. Three of these additional parameters were significant: support intercept factors were positively related to their respective influence intercept (parent: $\beta = .54$, $t = 5.98$, $p < .001$; friend: $\beta = .28$, $t = 5.98$, $p = .006$). The parent support slope was positively related to change in parent influence ($\beta = .28$, $t = 2.01$, $p = .047$).

A summary of the path coefficients estimated in each of the nested models is presented in Table 18. This table lists each of the relations between the predictor (rows) and outcome variables (columns). In Model C, the parent and friend support parameters are predictors of parent and friend influence parameters, respectively (i.e., parent support intercept predicts parent support intercept and slope while parent support slope predicts parent influence slope). Comparison of the paths across the models indicates that when the paths from the parent and friend influence parameters to the use parameters are added (Model B), two previously significant paths in Model A become nonsignificant: parent support intercept $\rightarrow$ use intercept, friend support intercept $\rightarrow$ use intercept. In a similar manner, when paths from the support parameters to the influence parameters are estimated (Model C), two other previously significant paths (in Model B) become nonsignificant: parent influence slope $\rightarrow$ use slope, friend influence intercept $\rightarrow$ use intercept. No other changes in significance levels were found when adding additional paths in the subsequent models.
Table 18
Comparison of Hierarchically Nested Models: Predictors and Outcomes

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Influence $\pi_0$ (Int)</th>
<th>$\pi_1$ (Slope)</th>
<th>Use $\pi_0$ (Int)</th>
<th>$\pi_1$ (Slope)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline model</td>
<td>- -</td>
<td>- -</td>
<td>.08 (.99)</td>
<td>.13 (1.68)</td>
</tr>
<tr>
<td>Maluse</td>
<td>- -</td>
<td>- -</td>
<td>.03 (-.42)</td>
<td>.02 (.29)</td>
</tr>
<tr>
<td>Faluse</td>
<td>- -</td>
<td>- -</td>
<td>-.28 (-2.75)</td>
<td>-.08 (-.78)</td>
</tr>
<tr>
<td>SES</td>
<td>- -</td>
<td>- -</td>
<td>-.08 (.99)</td>
<td>.13 (1.68)</td>
</tr>
<tr>
<td>Faluse</td>
<td>- -</td>
<td>- -</td>
<td>-.08 (.99)</td>
<td>.13 (1.68)</td>
</tr>
<tr>
<td>SES</td>
<td>- -</td>
<td>- -</td>
<td>-.08 (.99)</td>
<td>.13 (1.68)</td>
</tr>
<tr>
<td>Maluse</td>
<td>- -</td>
<td>- -</td>
<td>.08 (1.00)</td>
<td>.13 (1.64)</td>
</tr>
<tr>
<td>Faluse</td>
<td>- -</td>
<td>- -</td>
<td>-.08 (-1.04)</td>
<td>.02 (.21)</td>
</tr>
<tr>
<td>SES</td>
<td>- -</td>
<td>- -</td>
<td>-.26 (-2.64)</td>
<td>-.08 (-.83)</td>
</tr>
<tr>
<td>psup int</td>
<td>- -</td>
<td>- -</td>
<td>-.28 (-3.53)</td>
<td>- -</td>
</tr>
<tr>
<td>psup slope</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
</tr>
<tr>
<td>fsup int</td>
<td>- -</td>
<td>- -</td>
<td>.19 (2.35)</td>
<td>- -</td>
</tr>
<tr>
<td>fsup slope</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
</tr>
<tr>
<td>Model B</td>
<td>- -</td>
<td>- -</td>
<td>.07 (1.00)</td>
<td>.13 (1.64)</td>
</tr>
<tr>
<td>Maluse</td>
<td>- -</td>
<td>- -</td>
<td>-.08 (-1.04)</td>
<td>.02 (.21)</td>
</tr>
<tr>
<td>Faluse</td>
<td>- -</td>
<td>- -</td>
<td>-.26 (-2.64)</td>
<td>-.08 (-.83)</td>
</tr>
<tr>
<td>SES</td>
<td>- -</td>
<td>- -</td>
<td>-.10 (-1.37)</td>
<td>- -</td>
</tr>
<tr>
<td>psup int</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
</tr>
<tr>
<td>psup slope</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
</tr>
<tr>
<td>fsup int</td>
<td>- -</td>
<td>- -</td>
<td>.13 (1.74)</td>
<td>- -</td>
</tr>
<tr>
<td>fsup slope</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
</tr>
<tr>
<td>pinf int</td>
<td>- -</td>
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<td>-.40 (-5.19)</td>
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</tr>
<tr>
<td>pinf slope</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
</tr>
<tr>
<td>finf int</td>
<td>- -</td>
<td>- -</td>
<td>.21 (2.49)</td>
<td>- -</td>
</tr>
<tr>
<td>finf slope</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
</tr>
<tr>
<td>Model C</td>
<td>- -</td>
<td>- -</td>
<td>.07 (.99)</td>
<td>.12 (1.51)</td>
</tr>
<tr>
<td>Maluse</td>
<td>- -</td>
<td>- -</td>
<td>-.07 (-.94)</td>
<td>.01 (-.10)</td>
</tr>
<tr>
<td>Faluse</td>
<td>- -</td>
<td>- -</td>
<td>-.26 (-2.63)</td>
<td>-.08 (-.81)</td>
</tr>
<tr>
<td>SES</td>
<td>- -</td>
<td>- -</td>
<td>.54 (5.98)</td>
<td>.01 (.06)</td>
</tr>
<tr>
<td>psup int</td>
<td>- -</td>
<td>- -</td>
<td>.01 (.06)</td>
<td>- -</td>
</tr>
<tr>
<td>psup slope</td>
<td>- -</td>
<td>- -</td>
<td>.28 (2.01)</td>
<td>- -</td>
</tr>
<tr>
<td>fsup int</td>
<td>- -</td>
<td>- -</td>
<td>.28 (2.79)</td>
<td>.27 (1.16)</td>
</tr>
<tr>
<td>fsup slope</td>
<td>- -</td>
<td>- -</td>
<td>.16 (2.1)</td>
<td>- -</td>
</tr>
<tr>
<td>pinf int</td>
<td>- -</td>
<td>- -</td>
<td>-.40 (-3.81)</td>
<td>- -</td>
</tr>
<tr>
<td>pinf slope</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
</tr>
<tr>
<td>finf int</td>
<td>- -</td>
<td>- -</td>
<td>.12 (1.29)</td>
<td>- -</td>
</tr>
<tr>
<td>finf slope</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>.43 (-1.81)</td>
</tr>
</tbody>
</table>

Note. Standardized coefficients; $t$-values in parentheses. New paths in **bold italics**.
Revised Model. Because the hypothesized model in Figure 2 did not include direct paths from all of the parameters to the two substance use parameters, a more restrictive model was tested by deleting the direct paths from the social support growth parameters to the use parameters. Because only two covariates were found in any of the nested models to predict either of the substance use parameters (mother’s alcohol use to adolescent use slope and SES to adolescent use intercept), the other paths from the covariates were deleted.

Fitting this more restricted model to the data resulted in good fit, $\chi^2 (361, N = 230) = 431.59, p = .007$; NFI = .86. NNFI = .97, CFI = .97 RMSEA = .03. After this revision, two paths that were not significant in Model C were now significant: increases in parent influence were related to decreases in adolescent use ($\beta = -.28, t = -2.77, p = .006$) and higher initial levels of friend influence were related to higher initial levels of adolescent use ($\beta = .26, t = 2.97, p = .004$). Significant indirect effects on initial levels of adolescent use were found for initial levels of both sources of support (parent: $\beta = -.24, t = -4.31, p < .001$; friend: $\beta = .09, t = 2.26, p = .026$). The indirect effect of rate of change in parental support on the slope of adolescent use was marginally significant ($\beta = -.08, t = -1.64, p = .10$). Mean levels and variances of the growth parameters from this model are reported in Table 19. Correlations among the latent constructs are included in Appendix VII.

Table 19
Means and Variances for Growth Parameters in Revised Model

<table>
<thead>
<tr>
<th></th>
<th>Intercept</th>
<th></th>
<th></th>
<th></th>
<th>Slope</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M_i$</td>
<td>$t$</td>
<td>$D_i$</td>
<td>$t$</td>
<td>$M_t$</td>
<td>$t$</td>
<td>$D_t$</td>
<td>$t$</td>
</tr>
<tr>
<td>Parent Support</td>
<td>3.06</td>
<td>66.48</td>
<td>.38</td>
<td>6.87</td>
<td>-.05</td>
<td>2.27</td>
<td>.06</td>
<td>2.74</td>
</tr>
<tr>
<td>Friend Support</td>
<td>3.11</td>
<td>73.24</td>
<td>.30</td>
<td>6.11</td>
<td>-.02</td>
<td>.87</td>
<td>.05</td>
<td>2.24</td>
</tr>
<tr>
<td>Parent Influence</td>
<td>.96</td>
<td>1.60</td>
<td>1.22</td>
<td>4.96</td>
<td>-.12</td>
<td>.78</td>
<td>.07</td>
<td>3.50</td>
</tr>
<tr>
<td>Friend Influence</td>
<td>1.55</td>
<td>2.59</td>
<td>.96</td>
<td>4.03</td>
<td>.10</td>
<td>.59</td>
<td>.08</td>
<td>3.95</td>
</tr>
<tr>
<td>Substance Use</td>
<td>3.97</td>
<td>5.44</td>
<td>.47</td>
<td>5.88</td>
<td>.21</td>
<td>4.53</td>
<td>.04</td>
<td>6.37</td>
</tr>
</tbody>
</table>
Comparison of nested models. To compare nested models, it is necessary to compare the $\chi^2$ value of the first, more restricted model, to the second, less restricted model, taking into account the degrees of freedom associated with each model. A significant drop in the $\chi^2$ value, with the difference in degrees of freedom determined by the number of additional parameters estimated in the less restricted model, indicates that the less restricted model represents an improvement over the more restricted model.

Table 20 summarizes the goodness of fit indicators for the four nested models estimated above and displays the $\chi^2$ difference obtained with each additional less restrictive model. Each step in the nested models approach resulted in a significant $\chi^2$ difference, relative to the change in degrees of freedom associated with fitting a less restrictive model (all $ps < .01$), suggesting that Model C is the most appropriate model of the four nested models. Furthermore, adding restrictions to this model, Model C-revised, resulted in a non-significant change in $\chi^2$ value ($p = .120$). Because Model C-revised represents a more parsimonious model than Model C (i.e., fewer parameters are estimated), it was deemed the most appropriate. Figure 6 displays the estimates of relationships among the growth parameters in the final revised model.

Table 20
Comparison of Hierarchically Nested Models: $\chi^2$ Difference Tests

<table>
<thead>
<tr>
<th>Model</th>
<th>$\chi^2$</th>
<th>df</th>
<th>$\Delta \chi^2$</th>
<th>$\Delta df$</th>
<th>Sig. of Change</th>
<th>NFI</th>
<th>NNFI</th>
<th>CFI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>533.95</td>
<td>367</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>.82</td>
<td>.93</td>
<td>.94</td>
</tr>
<tr>
<td>Model A</td>
<td>516.32</td>
<td>363</td>
<td>17.63</td>
<td>4</td>
<td>.002</td>
<td>.83</td>
<td>.93</td>
<td>.94</td>
</tr>
<tr>
<td>Model B</td>
<td>486.15</td>
<td>359</td>
<td>30.17</td>
<td>4</td>
<td>&lt;.001</td>
<td>.84</td>
<td>.94</td>
<td>.95</td>
</tr>
<tr>
<td>Model C</td>
<td>418.81</td>
<td>353</td>
<td>67.34</td>
<td>6</td>
<td>&lt;.001</td>
<td>.86</td>
<td>.97</td>
<td>.97</td>
</tr>
<tr>
<td>Model C (revised)</td>
<td>431.59</td>
<td>361</td>
<td>12.78</td>
<td>8</td>
<td>.120</td>
<td>.86</td>
<td>.97</td>
<td>.97</td>
</tr>
</tbody>
</table>
Figure 6. Final model of social support, interpersonal influence, and substance use (standardized maximum likelihood estimates; $t$-values in parentheses).
5.7. Group Differences (Multi-sample Analyses)

Multi-group analyses can also be conducted using the LISREL 8.3 software (Joreskog & Sorbom, 2001). In these analyses, structural models are estimated simultaneously for multiple groups. Significant differences between the groups among the relationships can then be tested by constraining specific parameters to be equal across the groups. A significant difference in the chi-square value between this constrained model and one in which the parameters are freely estimated indicates that the nonconstrained model provides a better fit to the data and that a significant difference exists between the groups. Because earlier results showed several significant differences for the two age groups as well as between the males and females, two sets of multi-sample analyses were conducted.

5.7.1. Data Imputation

Before these tests could be performed, it was necessary to first obtain imputed covariance matrices for each of these four groups using the EM algorithm. These four new imputed covariance matrices were used as input for a series of equality tests across the groups. The first step tested the hypothesis that the covariance matrices for the two groups (e.g., younger and older age groups) were equal. Next, we tested the hypothesis of equal mean vectors of the observed variables for the two comparison groups. In the third step, the multivariate growth curve model (Model C-revised from above) was estimated for each group and the hypothesis that the path coefficients in the model are equal in the two groups was tested. This hypothesis was tested by proceeding in two steps. In the first step, a model was estimated that constrained all paths to be equal for the two groups. The fit of this constrained model was then compared to that of a stacked model in which all of the paths between constructs were freely estimated. A significant drop in $\chi^2$ from the constrained
model to the stacked model indicates that the paths differ in the two groups. The final test of group invariance involved the hypothesis that the mean levels of the latent growth parameters (intercepts and slopes) were the same across groups. This test is carried out using the same logic as for the path coefficients—comparing a model with the alpha vector constrained to be equal to a model in which the alpha vector is freely estimated in each group.

5.7.2. Gender Differences

The first step involving the comparison of males and females tested the hypothesis that the covariance matrices for the two groups were the same. The likelihood ratio test statistic for this test was $\chi^2(435) = 688.35, p < .001$. Thus, the hypothesis that the two covariance matrices are equal was rejected. Next, the hypothesis of equal mean vectors of observed variables for boys and girls was tested. This result was $\chi^2(464) = 840.30, p < .001$, so this hypothesis was also rejected.

The next step in the gender multisample analysis consisted of a test of equal path coefficients in the associative latent growth curve model. Freeing all paths in the two models resulted in a significant drop in chi-square ($\Delta\chi^2[12] = 29.90, p = .003$). Figure 7 displays the results of the final model stacked on gender. The unstandardized coefficients are presented in this figure because of the earlier finding that the male and female covariance matrices were not equal. In such situations (where samples differ in their variabilities), it is recommended that unstandardized coefficients be used because standardized solutions have a standard deviation-unit metric (Kline, 1998).

Examination of Figure 7 reveals two especially interesting differences between the male and female groups. The path from the parent support intercept to the parent influence slope was significant (and negative) only among the male subsample while the parent support
slope to parent influence slope path was significant (and positive) only among the females. Thus, males who reported lower levels of parent support at Time 1 were more likely to also report increasing levels of parent influence across the six waves of data. There was no relation between parent support at Time 1 and changes in parent influence among the females. In contrast, changing levels of parental support were related to changes in parental influence only among the females. Another interesting difference between the male and female participants concerns the path from the friend influence slope to substance use slope. For males, this path approached significance ($t = 1.89, p = .06$); thus, males who reported an increase in friend influence over time were more likely to also report increasing rates of substance use. However, there was virtually no relationship between these two constructs among the females.

Finally, the hypothesis that mean levels of the latent growth parameters were the same for both boys and girls was tested. The test of equality of the alpha vectors resulted in a significant change in chi-square ($\Delta \chi^2 [13] = 134.74, p < .001$), indicating that the mean levels of the latent growth parameters (intercept and slope) were different for the two genders (see Table 21). As seen in Table 21, there are several interesting differences between the male and female groups in regard to the parameters of growth, most involving the mean slope parameters. For instance, males reported significant decreases in parental support ($t = -3.86$) and increases in substance use ($t = 5.93$) across time. Females, however, reported significant decreasing rates of friend support ($t = -3.50$) and parent influence ($t = -2.57$). Though females also reported significant increases in their rates of substance use across time ($t = 3.45$), this rate of increase appeared to be less among females than among males.
Table 21
Means and Variances of Growth Parameters for Males and Females

<table>
<thead>
<tr>
<th></th>
<th>Intercept</th>
<th>Slope</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M_i$</td>
<td>$t$</td>
</tr>
<tr>
<td>Parent Support</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>2.99</td>
<td>72.10</td>
</tr>
<tr>
<td>Females</td>
<td>3.12</td>
<td>68.11</td>
</tr>
<tr>
<td>Friend Support</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>2.81</td>
<td>68.62</td>
</tr>
<tr>
<td>Females</td>
<td>3.39</td>
<td>102.82</td>
</tr>
<tr>
<td>Parent Influence</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>-.10</td>
<td>-.14</td>
</tr>
<tr>
<td>Females</td>
<td>1.61</td>
<td>3.27</td>
</tr>
<tr>
<td>Friend Influence</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>1.12</td>
<td>1.99</td>
</tr>
<tr>
<td>Females</td>
<td>1.75</td>
<td>2.30</td>
</tr>
<tr>
<td>Substance Use</td>
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<td></td>
</tr>
<tr>
<td>Males</td>
<td>3.95</td>
<td>5.66</td>
</tr>
<tr>
<td>Females</td>
<td>3.82</td>
<td>5.39</td>
</tr>
</tbody>
</table>
Figure 7. Final model stacked on gender. Values represent unstandardized coefficients ($t$-values in parentheses) for the two groups; males are above the line.
5.7.3. Age Differences

Differences between the younger and older age groups were tested in a parallel fashion to the gender differences above. The likelihood ratio test statistic for the test of equal covariance matrices for the two age groups was \( \chi^2 (435) = 790.03, p < .001 \), which indicated that the covariance matrices were not equal. The hypothesis of equal mean vectors of the observed variables for the ages was also rejected, \( \chi^2 (464) = 1168.82, p < .001 \). Therefore, the reported means of the observed variables were different for the younger and older age groups.

The hypothesis that the path coefficients in the model were the same for both ages was tested next and resulted in a nonsignificant difference, \( \chi^2 (12) = 14.34, p = .28 \). This indicated that the relationships among the growth parameters were not significantly different between younger and older participants. For example, in both groups the paths from the parent and friend support intercepts significantly predicted the respective intercepts of parental and friend influence, while the paths from the support intercepts to the influence slopes were not significant. However, testing the hypothesis of equal mean levels of latent growth parameters resulted in a significant change in chi-square \( (\Delta \chi^2 [13] = 27.07, p = .012) \). Therefore, these results suggested that the mean levels of the latent growth parameters (intercepts and slopes) were different for the two groups. Table 22 displays the mean levels of the growth parameters for younger and older groups. As seen in Table 22, the most striking difference between the two groups concerned the mean level of change in parent support. Younger adolescents reported a significant (and negative) rate of change in parent support \( (t = -2.76) \) whereas the rate of change in parent support was not significant among the older sample.
Table 22
Means and Variances of Growth Parameters for Younger and Older Age Groups

<table>
<thead>
<tr>
<th></th>
<th>Intercept</th>
<th></th>
<th></th>
<th>Slope</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M_i$</td>
<td>$t$</td>
<td>$D_i$</td>
<td>$t$</td>
<td>$M_s$</td>
<td>$D_s$</td>
</tr>
<tr>
<td>Parent Support</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Younger</td>
<td>3.06</td>
<td>70.95</td>
<td>.33</td>
<td>7.11</td>
<td>-0.06</td>
<td>-2.76</td>
</tr>
<tr>
<td>Older</td>
<td>3.14</td>
<td>69.09</td>
<td>.37</td>
<td>7.15</td>
<td>0.00</td>
<td>0.07</td>
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<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Younger</td>
<td>3.03</td>
<td>70.78</td>
<td>.36</td>
<td>7.05</td>
<td>-0.03</td>
<td>-1.32</td>
</tr>
<tr>
<td>Older</td>
<td>3.15</td>
<td>82.26</td>
<td>.23</td>
<td>5.68</td>
<td>0.01</td>
<td>0.41</td>
</tr>
<tr>
<td>Parent Influence</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Younger</td>
<td>0.86</td>
<td>1.47</td>
<td>.66</td>
<td>3.45</td>
<td>-0.14</td>
<td>-0.93</td>
</tr>
<tr>
<td>Older</td>
<td>0.88</td>
<td>1.37</td>
<td>1.58</td>
<td>6.51</td>
<td>-0.08</td>
<td>-0.51</td>
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CHAPTER 6. DISCUSSION AND CONCLUSIONS

6.1. Data Summarization

This study utilized latent growth curve methodology to test hypotheses concerning
the relative importance of parental and peer influences on adolescent substance use among a
panel of adolescents followed across a six-year time span. At Time 1, the younger age group
was, on average, 13 years old and the older group was 15 years old. Drawing from the social
convoy model of social support, an associative latent growth curve model was estimated.
This model hypothesized that parameters (intercepts and slopes) of social support would
predict growth parameters of interpersonal influence, which were hypothesized to have
independent and opposite effects on initial levels of and changes in adolescent substance use.

Results indicated that the reported use of alcohol, cigarettes, and marijuana among
these adolescents increased across the six years, in ways similar to those found in national
surveys, which show that the reported use of substances increases across this age period
(Johnston et al., 2003). Contrary to many published studies, however, the current study did
not find evidence that patterns of use for alcohol, cigarettes, and marijuana were similar.
While consistently modest correlations at each wave between alcohol and cigarette use were
found, only weak correlations existed between marijuana use and the other two substances.

Separate univariate growth curve models for each of the three substances also
indicated that the initial levels of reported marijuana use were lower than the initial levels of
either alcohol or cigarettes and that on average, use of marijuana increased at a lower rate
than either alcohol and cigarette use. In contrast, the initial levels and rates of change for
reported alcohol and cigarette use were quite similar. Further evidence that the patterns of
marijuana use were distinct from alcohol and cigarette use was found when first and second-
order latent growth curve models were estimated. All of these models resulted in less than adequate fit to the observed data, suggesting that a 3-factor higher order common substance use factor was not appropriate for this sample. Because of these results, a combined index of only alcohol and cigarette use was deemed appropriate for this sample.

6.2. Attrition Analyses

The general approach to missing values in many multivariate analyses is to use listwise deletion. This approach eliminates cases that have missing data for any of the variables included in the analyses, resulting in a truncated sample of only those with complete data for all variables. This approach has obvious limitations, however, including the loss of potential information from the excluded cases and the likelihood that the parameter estimates of resulting analyses may not be efficient.

Several steps were taken in the current study to determine if participants who stayed in the panel for the complete six waves differed from attriters. In general, complete data were more likely to be found among females as well as for participants whose fathers reported higher levels of education. These results suggested that the data could not be considered MCAR, though not ruling out the possibility that the missing pattern was MAR, meaning ignorable, or non-ignorable. The results of a model-based approach confirmed that the hypothesis of MAR was tenable and therefore, the possible differences can be ignored. Specifically, the fit of unrestricted \( H_1 \) models for both alcohol and cigarette use were quite good, suggesting that the means, variances, and covariances of the observed variables did not differ among the missing data groups. As such, it was possible to conclude that the six different attrition groups did not significantly differ in terms of the variables used in the study and that corresponding parameters of growth should be equal across the groups.
Therefore, in order to make use of all available information in obtaining correct maximum likelihood estimates of the latent variable model, the EM algorithm was used to obtain imputed estimates of missing values. This resulted in an imputed covariance matrix that took full advantage of all the observed data. The imputed covariance matrix was used in the subsequent tests of the study’s hypotheses.

6.3. Hypothesis Testing

6.3.1. Hypothesis 1: Decreases In Parental Support

The first hypothesis stated that the level of parental support would decrease across the adolescent period. Fitting a two-factor LGC to the observed values of parent support at the first three waves of data collection resulted in a less than ideal fit. All of the goodness of fit indices (NFI, NNFI, and CFI) were less than the .90 levels commonly used to indicate a good fit of a model to the observed data. With only one degree of freedom, it is not possible to respecify this model to obtain a better fit to the observed data (e.g., by estimating correlations among the error terms). As such, the results of the maximum likelihood estimates for the growth parameters should be viewed with caution. For instance, the estimated value for the mean rate of change in parental support was significant and negative ($M_r = -0.05, t = -2.19$). Thus, some support for this hypothesis was found—as a whole, adolescents reported a decline in parental support across the first three waves of the study.

This result, therefore, offers tentative support for one of the assumptions of the social convoy model, that as children move into adolescence, the structure of their social convoy networks also changes. The finding that parental support decreased across the first three waves of data collection suggests that adolescents relied on parents as primary sources of support less and less during this time. The finding that the variance of the slope factor for
parental support was not significant further strengthens the notion that parental support decreases during this time. That is, the conclusion that parental support decreases across adolescence seems to apply to most of the adolescents in this sample.

6.3.2. Hypothesis 2: Increases In Friend Support

Conversely, it was expected that as the parents' placement in the adolescents' social support network decreased in importance, inclusion of friends in the social convoy would increase. Fitting a second two-factor LGC to the repeated measures of friend support tested this hypothesis. Although the two-factor LGC of friend support provided a good fit to the observed data (e.g., CFI = .99), the estimated value of the mean slope parameter was not significant in this sample ($M_e = -.02$, $t = -.74$). This result implies that there was no significant change in the amount of support the adolescents reported from friends during this time. Therefore, no evidence was found for the hypothesis that the amount of support from friends increases across adolescence. This contrasts with the assumption of the social convoy model that friends may play an increasing role in providing support to adolescents during this time of transition.

6.3.3. Hypothesis 3: Decreases In Parent Influence

Research has shown that although the level of parental influence on adolescents' decision-making processes exceeds that of peer influence in early adolescence, by mid- to late-adolescence, the two sources of interpersonal influence are similar in level. Therefore, it was hypothesized that the level of parental influence on adolescents' decisions regarding the use of alcohol and cigarettes would decrease across the six waves of the study. This hypothesis was tested by fitting a two-factor LGC to the observed data for parental influence to use substances. The fit of this model was quite good as evidenced by the fit indices, which
were all greater than .90. Furthermore, the estimated rate of change in parental influence in this LGC model was significant and negative ($M_3 = -0.20, t = -7.47$), offering support for the hypothesis that the level of adolescents’ reports of parental influence on adolescents’ substance use decisions decreased across the adolescent period.

This finding is also consistent with the assumption of the social convoy model that social networks evolve during adolescence. It seems that as the position of parents in the adolescents’ inner circle of support changes (as seen in hypothesis 1 above), a corresponding decrease in parental influence on adolescents’ substance use decisions also occurs during this time. This conclusion, however, must also be interpreted in light of two other findings from the latent growth curve model. First, significant variation was found to exist among the adolescents in their individual trajectories of parental influence. This suggests that not all of the adolescents reported the same decrease in parental influence across the six waves of data collection. The significant inverse relation between the initial level and slope of parental influence ($Cov_{ls} = -0.18, t = -3.04$) suggests that the rate of decrease was due at least in part to the adolescents’ initial levels of parental influence. Adolescents who reported higher levels of parental influence at Time 1 were more likely to also report less decline in parental influence.

6.3.4. Hypothesis 4: Increases In Friend Influence

In a similar fashion, the hypothesis that the level of friend influence would increase across adolescence was tested by fitting a two-factor LGC model to the observed data for friend influence to use substances (waves 1 – 6). Again, the goodness of fit indices provided evidence that the fit of this model was good (e.g., CFI = .95). The estimates of growth parameters in this model resulted in a significant and negative value for the estimated rate of
change in friend influence across time ($M_t = -0.12, t = -4.70$). This result does not support the hypothesis of increasing friend influence and instead suggests that like parental influence, the influence of friends on adolescents' substance use decisions also decreased during adolescence.

However, like parental influence, significant variation in this decrease in friend influence was found among the adolescents ($D_t = -0.07, t = -4.26$). This suggests that adolescents differed in their individual rates of change in friend influence. The significant negative relation between the initial level and slope ($Cov'I = -0.12, t = -2.41$) indicates that adolescents in this sample who reported the highest initial levels of friend influence also reported the smallest rates of decrease in friend influence.

It is also important to note that the estimated rate of decrease in friend influence was less than that of the decrease in parental influence (−.12 vs. −.20), and that friend influence started at a lower initial level (3.49) than parental influence (4.54). These findings imply that when the adolescents in this sample were between 13-15 years old, the influence of parents on their substance use-related decisions exceeded that of their friends. Furthermore, both sources of influence were found to decrease over time. However, because parental influence declined at a greater rate than friend influence, the relative influence of parents and friends on older adolescents' (ages 19-21) substance use decisions are similar. This finding is consistent with the assumption that as adolescents age, their social convoy networks expands. By late adolescence, it appears that adolescents in this sample relied equally on their parents and friends to guide their decisions relating to alcohol and cigarette use.
6.3.5. Hypothesis 5: Support Parameters Predict Influence Parameters

The remaining hypotheses were tested after first conducting a hierarchical nested-models approach to establish mediation. Specifically, these hypotheses stated that parental and peer support would have opposite effects on adolescent substance use, and that these relations would be mediated by parent and peer influence on adolescents' decision making, respectively. The models were tested in a hierarchical manner, adding parameters to each subsequent nested model. In all instances, the difference in chi-square values between the two models was significant, which suggested that the less restricted models were more accurate representations of the data.

An important exception to this pattern, however, was found when the least restricted model was “trimmed” to reflect the hypothesized parameters in the model of interest by constraining the direct paths from social support parameters to the substance use parameters to zero. In this case, the chi-square difference was not significant, which indicated that the more restricted model was preferable to the model that estimated direct paths from the social support parameters to parameters of adolescent substance use. This suggests that the effects of social support were mediated through the growth parameters of social influence.

Furthermore, the final model showed that for both parents and friends, high initial levels of support predicted high initial levels of social influence, as hypothesized. There was also support for the hypothesized path from the slope of parental support to the slope of parental influence. The positive value of this path indicated that as adolescents reported decreasing levels of parent support during the first three years of the study, their level of reported parental influence on their substance use decisions also decreased across the six-year time span. It is important to note that the hypothesized path between friend support and
friend influence was not significant. Thus, changes in friend support over the first three waves did not predict changes in friend influence over time. The other hypothesized paths from support to influence were also non-significant (i.e., from parent support intercept $\rightarrow$ parent influence slope, friend support intercept $\rightarrow$ friend influence slope). Thus, only partial support was found for the hypothesis that growth parameters of social support would predict growth parameters of interpersonal influence on adolescents' substance use decisions. In particular, these results suggest that these relationships between social support and social influence parameters are more likely to exist for parents rather than friends.

These results need to be interpreted in conjunction with the earlier findings that showed that, although significant decreases in parental support and influence were found, overall levels of friend support and influence remained the same across time for this sample. The social convoy model predicts that as individuals move into and out of the adolescents' social support network, their relative influence changes accordingly. The results of the present study indicate that this assumption seems to hold for parental support and influence. However, because the adolescents in this sample did not report significant changes in the levels of either friend support or friend influence, the assumption that friends become increasingly important to adolescents' social support networks may not be valid, at least between the ages of 13 and 19 years.

It may be the case, however, that friends have already taken their place in the adolescents' social networks by the age of 13 (Time 1 in this study) and they simply retain this position across the next several years while parental influence continues to drop. Examining the means of the initial levels of parent support and influence from the univariate models and comparing them to the means of the initial levels of friend support and influence
support this conclusion. At Time 1, the initial levels of parent and friend support were nearly identical (3.05 vs. 3.10); however initial levels of parent influence are higher than initial levels of friend influence (4.54 vs. 3.49). These results suggest that although changes in the adolescents’ social support networks have already taken place by the age of 13, with parents and friends equally represented, the relative influence of parents and friends on adolescents’ substance use decisions changes as they move across the adolescent period.

6.3.6. Hypothesis 6: Parental Influence Negatively Related To Use

Research has shown that adolescents who report high support from parents tend to report lower levels of risk behaviors such as substance use. Parental support, therefore, can be considered a protective factor against adolescent substance use. On the other hand, high levels of support from friends has been linked to higher rates of adolescent risk behaviors. Thus, peer support can be viewed as a risk factor for adolescent substance use. The current study hypothesized that these opposing forces (parental vs. peer support) would be mediated through the adolescents’ self-reports of the extent to which each source of support influenced their decisions to use alcohol and cigarettes.

The next hypothesis tested, therefore, was that an inverse relationship existed between the growth parameters of parental influence and the initial level and rate of change in adolescent substance use. This hypothesis was supported: both paths from parent influence parameters (initial level and slope) were negatively related to the respective parameters of use (initial level and slope). Thus, adolescents with the highest levels of parent influence at Time 1 reported the lowest initial levels of alcohol and cigarette use. Furthermore, as the reported level of parental influence decreased, adolescents, on average, reported an increase in substance use over time.
6.3.7. Hypothesis 7: Friend Influence Positively Related To Use

Only partial support was found for the hypothesis that growth parameters of friend influence would be positively related to the growth parameters of adolescent substance use. The results suggested that adolescents with high initial levels of friend influence tended to also report high initial levels of alcohol and cigarette use. However, no relationship was found between the parameters of change for friend influence and adolescent substance use. Again, these results need to be interpreted in conjunction with the earlier findings that showed that levels of friend support and influence remained the same across time for this sample, with no significant individual differences among the sample. Because the average rate of change in friend influence was not significant, it is not surprising that this growth parameter was not a significant predictor in the multivariate model. That is, because there was no significant variation in the predictor variable, it follows that it is not likely to predict other variables.

6.4. Multi-Sample Analyses

Although no specific hypotheses were made about gender or age differences, several noteworthy results were found. First, at the descriptive level, female adolescents reported higher levels of support from both parents and friends when compared to male adolescents, and older adolescents reported higher levels of friend support compared to their younger counterparts. Multi-sample analyses using SEM with mean structures also indicated that the covariance matrices for the males and females differed, as did the matrices for the two age groups, as seen in the significant chi-square values when simultaneous models were tested. Additionally, mean levels of observed variables were different for the two genders and two ages.
Relations among the growth parameters in the overall model were found to be different only for the two genders; no difference was found for the relations among the constructs for the age groups. Comparison of the estimated paths in the model stacked on gender indicated several differences for the relations among constructs for the two groups. For example, the path from the parent support slope to the parent influence slope was significant only among the female participants, whereas the path from the initial level of parent support to change in parent influence was significant only for males.

There were also several differences in the mean levels of growth parameters between both sets of groups. For example, while the rate of change in parental support was stronger for males compared to females, the female adolescents reported higher initial levels of parental influence as well as a more significant decrease in parental influence. Males were also more likely to report a greater increase in substance use across the six waves of data collection, even though the two genders reported similar levels of use at Time 1. The stacked model comparisons also indicated that the younger age group reported a more significant decline in parental support than the older group. This suggests that parental support may “bottom out” sometime around mid-adolescence (Time 1 for the older age group) and not show the same decline during the ages of 15 to 17 as it does from ages 13 to 15.

6.5. General Conclusions

6.5.1. The Social Convoy Model

The present study tested the assumptions of the social convoy of social support model in the framework of latent growth curve methodology. Primary among these assumptions is that changes occur in individuals’ social networks (social convoys) as they move through normative life transitions such as during adolescence. More specifically, according to the
social convoy model, there is both continuity and change in the individual’s innermost
network of support as certain providers of support move into the innermost circle while other
members may remain. The adolescents in this sample reported a significant decrease in the
level of support from parents during the first three years of the study. At the same time, no
changes were seen in the reported levels of support from friends. Thus, it does appear that
both continuity and change were present in the adolescents’ social networks. As mentioned
above, further research with younger children is needed to determine if the movement of
friends into the adolescents’ social networks occurs prior to the age of thirteen, however.

The social convoy model also posits that the relative influence of significant others is
determined by their placement within the convoy structure. Thus, the latent growth curve
tested in this study incorporated growth parameters of social support, interpersonal influence,
and adolescent substance use into a single model that allowed for the simultaneous
estimation of parental and peer influences on adolescent substance use. This model was
found to provide a good fit to the observed data and provided evidence that parental and peer
influences act in an opposing manner on adolescents’ use of alcohol and cigarettes.
Adolescents who reported that they received high levels of support from their parents tended
to also report higher levels of parental influence in their decisions related to substance use,
which in turn, was negatively related to initial levels and decreasing rates of adolescent
substance use. Thus, having parents in one’s inner circle of support provided a buffer to these
adolescents.

Conversely, adolescents who reported high initial levels of friend support were more
likely to also report high initial levels of influence from friends, which increased the
likelihood that they reported use of alcohol and cigarettes at the first wave. Contrary to
hypotheses, changes in levels of friend support and influence were not predictive of changes in adolescent substance use. The implication of this finding is not clear, though, as mentioned earlier, because the results of univariate growth curve models indicated no significant changes in these parameters over time, it is not surprising that the slope parameters did not play significant roles as predictors of other outcomes. Again, further studies are warranted that would explore these hypotheses among a younger sample and perhaps detect changes in the levels of friend support and influence as youngsters move from childhood into early adolescence.

6.5.2. The Relative Influence Of Parents And Peers

This study represented a new approach to studying the relative influence of parents and friends on adolescents' substance use behaviors. The social convoy model represented an ecological approach to studying this controversial issue and thus distinguished this study from earlier research in this area. Drawing on the assumptions of the ecological systems theory (Bronfenbrenner, 1989), the social convoy model explicitly acknowledges that individuals have agency in choosing their social environments. Thus, adolescents' are expected to actively participate in determining the nature of their social support convoys. Furthermore, the social convoy model offers an advantage over traditional approaches to studying the relative influence of parents and peers because it allows one to form hypotheses about why some relationships are more influential than others. These hypotheses were tested in the multivariate model by estimating a series of hierarchically related models.

The results of the final model in the series of nested models provided evidence that the effect of support adolescents reported receiving from parents and friends on their use of alcohol and cigarettes was indeed mediated through adolescents' indications of how
influential each of these two sources of support were in their substance use decisions. In each case, significant paths were found between the intercepts of the support and influence parameters as well as between the parental support slope parameter and the parental influence slope parameter. Furthermore, significant indirect effects on initial levels of adolescent substance use were found for initial levels of each source of support, though of varying magnitude and in opposite directions. These results suggest that high levels of parental support predicted low levels of adolescent substance use while high levels of friend support were associated with high levels of adolescent use.

Similar patterns were found between the influence and use growth parameters and allow for conclusions to be drawn regarding the relative influence of parents and friends on adolescents' patterns of alcohol and cigarette use. First, both of the interpersonal influence intercept parameters were significant predictors of the initial level of alcohol and cigarette use, though in opposite directions. Thus, as expected, level of parental influence was a protective factor, decreasing the likelihood of adolescents' substance use, while peer influence was a risk factor that increased the likelihood of adolescent substance use.

Comparison of the standardized maximum likelihood estimates of these paths suggests that the absolute magnitude of the path from the initial level of parent influence was nearly twice that of the path from initial levels of friend influence to adolescent substance use at Time 1. Thus, at early ages, the relative influence of parents on adolescents' substance use was significantly higher than that of friends. Furthermore, parents continued to act as a protective buffer against changes in adolescent substance use across the adolescent period as seen in the significant negative relation between the parent influence slope and rate of change in adolescent substance use. This negative relation implies that as parental influence
increased between the ages of 14 and 19, adolescent substance use decreased during the same
time. The nonsignificance of the path from the corresponding path from change in friend
influence to change in adolescent substance use strengthens the conclusion that not only is
parental influence stronger than peer influence at early ages, but that this difference remains
throughout the adolescent period.

These results are consistent with findings from other researchers who have examined
the differential effects of family and peers from a social support perspective (e.g., Barrera &
Li, 1996; Wills et al., 1996). The present study extends these findings by incorporating a
multivariate latent growth model to study hypotheses derived from a recently developed
model of social support, the social convoy model. Previous research has been limited by
studying these relationships by using more traditional methods and has often relied on cross-
sectional study designs. The current study, however, examined these relations among a panel
of adolescents followed over a time of six years and employed a superior method of studying
developmental change, latent growth curve modeling.

6.2. Limitations

Despite these advances, the current study was not without its limitations. First among
these is the possibility that the sample was not representative of adolescents as a whole. All
of the participants were from rural areas in Iowa and nearly all participants were white.
Further research with samples from different types of environments (e.g., urban or suburban
areas) as well as with different races and ethnicities is needed before generalizations can be
made about the results of the study.

Another limitation of the study is that parental and friend support were only measured
at the first three waves of the study. Thus, the research concerned an incomplete picture of
developmental changes in the convoy of social support. It is possible that extending these measures to either earlier or later periods of individual development may result in different patterns of social support.

It is also important to note that although established scales were used to measure parental and friend support, this operationalization of social support was different from how social support has been operationalized in other research with the social convoy model (e.g., Levitt et al., 1993). In these studies, the convoy mapping procedure (Antonucci, 1986) was used to measure convoy structure and function. Finally, single indicators of parental and friend influence to use alcohol and cigarettes at each wave were used in the current study. Thus the measures of social influence were based solely on the adolescents' self-reports of the extent to which parents and peers influenced their alcohol and smoking decisions. More reliable measures of these interpersonal influences may be found with multiple indicators at each wave.
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APPENDIX I.
EQS PROGRAM FOR ASSOCIATIVE MODEL

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APPENDIX II.
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v13 = mar_t1; v14 = mar_t2; v15 = mar_t3; v16 = mar_t5; v17 = mar_t6; 
f1 = alc_int; f2 = alc_slp; 
f3 = smk_int; f4 = smk_slp; 
f5 = mar_int; f6 = mar_slp; 
f7 = com_int; f8 = com_slp;
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v2 = f1 + f2 + e2;
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v4 = f1 + 3f2 + e4;
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v6 = f1 + 5f2 + e6;
v7 = f3 + 0f4 + e7;
v8 = f3 + 1f4 + e8;
v9 = f3 + 2f4 + e9;
v10 = f3 + 3f4 + e10;
v11 = f3 + 4f4 + e11;
v12 = f3 + 5f4 + e12;
v13 = f5 + 0f6 + e13;
v14 = f5 + 1f6 + e14;
v15 = f5 + 2f6 + e15;
v16 = f5 + 4f6 + e16;
v17 = f5 + 5f6 + e17;
f1 = 0v999 + f7 + d1;
f2 = 0v999 + f8 + d2;
f3 = 0*v999 + *f7 + d3;
f4 = 0*v999 + *f8 + d4;
f5 = 0*v999 + *f7 + d5;
f6 = 0*v999 + *f8 + d6;
f7 = 0*v999 + d7;
f8 = 0*v999 + d8;
/variances
e1 to e17 = *; d1 to d6 = *; 
d7 = *; d8 = *;
covariances
d1 to d6 = 0; d7 to d8 = *;
/constraints
(f3, f7) = (f4, f8); 
(f5, f7) = (f6, f8);
```
0.756
0.607  1.165
0.460  0.803  1.302
0.392  0.735  0.886  2.020
0.330  0.574  0.693  1.355  1.615
0.199  0.308  0.423  0.895  1.013  1.387
0.410  0.389  0.397  0.220  0.193  0.132
0.429  0.655  0.561  0.423  0.338  0.225
0.462  0.800  0.897  0.724  0.542  0.331
0.503  0.746  0.867  1.139  0.817  0.485
0.409  0.697  0.786  1.119  1.026  0.662
0.349  0.622  0.716  0.991  0.922  0.718
-0.001  -0.003  -0.003  -0.005  -0.003  -0.001
0.003  0.036  0.036  0.022  0.030  0.024
0.035  0.123  0.159  0.090  0.083  0.067
0.123  0.231  0.315  0.411  0.344  0.225
0.097  0.216  0.259  0.381  0.446  0.447
0.633
0.585  1.064
0.601  1.016  1.541
0.549  0.891  1.281  1.876
0.421  0.808  1.231  1.609  2.055
0.391  0.767  1.148  1.487  1.784  2.109
0.005  0.004  0.003  0.002  -0.002  -0.002
0.014  0.041  0.047  0.044  0.043  0.036
0.066  0.129  0.182  0.155  0.164  0.128
0.089  0.236  0.369  0.509  0.557  0.486
0.105  0.247  0.287  0.437  0.581  0.580
0.003
0.003  0.030
0.000  0.035  0.181
-0.001  0.042  0.113  0.713
-0.002  0.040  0.123  0.546  1.110

/means
1.443  1.763  1.890  2.630  3.010  3.410
1.387  1.690  1.977  2.283  2.457  2.733
1.003  1.023  1.080  1.340  1.557

/sta
.869  1.079  1.142  1.421  1.271  1.1775
.795  1.032  1.241  1.370  1.434  1.4523
.058  0.172  0.425  0.845  1.054

/print
/end
```
APPENDIX III.
EQS PROGRAM FOR CURVE-OF-FACTORS MODEL

/TITLE
curve of factors model (listwise deletion)
/specifications
var = 17; cases=300; me=ml; anal=mom; ma=cov; fi=6;
/labels
v1=alc_t1; v2=alc_t2; v3=alc_t3; v4=alc_t4; v5=alc_t5; v6=alc_t6;
v7=smk_t1; v8=smk_t2; v9=smk_t3; v10=smk_t4; v11=smk_t5; v12=smk_t6;
v13=mar_t1; v14=mar_t2; v15=mar_t3; v16=mar_t5; v17=mar_t6;
f1=1factor; f2=2factor;
f3=3factor; f4=4factor;
f5=5factor; f6=6factor;
f7=com_int; f8=com_slp;
/equations
v1=*v999++f1+e1;
v2=*v999++f2+e2;
v3=*v999++f3+e3;
v4=*v999++f4+e4;
v5=*v999++f5+e5;
v6=*v999++f6+e6;
v7=0v999+f1+e7;
v8=0v999+f2+e8;
v9=0v999+f3+e9;
v10=0v999+f4+e10;
v11=0v999+f5+e11;
v12=0v999+f6+e12;
v13=0v999+f7+e13;
v14=0v999+f2+e14;
v15=0v999+f3+e15;
v16=0v999+f5+e16;
v17=0v999+f6+e17;
f1=f7+0f8+d1;
f2=f7+1f8+d2;
f3=f7+2f8+d3;
f4=f7+3f8+d4;
f5=f7+4f8+d5;
f6=f7+5f8+d6;
f7=0*v999+d7;
f8=0*v999+d8;
/variances
e1 to e17 = *;
d1 to d6=0*;
d7=*; d8=*;
/covariances
e1 to e17=0;
e1 to e6=*
e7 to e12=*
e13 to e17=*
d1 to d6=0; d7 to d8=*
/constraints
(v1,f1)=(v2,f2)=(v3,f3)=(v4,f4)=(v5,f5)=(v6,f6);
(v13,f1)=(v14,f2)=(v15,f3)=(v16,f5)=(v17,f6);
/matrix
0.756
0.607 1.165
0.460 0.803 1.302
0.392 0.735 0.886 2.020
0.330 0.574 0.693 1.355 1.615
0.199 0.308 0.423 0.895 1.013 1.387
0.410 0.389 0.397 0.220 0.193 0.132
0.429 0.655 0.561 0.423 0.338 0.255
0.462 0.800 0.897 0.724 0.542 0.331
0.503 0.746 0.867 1.139 0.817 0.485
0.409 0.697 0.786 1.119 1.026 0.662
0.349 0.622 0.716 0.991 0.922 0.718
-0.001 -0.003 -0.003 -0.005 -0.003 -0.001
0.003 0.036 0.036 0.022 0.030 0.024
0.035 0.123 0.159 0.090 0.083 0.067
0.123 0.231 0.315 0.411 0.344 0.225
0.097 0.216 0.259 0.381 0.446 0.447
0.633
0.585 1.064
0.601 1.016 1.541
0.549 0.891 1.281 1.876
0.421 0.808 1.231 1.609 2.055
0.391 0.767 1.148 1.487 1.784 2.109
0.005 0.004 0.003 0.002 -0.002 -0.002
0.014 0.041 0.047 0.044 0.043 0.036
0.066 0.129 0.182 0.155 0.164 0.128
0.089 0.236 0.369 0.509 0.557 0.486
0.105 0.247 0.287 0.437 0.581 0.580
0.003
0.003 0.030
0.000 0.035 0.181
-0.001 0.042 0.113 0.713
-0.002 0.040 0.123 0.546 1.110
/means
1.443 1.763 1.890 2.630 3.010 3.410
1.387 1.690 1.977 2.283 2.457 2.733
1.003 1.023 1.080 1.340 1.557
/sta
.869 1.079 1.142 1.421 1.271 1.1775
.795 1.032 1.241 1.370 1.434 1.4523
.058 0.172 0.425 0.845 1.054
/print
/end
## APPENDIX IV.
### COVARIANCE MATRICES FOR ATTRITION GROUPS

<table>
<thead>
<tr>
<th>Alcohol Use</th>
<th>Group 1 (N = 314)</th>
<th>Group 2 (N = 44)</th>
</tr>
</thead>
<tbody>
<tr>
<td>.785</td>
<td>.626 1.166</td>
<td>.681 1.004</td>
</tr>
<tr>
<td>.640</td>
<td>.460 .812 1.347</td>
<td>.926 .918 1.684</td>
</tr>
<tr>
<td>.401</td>
<td>.401 .729 .897 2.004</td>
<td>.613 .612 .695 2.302</td>
</tr>
<tr>
<td>.313</td>
<td>.313 .545 .692 1.341 1.619</td>
<td>.275 .342 .469 1.387 1.911</td>
</tr>
<tr>
<td>.197</td>
<td>.197 .293 .424 .897 1.029 1.399</td>
<td>.000 .000 .000 .000 .000 1.000</td>
</tr>
<tr>
<td><strong>Means</strong></td>
<td><strong>1.452 1.761 1.895 2.631 3.022 3.411</strong></td>
<td><strong>1.636 1.705 2.114 3.023 3.364 .000</strong></td>
</tr>
<tr>
<td><strong>Standard Deviations</strong></td>
<td><strong>.886 1.080 1.160 1.416 1.273 1.183</strong></td>
<td><strong>.990 1.002 1.298 1.517 1.382 .000</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Group 3 (N = 13)</th>
<th>Group 4 (N = 19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>.077</td>
<td>1.228</td>
</tr>
<tr>
<td>.026</td>
<td>.836 .029</td>
</tr>
<tr>
<td>.064</td>
<td>.064 .827 1.526</td>
</tr>
<tr>
<td>.090</td>
<td>.090 .974 .436 2.410</td>
</tr>
<tr>
<td>.000</td>
<td>.000 .000 .000 .000 1.000</td>
</tr>
<tr>
<td><strong>Means</strong></td>
<td><strong>1.077 1.692 2.231 2.923 .000 .000</strong></td>
</tr>
<tr>
<td><strong>Standard Deviations</strong></td>
<td><strong>.277 1.032 1.235 1.553 .000 .000</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Group 5 (N = 11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>.855</td>
</tr>
<tr>
<td>.000 .000 1.000</td>
</tr>
<tr>
<td>.000 .000 .000 1.000</td>
</tr>
<tr>
<td>.000 .000 .000 .000 1.000</td>
</tr>
<tr>
<td><strong>Means</strong></td>
</tr>
<tr>
<td><strong>Standard Deviations</strong></td>
</tr>
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</table>
### Cigarette Use

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (N = 307)</th>
<th></th>
<th>Group 2 (N = 42)</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>.621</td>
<td></td>
<td>.199</td>
<td></td>
</tr>
<tr>
<td></td>
<td>.575</td>
<td>1.048</td>
<td>.659</td>
<td>1.215</td>
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<tr>
<td></td>
<td>.591</td>
<td>1.006</td>
<td>.756</td>
<td>1.089</td>
</tr>
<tr>
<td></td>
<td>.544</td>
<td>1.283</td>
<td>.488</td>
<td>1.106</td>
</tr>
<tr>
<td></td>
<td>.885</td>
<td>1.875</td>
<td>.1.358</td>
<td>2.276</td>
</tr>
<tr>
<td></td>
<td>.420</td>
<td>1.236</td>
<td>.505</td>
<td>1.000</td>
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<tr>
<td></td>
<td>.390</td>
<td>1.613</td>
<td>.829</td>
<td>1.000</td>
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<tr>
<td></td>
<td>.764</td>
<td>2.051</td>
<td>1.000</td>
<td>1.585</td>
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<tr>
<td></td>
<td></td>
<td>2.107</td>
<td>.659</td>
<td>2.355</td>
</tr>
<tr>
<td>Means</td>
<td>1.378</td>
<td>1.681</td>
<td>1.857</td>
<td>2.167</td>
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<tr>
<td>Standard Deviations</td>
<td>.788</td>
<td>1.967</td>
<td>2.333</td>
<td>2.667</td>
</tr>
<tr>
<td></td>
<td>1.024</td>
<td>2.264</td>
<td>2.676</td>
<td>3.286</td>
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<tr>
<td></td>
<td>1.239</td>
<td>2.436</td>
<td>.000</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>1.369</td>
<td>2.713</td>
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<tr>
<td></td>
<td>1.432</td>
<td></td>
<td>.000</td>
<td>.000</td>
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<tr>
<td></td>
<td>1.452</td>
<td></td>
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<td></td>
<td>.000</td>
<td>.000</td>
</tr>
</tbody>
</table>

### Group 3 (N = 12)

|                  | .636              |                  | 1.246            |                  |
|                  | 1.136             | 2.265            | .640             | 1.620            |
|                  | 1.045             | 2.068            | .892             | 1.462            |
|                  | 1.182             | 2.197            | 2.409            | 3.035            |
|                  | .000              | 3.788            | .000             | .000             |
|                  | .000              | .000             | .000             | .000             |
|                  | .000              | .000             | 1.000            | .000             |
|                  | .000              | .000             | .000             | .000             |
|                  | .000              | .000             | 1.000            | .000             |
| Means            | 1.500             | 2.083            | 2.383            | 2.833            |
| Standard Deviations | .798             | 2.250            | .000             | .000             |
|                  | 1.505             | 2.833            | .000             | .000             |
|                  | 1.485             | .000             | .000             | .000             |
|                  | 1.946             | .000             | .000             | .000             |
|                  |                  |                  | .000             | .000             |
|                  |                  |                  | .000             | .000             |
|                  |                  |                  | .000             | .000             |

### Group 4 (N = 19)

|                  | .636              |                  | 1.246            |                  |
|                  | 1.136             | 2.265            | .640             | 1.620            |
|                  | 1.045             | 2.068            | .892             | 1.462            |
|                  | 1.182             | 2.197            | 2.409            | 3.035            |
|                  | .000              | 3.788            | .000             | .000             |
|                  | .000              | .000             | .000             | .000             |
|                  | .000              | .000             | 1.000            | .000             |
|                  | .000              | .000             | .000             | .000             |
|                  | .000              | .000             | 1.000            | .000             |
| Means            | 1.500             | 2.083            | 2.383            | 2.833            |
| Standard Deviations | .798             | 2.250            | .000             | .000             |
|                  | 1.505             | 2.833            | .000             | .000             |
|                  | 1.485             | .000             | .000             | .000             |
|                  | 1.946             | .000             | .000             | .000             |
|                  |                  |                  | .000             | .000             |
|                  |                  |                  | .000             | .000             |
|                  |                  |                  | .000             | .000             |

### Group 5 (N = 11)

|                  | 2.218             |                  | 1.116            |                  |
|                  | 2.164             | 2.473            | 1.273            |                  |
|                  | .000              | 1.000            | 1.742            |                  |
|                  | .000              | .000             | .000             |                  |
|                  | .000              | .000             | .000             |                  |
|                  | .000              | .000             | .000             |                  |
|                  | .000              | .000             | .000             |                  |
| Means            | 2.273             | 2.455            | .000             | .000             |
| Standard Deviations | 1.489             | 1.572            | .000             | .000             |
|                  |                  |                  | .000             | .000             |
|                  |                  |                  | .000             | .000             |
APPENDIX V.
EQUATION PROGRAM FOR H MODEL (ALCOHOL USE)

/TITLE
alcohol missing group 1
/specifications
var = 6; cases=314; me=ml; anal=mom; ma=cov; groups = 5;
/labels
v1=alc_t1; v2=alc_t2; v3=alc_t3; v4=alc_t5; v5=alc_t5; v6=alc_t6;
f1=alc_int; f2=alc_slp;
/equations
v1=\*v999+e1;
v2=\*v999+e2;
v3=\*v999+e3;
v4=\*v999+e4;
v5=\*v999+e5;
v6=\*v999+e6;
/variances
e1 to e6 = *;
/covariances
e1 to e6 = *;
/constraints
/matrix
.785
.626 1.166
.460 .812 1.347
.401 .729 .897 2.004
.313 .545 .692 1.341 1.619
.197 .293 .424 .897 1.029 1.399
/means
1.452 1.761 1.895 2.631 3.022 3.411
/sta
.886 1.080 1.160 1.416 1.273 1.183
/print
/end

/TITLE
alcohol missing group 2
/specifications
var = 6; cases=44; me=ml; anal=mom; ma=cov;
/labels
v1=alc_t1; v2=alc_t2; v3=alc_t3; v4=alc_t5; v5=alc_t5; v6=alc_t6;
f1=alc_int; f2=alc_slp;
/equations
v1=\*v999+e1;
v2=\*v999+e2;
v3=\*v999+e3;
v4=\*v999+e4;
v5=\*v999+e5;
v6=\*v999+e6;
/variances
  e1 to e4 = *;
/covariances
  e1 to e4 = *;
/constraints
/matrix
  .981
  .681 1.004
  .926 .918 1.684
  .613 .612 .695 2.302
  .275 .342 .469 1.387 1.911
  .000 .000 .000 .000 .000 1.000
/means
  1.636 1.705 2.114 3.023 3.364 .000
/sta
  .990 1.002 1.298 1.517 1.382 .000
/print
/end

/TITLE
  alcohol missing group 3
/specifications
  var = 6; cases=13; me=ml; anal=mom; ma=cov;
/labels
  v1=alc_t1; v2=alc_t2; v3=alc_t3; v4=alc_t5; v5=alc_t5; v6=alc_t6;
  f1=alc_int; f2=alc_slp;
/equations
  v1=*v999+e1;
  v2=*v999+e2;
  v3=*v999+e3;
  v4=*v999+e4;
/variances
  e1 to e4 = *;
/covariances
  e1 to e4 = *;
/constraints
/matrix
  .077
  .026 1.064
  .064 .827 1.526
  .090 .974 .436 2.410
  .000 .000 .000 .000 1.000
  .000 .000 .000 .000 .000 1.000
/means
  1.077 1.692 2.231 2.923 .000 .000
/sta
  .277 1.032 1.235 1.553 .000 .000
/print
/end
/TITLE
alcohol missing group 4
/specifications
var = 6; cases=19; me=ml; anal=mom; ma=cov;
/labels
v1=alc_t1; v2=alc_t2; v3=alc_t3; v4=alc_t5; v5=alc_t5; v6=alc_t6;
f1=alc_int; f2=alc_slp;
equations
v1=*v999+el;
v2=*v999+e2;
v3=*v999+e3;
/variances
e1 to e3 = *;
/covariances
e1 to e3 = *;
/constraints
/matrix
1.228
.836 1.029
.904 .924 2.064
.000 .000 .000 1.000
.000 .000 .000 .000 1.000
.000 .000 .000 .000 .000 .000 1.000
/matrix
1.684 1.842 2.211 .000 .000 .000
/sta
1.108 1.015 1.437 .000 .000 .000
/print
/end

/TITLE
alcohol missing group 5
/specifications
var = 6; cases=11; me=ml; anal=mom; ma=cov;
/labels
v1=alc_t1; v2=alc_t2; v3=alc_t3; v4=alc_t5; v5=alc_t5; v6=alc_t6;
f1=alc_int; f2=alc_slp;
equations
v1=*v999+e1;
v2=*v999+e2;
/variances
e1 to e2 = *;
/covariances
e1 to e2 = *;
/constraints
/matrix
.855
1.009 1.818
.000 .000 1.000
.000 .000 .000 1.000
.000 .000 .000 .000 1.000
.000 .000 .000 .000 .000 1.000

/means
1.636 2.273 .000 .000 .000 .000
/sta
.924 1.348 .000 .000 .000 .000
/print

/constraints
(1,v1,v999)=(2,v1,v999)=(3,v1,v999)=(4,v1,v999)=(5,v1,v999);
(1,v2,v999)=(2,v2,v999)=(3,v2,v999)=(4,v2,v999)=(5,v2,v999);
(1,v3,v999)=(2,v3,v999)=(3,v3,v999)=(4,v3,v999);
(1,v4,v999)=(2,v4,v999)=(3,v4,v999);
(1,v5,v999)=(2,v5,v999);
(1,e1,e1)=(2,e1,e1)=(3,e1,e1)=(4,e1,e1)=(5,e1,e1);
(1,e2,e2)=(2,e2,e2)=(3,e2,e2)=(4,e2,e2)=(5,e2,e2);
(1,e3,e3)=(2,e3,e3)=(3,e3,e3)=(4,e3,e3);
(1,e4,e4)=(2,e4,e4);
(1,e5,e5)=(2,e5,e5);
(1,e1,e2)=(2,e1,e2)=(3,e1,e2)=(4,e1,e2)=(5,e1,e2);
(1,e3,e3)=(2,e3,e3)=(3,e3,e3)=(4,e3,e3);
(1,e1,e4)=(2,e1,e4)=(3,e1,e4); 
(1,e5,e5)=(2,e1,e5);
(1,e2,e3)=(2,e2,e3)=(3,e2,e3)=(4,e2,e3); 
(1,e2,e4)=(2,e2,e4)=(3,e2,e4); 
(1,e2,e5)=(2,e2,e5); 
(1,e3,e4)=(2,e3,e4); 
(1,e3,e5)=(2,e3,e5); 
(1,e4,e5)=(2,e4,e5); 

/Imtest
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iter=100;
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APPENDIX VI.
EQS PROGRAM FOR H₀ MODEL (ALCOHOL USE)

/TITLE
alcohol missing group 1 (h₀)
/specifications
var = 6; cases=314; me=ml; anal=mom; ma=cov; groups = 5;
/labels
vl=alc_tl; v2=alc_t2; v3=alc_t3; v4=alc_t4; v5=alc_t5; v6=alc_t6;
f1=alc_int; f2=alc_slp;
equations
v1=f1+0f2+e1;
v2=f1+f2+e2;
v3=f1+2*f2+e3;
v4=f1+3*f2+e4;
v5=f1+4*f2+e5;
v6=f1+5*f2+e6;
f1=0*v999+d1;
f2=0*v999+d2;
/variances
e1 to e6 = *; d1 to d2=*
/covariances
d1 to d2=*
/constraints
/matrix
.785
.626 1.166
.460 .812 1.347
.401 .729 .897 2.004
.313 .545 .692 1.341 1.619
.197 .293 .424 .897 1.029 1.399
/means
1.452 1.761 1.895 2.631 3.022 3.411
/sta
.886 1.080 1.160 1.416 1.273 1.183
/print
/end

/TITLE
alcohol missing group 2
/specifications
var = 6; cases=44; me=ml; anal=mom; ma=cov;
/labels
vl=alc_tl; v2=alc_t2; v3=alc_t3; v4=alc_t4; v5=alc_t5; v6=alc_t6;
f1=alc_int; f2=alc_slp;
equations
v1=f1+0f2+e1;
v2=f1+f2+e2;
v3=f1+2*f2+e3;
v4=f1+3*f2+e4;
v5=f1+4*f2+e5;
f1=0*v999+d1;
f2=0*v999+d2;

/variances
  e1 to e5 = *; d1 to d2=*;
/covariances
  d1 to d2=*;
/constraints

/matrix
  .981
  .681 1.004
  .926 .918 1.684
  .613 .695 .926 .918 1.684
  .275 .469 1.387 1.911 .000 .000 .000 .000 1.000

/means
  1.636 1.705 2.114 3.023 3.364 .000
/sta
  .990 1.002 1.298 1.517 1.382 .000
/print
/end

/TITLE
  alcohol missing group 3
/specifications
  var = 6; cases=13; me=ml; anal=mom; ma=cov;
/labels
  v1=alc_t1; v2=alc_t2; v3=alc_t3; v4=alc_t4; v5=alc_t5; v6=alc_t6;
  f1=alc_int; f2=alc_slp;
/equations
  v1=f1+0f2+e1;
  v2=f1+f1f2+e2;
  v3=f1+2*f2+e3;
  v4=f1+3*f2+e4;
  f1=0*v999+d1;
  f2=0*v999+d2;
/variances
  e1 to e4 = *; d1 to d2=*;
/covariances
  d1 to d2=*;
/constraints

/matrix
  .077
  .026 1.064
  .064 .827 1.526
  .090 .974 .436 2.410
  .000 .000 .000 .000 1.000
  .000 .000 .000 .000 1.000
/means
1.077 1.692 2.231 2.923 .000 .000
/sta
.277 1.032 1.235 1.553 .000 .000
/print
/end

/TITLE
alcohol missing group 4
/specifications
var = 6; cases=19; me=ml; anal=mom; ma=cov;
/labels
v1=alc_t1; v2=alc_t2; v3=alc_t3; v4=alc_t4; v5=alc_t5; v6=alc_t6;
f1=alc_int; f2=alc_slp;
/equations
v1=f1+0f2+e1;
v2=f1+1f2+e2;
v3=f1+2*f2+e3;
f1=0*v999+d1;
f2=0*v999+d2;
/variances
e1 to e3 =*; d1 to d2=*;
/covariances
d1,d2=*;
/constraints
/matrix
1.228
 .836 1.029
 .904 .924 2.064
 .000 .000 .000 1.000
 .000 .000 .000 .000 1.000
 .000 .000 .000 .000 1.000
/means
1.684 1.842 2.211 .000 .000 .000
/sta
1.108 1.015 1.437 .000 .000
/print
/end

/TITLE
alcohol missing group 5
/specifications
var = 6; cases=11; me=ml; anal=mom; ma=cov;
/labels
v1=alc_t1; v2=alc_t2; v3=alc_t3; v4=alc_t4; v5=alc_t5; v6=alc_t6;
f1=alc_int; f2=alc_slp;
/equations
v1=f1+0f2+e1;
v2=f1+1f2+e2;
fl=0*v999+d1;
f2=0*v999+d2;

/variances
e1 to e2 = *; d1 to d2 = *
/covariances
d1 to d2 = *
/constraints

/matrix
.855
1.009 1.818
.000 .000 1.000
.000 .000 .000 1.000
.000 .000 .000 .000 1.000
.000 .000 .000 .000 .000 .000 1.000

/means
1.636 2.273 .000 .000 .000 .000
/sta
.924 1.348 .000 .000 .000

/print

/constraints
(1,d1,d2)=(2,d1,d2)=(3,d1,d2)=(4,d1,d2)=(5,d1,d2);
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(1,d2,d2)=(2,d2,d2)=(3,d2,d2)=(4,d2,d2)=(5,d2,d2);
(1,f1,v999)=(2,f1,v999)=(3,f1,v999)=(4,f1,v999)=(5,f1,v999);
(1,f2,v999)=(2,f2,v999)=(3,f2,v999)=(4,f2,v999)=(5,f2,v999);

(1,e1,e1)=(2,e1,e1)=(3,e1,e1)=(4,e1,e1)=(5,e1,e1);
(1,e2,e2)=(2,e2,e2)=(3,e2,e2)=(4,e2,e2)=(5,e2,e2);
(1,e3,e3)=(2,e3,e3)=(3,e3,e3)=(4,e3,e3);
(1,e4,e4)=(2,e4,e4)=(3,e4,e4);
(1,e5,e5)=(2,e5,e5);

(1,v3,f2)=(2,v3,f2)=(3,v3,f2)=(4,v3,f2);
(1,v4,f2)=(2,v4,f2)=(3,v4,f2);
(1,v5,f2)=(2,v5,f2)

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Note. Values represent standardized coefficients (t-values in parentheses); Maluse = mother's alcohol use, Faluse = father's alcohol use, SES = Socioeconomic status, Psup = parent support, Fsup = friend support, Pinf = parent influence, Finf = friend influence.