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A Latent Curve Framework for the Study of Developmental Trajectories in Adolescent Substance Use

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There is a great need to better link theoretical models and statistical models in studies of change over time, and this is especially evident in longitudinal studies of substance use and abuse. Historically, linking appropriate statistical models to developmental models has been difficult because the necessary statistical techniques had not yet been developed, or had been developed but were largely inaccessible to applied researchers. Although many of the conventional analytic techniques commonly used to test developmental research hypotheses perform exceptionally well given certain strict assumptions, these methods can be biased when applied under conditions in which the assumptions have been violated. These assumptions are often violated in longitudinal studies of substance use. A broad class of newly developed statistical techniques has recently become available that are much better suited for studying individual differences in development and change. One such approach is the structural equation modeling-based latent curve analysis. Latent curve analysis utilizes multiple indicator latent factors to estimate individual and group level components of growth over time and is the focus of the chapter. The chapter begins with a brief introduction to six research hypotheses about the developmental relation between adolescent substance use and peer substance use over time. A traditional cross-lagged model is used to evaluate these six research hypotheses using a large longitudinal data set, and the potential limitations of this modeling approach are discussed. An introduction is then provided to growth modeling in general and to latent curve analysis in particular. A series of latent curve models are then presented in detail that evaluate the six research questions of interest. The relative advantages and disadvantages of these models are discussed, and it is argued that latent curve analysis has much to offer the study of substance use over time.

There are a variety of theoretical models that have been developed to better understand the initiation and escalation of alcohol and drug use in children and adolescents (for a review, see Chassin, Presson, Sherman, & Curran, 1992; Maisto & Carey, 1985; Petraitis, Flay, & Miller, 1995; Sher, 1991). These models are typically used to generate specific research hypotheses that are amenable to empirical evaluation using some observed sample of observations and an appropriate statistical model. The sample results of the statistical models then provide some basis for making inferences back to the original theoretical model of interest. A critically important step in this process is the appropriate matching of the statistical model to the theoretical model that generated the research hypotheses to be evaluated. In empirical studies of adolescent substance use, the process of matching theoretical and statistical models is particularly difficult given the need to not only articulate theoretical causal mechanisms of influence, but to also clearly delineate theoretical expectations about individual differences in development and change over time. The selection of a statistical model that optimally evaluates the proposed research hypotheses directly depends upon the proper theoretical articulation of change over time.

For example, a developmental theory of substance use might generate a research hypothesis that describes the relation between characteristics of a school setting in the prediction of changes in adolescent alcohol use over time. Here, given that the behavior of multiple individuals within the school is of key interest, individual change in alcohol use over time might be best evaluated relative to all of the adolescents in the school. Change is thus conceptualized as the average difference between each individual within the group and the mean of that group over time. This type of change is sometimes referred to as *average group change*. Alternatively, a theory might generate a research hypothesis that describes the onset and developmental course of adolescent substance use during the transition from middle school to high school. Unlike the previous example, the change of interest here is *not* individual change relative to a larger group, but change within each individual adolescent. This type of change is sometimes referred to as *intraindividual*, or *within-person, change*. Finally, a theory might generate a research hypothesis that describes the relation between characteristics of the adolescent's peer group in the prediction of patterns of intraindividual change in adolescent substance use during the transition from middle school to high school. In this case, the theoretical model posits

between-person (or *interindividual*) differences in peer group characteristics that are related to within-person (or *intraindividual*) change in substance use over time. This type of change might be considered a comprehensive model of interindividual differences in intraindividual development over time.

All three of these examples are valid questions about stability and change, but each approaches change from a slightly different perspective and thus each is able to make unique inferences about development over time. One of the many reasons why it is so important for developmental theories of substance use to clearly articulate what type of change is being posited is that the statistical model selected to empirically evaluate the research hypotheses will directly depend on precisely what type of change or developmental process is hypothesized to exist. If the theory fails to specify, or incorrectly specifies, the type of change that is under study, the statistical model selected to evaluate the research hypotheses may be inappropriate. The failure to properly match the statistical model to the theoretical model can significantly limit, if not wholly bias, the inferences that can be made about the theoretical model of interest (Curran, 1997).

There is a great need to better link theoretical models and statistical models in applied developmental research and studies of change over time, and this is especially evident in developmental studies of substance use. Historically, linking appropriate statistical models to developmental models has been difficult because the statistical techniques needed to evaluate the theoretical research hypotheses had not yet been developed, or had been developed but were largely inaccessible to applied researchers. Although many of the conventional analytic techniques historically used to test developmental research hypotheses perform exceptionally well given certain rather strict assumptions, these same methods can be severely biased when applied under research conditions in which the assumptions have been violated (Rogosa, 1995; Rogosa & Willett, 1985; Willett, 1988). These assumptions are commonly violated in the study of individual differences in developmental trajectories of substance use, and this can unnecessarily limit the utility of many conventional statistical methods when empirically examining the onset and escalation of substance use in children and adolescents.

A broad class of newly developed statistical techniques are becomingly increasingly available that are much better suited for studying

individual differences in development and change. These recently developed methods are often referred to as *random coefficient models*, and the historical roots of these methods can be traced to the fields of biostatistics (e.g., Diggle, Liang, & Zeger, 1994; Laird & Ware, 1982; Rao, 1958), education (e.g., Bryk & Raudenbush, 1992; Burstein, 1980; Cronbach, 1976; Goldstein, 1987) and psychometrics (e.g., McArdle, 1988; McArdle & Epstein, 1987; Meredith & Tisak, 1984, 1990; Tucker, 1958). Latent curve analysis, a specific type of random coefficient model developed in the psychometric tradition, utilizes multiple indicator latent factors to estimate the fixed (or group level) and random (or individual level) components associated with individual differences in developmental trajectories over time. This is a powerful and flexible analytic technique that overcomes many of the challenges commonly encountered in the empirical study of adolescent substance use over time. Latent curve analysis is the primary focus of this chapter.

This chapter provides both a conceptual and a statistical introduction to latent curve analysis. This is accompanied by a detailed presentation of a series of latent curve models designed to study six research questions relating to adolescent and peer alcohol use previously addressed in Curran, Stice, & Chassin (1997). I begin with a brief introduction to three competing theoretical models of adolescent substance use relating to the role of peer group influences on individual behavioral development. Drawing from these three theories and from previous empirical research, I propose six specific research questions, followed by a description of the sample of adolescents and the measures that are used to empirically evaluate these six questions. I then present the findings from a more conventional longitudinal statistical model that serve both as a logical starting point for the analysis as well as providing a comparison to the later latent curve models. After describing the potential limitations of this conventional statistical model, I provide a general conceptual introduction to the modeling of individual differences in growth, followed by a more technical introduction specifically related to latent curve analysis. This is followed by the presentation of a series of detailed latent curve models designed to evaluate the six research questions. These models are constructed in increasing complexity, and each is explicitly tied back to the six proposed research questions. Finally, I conclude with a discussion of further extensions and potential limitations of the latent curve model and directions for future research.

A THEORETICAL MODEL OF ADOLESCENT SUBSTANCE USE: THE ROLE OF DEVIANT PEER AFFILIATIONS

Of the influences found to be related to adolescent substance use, peer substance use is consistently one of the strongest (Brook, Brook, Gordon, Whiteman, & Cohen, 1990; Hawkins, Catalano, & Miller, 1992). These findings have prompted many to conclude that peer substance use is a key proximal determinant of subsequent adolescent substance use (Oetting & Beauvais, 1986, 1987; Swaim, Oetting, Edwards, & Beauvais, 1989). This theoretical model is sometimes referred to as a *peer socialization* model such that adolescents who affiliate with substance-using friends are socialized to the behavior of the group and are thus more likely to use substances themselves. However, an alternative explanation is that adolescents who initiate illicit substance use as a function of other risk factors seek out a peer group that more closely matches their newly acquired behavior and attitudes (Farrell, 1994; Farrell & Danish, 1993). This theoretical model is sometimes referred to as a *peer selection* model such that adolescents who are already using substances are choosing to affiliate with a substance-using group. A third explanation combines these two processes such that adolescents tend to select friends who are similar to themselves in their substance use but are also susceptible to pressures of conformity from these same selected friends (Bauman & Ennett, 1994; Fisher & Bauman, 1988; Kandel, 1985). This theoretical model is sometimes referred to as a *bidirectional influences* model that combines elements of both the peer selection and the peer socialization models.

Previous empirical studies of the relation between peer substance use and adolescent substance use resulted in conflicting findings both in support and in refutation of each of the three theoretical models of peer influence (e.g., Bauman & Ennett, 1994; Farrell & Danish, 1993; Swaim et al., 1989). However, all of these previous studies used more conventional statistical models to evaluate rather complex theoretical questions about interindividual differences in intraindividual developmental trajectories of alcohol use over time. All of these previous studies provide important information about the relation between peer and adolescent substance use. However, one potential explanation for the discrepant findings may be related to a less than optimal match between the theoretical model of substance use and the statistical model used to

evaluate the research hypotheses. The goal of this chapter is to try to clearly articulate the theoretically derived research questions and to select a statistical model that most closely corresponds to the theoretical model under consideration. A variety of latent curve models is used to explore these three competing models of peer influences in adolescent substance use.

The first step in the study of these competing theories is to articulate clearly the specific research questions of interest by drawing directly on the theoretical models as well as on previous empirical findings in this area. The six research questions of interest are:

Question 1 (Q1): What are the characteristics of individual differences in developmental growth trajectories of adolescent alcohol use over time?

Question 2 (Q2): Are individual differences in developmental growth trajectories of adolescent alcohol use systematically related to the child's age, gender, or parental alcoholism diagnosis?

Question 3 (Q3): What are the characteristics of individual differences in developmental growth trajectories of peer alcohol use over time?

Question 4 (Q4): Are individual differences in developmental growth trajectories of peer alcohol use systematically related to the child's age, gender or parental alcoholism diagnosis?

Question 5 (Q5): How are individual differences in developmental trajectories of adolescent alcohol use related to individual differences in developmental trajectories of peer alcohol use?

Question 6 (Q6): Are earlier levels of adolescent alcohol use predictive of later developmental trajectories in peer alcohol use, and are earlier levels of peer alcohol use predictive of later developmental trajectories of adolescent alcohol use?

Questions 1 and 3 are based on previous empirical epidemiological findings describing the developmental trajectories of alcohol use in adolescence (e.g., Schulenberg, O'Malley, Bachman, Wadsworth, & Johnston, 1996); these questions address intraindividual differences in the development of adolescent and peer alcohol use over time. Questions 2 and 4 are based on both empirical and theoretical results that relate individual differences in age, gender, and parental alcoholism diagnosis to adolescent alcohol use and peer affiliations (e.g., Chassin, Curran, Hussong, & Colder, 1996; Sher, 1991); these questions address interindividual differences in intraindividual development over time.

Finally, Questions 5 and 6 are drawn directly from the three competing models of peer influences that explore the interrelations between adolescent alcohol use and peer alcohol use (e.g., Bauman & Ennett, 1994; Farrell & Danish, 1993; Oetting & Beauvais, 1987). Like Questions 2 and 4, Questions 5 and 6 also address interindividual differences in intraindividual development over time. Taken together, these six empirically and theoretically derived research questions address the developmental course, correlates, and predictors of adolescent and peer alcohol use over time.

Description of Data Set

Data were drawn from the Adolescent and Family Development Project (AFDP) to evaluate the six proposed research questions. AFDP is a longitudinal study of a large sample of children and their parents. Approximately one half of the sample consisted of children from families in which one or both of the parents were diagnosed as alcoholic; the remainder of the sample consisted of demographically matched control children from families in which neither of the parents were diagnosed as alcoholic. AFDP was funded by a grant from the National Institute on Drug Abuse to Laurie Chassin and Manuel Barrera at Arizona State University. Details about the design, recruitment strategies, and measures are presented in Chassin, Rogosch, & Barrera (1991), Chassin, Barrera, Bech, & Kossak-Fuller (1992), and Curran et al. (1997).

Subjects. The total sample at the first wave of measure (Time 1) consisted of 454 adolescents aged 10.5 to 15.5 years ($M = 12.7$, $SD = 1.45$) and their parents. Children of alcoholics (COAs, $N = 246$) had at least one biological and custodial alcoholic parent, and controls (CONs, $N = 208$) had no biological or custodial alcoholic parents. Of these 454 adolescents, 449 (99%) were interviewed at Time 2, 445 (98%) were interviewed at Time 3, and 442 (97%) were interviewed at all three time points. Because the current research questions focused on the relation between changes in adolescent alcohol use and changes in peer alcohol use, 74 adolescents were not included because they reported no substance use themselves *and* no peer substance use at any time point (e.g.,

complete abstainers).[1] Finally, five observations were found to be extreme outliers and exerting an inappropriate influence on the statistical models, and were subsequently dropped (see Curran et al., 1997, for details). The final sample used for the current analyses consisted of 363 families. The average adolescent age at Time 1 was 12.9 years, 56% were COAs, 48% were female, 25% were Hispanic, and 75% were non-Hispanic Caucasian.

Measures. Demographic variables consisted of *child age* (measured in years at Time 1) and *child gender* (male coded 1, female coded 0). Lifetime DSM-III diagnoses of *parental alcohol abuse or dependence* was a dichotomous variable reflecting the presence (coded 1) or absence (coded 0) of alcoholism diagnosis in either or both parents. Adolescents reported how many of their friends drank alcohol occasionally (one item) and how many of their friends drank alcohol regularly (one item) over the previous 12 months. Response options ranged from 0 (*none*) to 5 (*all*), and a single *peer alcohol use* score was calculated by summing the two items. Adolescents self-reported their frequency of consumption of beer/wine and hard liquor (2 items), frequency of consumption of 5 or more drinks in a row (1 item), and frequency of getting drunk (1 item) in the past 12 months. Response options ranged from 0 (*not at all*) to 7 (*every day*). A single *adolescent alcohol use* score was calculated by summing the four items. Correlations, standard deviations, means, skewness and kurtosis for all variables are presented in Table 1.1.

Conventional Fixed Effects Model

Prior to estimating the latent curve models, it is often helpful to begin by examining the data using more traditional analytical methods. This provides important insights into the general characteristics of the data, and also allows for a comparison between the conventional analysis and the latent curve models. To test the six research questions, I first used an auto-regressive, cross-lagged (ARCL) panel model (Dwyer, 1983). The

[1]These complete abstainers were not included in the analyses given the focus on studying the inter-relations among changes in adolescent alcohol use and changes in peer alcohol use, and this subgroup reported no use on either construct at any time point. For comparative purposes, all models were re-estimated including the complete abstainers. Although there were mean and variance differences in the growth trajectories, there were no differences in the substantive conclusions compared to the analysis without the abstainers.

TABLE 1.1

Means, Standard Deviations, Univariate Skewness and Zero-Order Correlations for all Predictor and Criterion Variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. Time 1 Adolescent Alcohol Use	1.00								
2. Time 2 Adolescent Alcohol Use	.68	1.00							
3. Time 3 Adolescent Alcohol Use	.50	.68	1.00						
4. Time 1 Peer Alcohol Use	.66	.58	.46	1.00					
5. Time 2 Peer Alcohol Use	.53	.65	.52	.65	1.00				
6. Time 3 Peer Alcohol Use	.38	.51	.61	.48	.58	1.00			
7. Age	.31	.31	.23	.48	.44	.29	1.00		
8. Gender	.01	.06	.10	-.09	-.02	-.13	.14	1.00	
9. COA Status	.12	.17	.20	.09	.12	.16	.12	-.15	1.00
Mean	1.36	2.12	3.18	1.33	1.75	2.48	12.91	0.52	0.56
Standard Deviation	2.81	3.98	4.79	1.74	1.82	2.01	1.40	0.50	0.49
Skewness	3.23	2.73	2.07	1.57	1.16	0.66	-.28	-.06	-.26

Note: Absolute values of correlations greater than $r = .19$ represent a per-test $p < .002$ and a familywise $p < .10$. Statistics based on $N = 363$.

most common form of the ARCL model is a fixed-effects, regression-based linear model for studying stability and change in a construct over time. These models can be estimated using a series of multiple regression analyses, but more typically ARCL models are estimated using a structural equation modeling (SEM) framework. The ARCL model estimates the stability of a construct over time by regressing later measures of a construct onto earlier measures of the same construct. For example, Time 1 alcohol use predicts Time 2 alcohol use, and Time 2 alcohol use in turn predicts Time 3 alcohol use. These across-time/within-construct predictions are often referred to as *stability coefficients*, or simply *stabilities*. Once these stabilities are estimated, then earlier measures of one construct are used to predict later measures of another construct. For example, Time 1 peer alcohol use predicts Time 2 adolescent alcohol use, above and beyond the effects of Time 1 adolescent alcohol use. This parameterization provides a test of whether earlier peer alcohol use predicts later adolescent alcohol use, above and beyond the effects of earlier alcohol use. In principal, ARCL models can be extended to include multiple time points across multiple constructs.

The current ARCL model was built in five progressive steps (see Curran et al., 1997, for details). First, the Time 1 measures of adolescent alcohol use and peer alcohol use were regressed on adolescent age, gender, and parent alcoholism diagnosis. Second, the stabilities between time-adjacent measures of adolescent alcohol use and peer alcohol use were estimated. Third, the errors of equations (or disturbances) of the repeated measures were correlated within-time/across-construct (e.g., Time 1 adolescent use was correlated with Time 1 peer use, etc.). Fourth, the four *cross-lagged* effects were estimated (the across-time/across-construct regression parameters). Each subsequent step was associated with a significant improvement in model fit assessed using nested chi-square tests. Finally, Lagrange multipliers suggested the post hoc inclusion of a direct effect from age to Time 2 peer alcohol use and from gender to Time 3 peer alcohol use. The final ARCL model was found to fit the observed data well ($\chi^2(14, N = 363) = 29.3$, $p = .01$; Comparative Fit Index [CFI] = .99; Tucker–Lewis Index [TLI] = .97; Root mean square error of approximation [RMSEA] = .055 with a 90% confidence interval [90CI] = .03, .08). The final model is shown in Fig. 1.1 and the EQS (Bentler, 1997) computer code is presented in Appendix A.

The ARCL model resulted in large and positive stability coefficients for both adolescent alcohol use and peer alcohol use suggesting that the average adolescent standing relative to the group mean on either construct at an earlier time point tended to be quite similar to the average adolescent standing relative to the group mean at a later time point. That is, on average, adolescents reporting alcohol use above the mean at Time 1 tended to report alcohol use above the mean at Time 2, and adolescents reporting alcohol use above the mean Time 2 tended to report alcohol use above the mean at Time 3. Note that, although research questions *Q1* and *Q3* addressed issues of *individual* change in developmental trajectories of adolescent and peer alcohol use, the ARCL model analyzed change relative to the *group* average over time. Because of this, it is quite difficult to make any meaningful inferences about research questions *Q1* and *Q3* based on these ARCL results.

Research questions *Q2* and *Q4* addressed the issue of systematic individual differences in developmental change in adolescent and peer alcohol use as a function of child age, gender, and parental alcoholism diagnosis. As with the previous questions, clear inferences about Questions 2 and 4 are difficult to make based on the ARCL model results. For example, older adolescents and COAs reported significantly ($p < .05$)

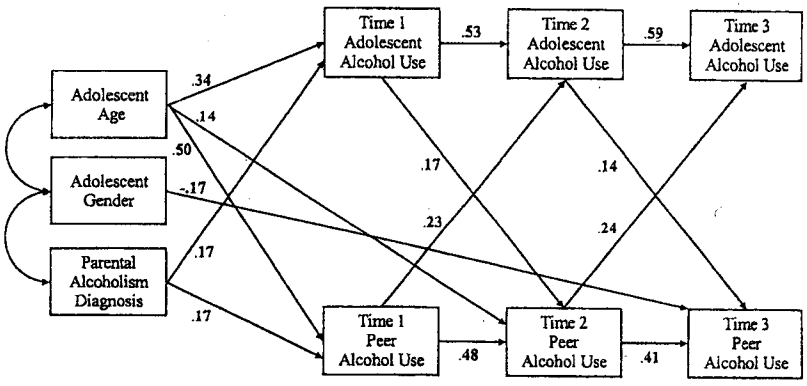

Fig. 1.1. Conventional auto-regressive cross-lagged panel model.
Note: Model fit was $\chi^2(14, N = 363) = 29.3$, $p = .01$, TLI = .97, CFI = .99, RMSEA = .055. Parameter estimates are standardized and were all significant ($p < .05$). Covariances among the within time disturbances of the repeated measures are not shown.

higher Time 1 adolescent alcohol use and higher Time 1 peer alcohol use, older adolescents reported higher Time 2 peer alcohol use, and males reported lower Time 3 peer alcohol use. Again these ARCL inferences are made with reference to average relative standing at a specific time point. So, parental alcoholism diagnosis was positively related to Time 1 adolescent and peer alcohol use, but in this modeling framework could not be used to predict continuous changes in alcohol use across Times 1, 2, and 3. This again severely limits our ability to draw meaningful inferences about the hypothesized research questions from the ARCL model results.

Finally, research question *Q5* inquired about the correlation between individual differences in developmental trajectories of adolescent and peer alcohol use, and research question *Q6* explored the existence of systematic individual differences in developmental trajectories as a function of earlier levels of one construct predicting later changes in the other construct. Unfortunately, the ARCL model allows no inferences to be made about question *Q5* in that this model is not estimating characteristics of individual growth, but instead of average relative standing across time. With respect to *Q6*, the ARCL model resulted in large and significant prospective bidirectional effects between adolescent alcohol use and peer alcohol use. That is, earlier peer use positively predicted later adolescent use, above and beyond earlier adolescent use; similarly, earlier adolescent use positively predicted later peer use, above and beyond earlier peer use. These prospective bidirectional paths were significant across both time lags. Again, because these prospective paths represent average relative standing between two discrete time points, and not continuous change across all three time points, these results only partially inform research question *Q6*.

In sum, the results from the ARCL model suggest that there is significant stability of adolescent alcohol use and peer alcohol use over time, that age, gender, and parental alcoholism diagnosis are related to time specific measures of alcohol use, and that there is support for a prospective bidirectional relation between adolescent and peer alcohol use such that earlier measures of one construct predicted later measures on the other construct. However, when comparing how developmental change was conceptualized by the three competing theories with how change is actually being evaluated in the statistical model, there seems to be a poor match between the theoretical and the statistical models of interest. To better understand this mismatch, we need to more closely

consider the behavior of the ARCL model under conditions of systematic development over time. Rogosa (1988, 1995), Rogosa & Willett (1985), and Willett (1988) present excellent discussions of the general assumptions and related limitations of these types of models, so I will not review these issues fully here. I will, however, comment on several of the more problematic issues relating to the particular research questions at hand.

First, note that a single parameter characterizes the stability of each construct over two time points for *all* subjects in the sample. For example, the standardized regression parameter estimate predicting Time 2 alcohol use from Time 1 alcohol use for the current sample is $\beta = .53$; this single value is meant to capture the relation between Time 1 and Time 2 alcohol use for all $N = 363$ subjects in the sample. Characterizing this prospective relation using a single value for all subjects is likely not feasible given the expected heterogeneity in change within the sample, and this certainly does not tell us much about the characteristics of growth in alcohol use over time. We will see that it is often extremely important to allow parameter values describing change over time to vary freely *across* individuals instead of having one value fixed for all individuals.

Second, the ARCL model calculates individual change between two time points relative to the group, *not* relative to the individual. This can be of profound importance depending upon how a particular theory construes change over time. For example, the average alcohol use for all $N = 363$ adolescents at Time 1 and Time 2 was 1.4 and 2.1, respectively. So, as a group, average alcohol use increased by .70 between Time 1 and Time 2. Say that Adolescent 1 reported alcohol use of 1.75 at both the first and the second time points. Given that the group mean increased .7 units over the two time points, but the individual reported equal rates of use at both time points, conventional statistical models would consider this individual to be making a *negative* contribution to the average covariance between the two time points. This is because Adolescent 1 reported alcohol use *above* the mean at Time 1, but *below* the mean at Time 2, even though their *own* alcohol use did not change. In contrast, say that Adolescent 2 reported alcohol use of .7 at Time 1 and 1.4 at Time 2. Although reporting a .70 increase in alcohol use between the two time points, this individual would subsequently make a contribution of *perfect stability* to the average covariance between the two time points because Adolescent 2 increased at a rate *equal* to that reported by the

overall group. So, although Adolescent 2 reported an increase in their own alcohol use between the two time points, conventional statistical models consider Adolescent 2's alcohol use to be stable over time. As can be seen, it is of great importance for the researcher to articulate clearly whether research questions of interest relate to change *relative to the group*, or change *relative to the individual*. Conventional statistical models typically construe change relative to the group, but developmental theory is often interested in change relative to the individual.

A final limitation of the ARCL model is that we often assume from a theoretical perspective that the onset and escalation of alcohol use tend to follow a rather continuous developmental trajectory over time, particularly during childhood and adolescence. That is, substance use is typically characterized by some point of initiation in early adolescence, followed by an accelerated rate of escalation in middle to late adolescence, peaking and gradual decrease in use in the early 20s, and, finally, the development of some stable level of use in the middle or late 20s (Muthén & Muthén, in press). Given the existence of such a developmental trajectory, examining growth as a series of time adjacent comparisons (Time 1 vs. 2, Time 2 vs. 3) is artificially carving up the continuous trajectory into a series of two-timepoint snapshots of change (Willett, 1988). This further highlights the problem of using a statistical model to test a research hypothesis that does not optimally correspond to the theoretical model that gave rise to the research hypothesis.

Because of these limitations, and several others not detailed here, conventional approaches to longitudinal data analysis tend not to be well suited for studying interindividual differences in intraindividual developmental change over time. Let's now consider an alternative analytic approach that might allow for stronger empirical tests of our developmental research hypotheses.

Models of Individual Growth

How can we work toward overcoming the limitations of the more conventional statistical models? One solution is to approach the question of development and change from an entirely different perspective. The ARCL model predicted Time 3 alcohol use from Time 2 alcohol use, and Time 2 alcohol use from Time 1 alcohol use. Say instead that we were to use the three observed repeated measures of alcohol use to estimate a *single* underlying true growth trajectory for each person

across all three time points. An example of this is displayed in Fig. 1.2 for a single subject drawn from the sample of $N = 363$.

Instead of examining the time-adjacent relations of the alcohol use measures between Times 1 and 2, and Times 2 and 3 (as was done in the ARCL model), we have estimated a single continuous line that "best fits" the three observed measures. This line of best fit can be thought of in much the same way we would fit a regression line to a scatter plot of data points between two measures. However, in this case, the x variable is time (where x equals 0, 1, and 2), the y variable is alcohol use, and we consider one subject at a time. We consider this line of best fit as an estimate of the *individual's true growth trajectory* of alcohol use over time.

Whereas three repeated measures were used to estimate the growth trajectory, the trajectory is completely characterized by just two pieces of information: a starting point (or intercept, which we will refer to as α_i to denote the α value specific to subject i) and a rate of change over time (or slope, which we will refer to as β_i to denote the β value specific to the same subject i). The three observed repeated alcohol use measures

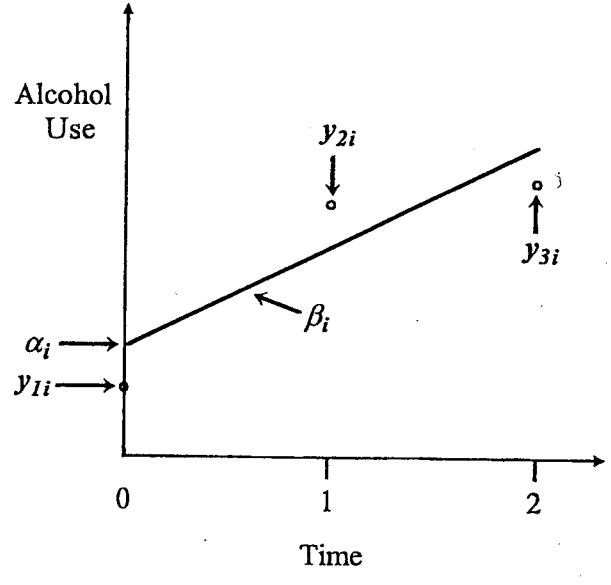

Fig. 1.2. Hypothetical growth trajectory fit to three observed measures of alcohol use. Note: α represents the intercept of the trajectory and β represents the individual slope of the trajectory.

for subject i can now be expressed as an additive function of the individual intercept and slope estimates such that:

$$y_{it} = \alpha_i + \beta_i a_t + \varepsilon_{it} \quad (1.1)$$

where y_{it} represents the observed alcohol use measure for person i at timepoint t, α_i represents the true intercept of the growth trajectory for person i, β_i represents the true slope of the growth trajectory for person i, a_t represents the value of time at timepoint t (e.g., 0, 1, or 2), and ε_{it} represents the time specific residual for person i at time t. Equation 1.1 is sometimes referred to as the *Level 1,* or *within person* model.

Say that we now fit trajectories for a sample of four individuals as is shown in Fig 1.3.

Now α_i and β_i from Equation 1.1 are random variables, and variation in these variables across individuals can be expressed as

$$\alpha_i = \mu_\alpha + u_{\alpha i} \quad (1.2a)$$

$$\beta_i = \mu_\beta + u_{\beta i} \quad (1.2b)$$

Equations 1.2a and 1.2b are sometimes referred to as the *Level 2,* or the *between person,* model. These equations highlight that the individual intercept and slope values describing the developmental trajectories are now the dependent variables of most interest to the model, and not the individual observed repeated observations of y over time.

The Level 2 model (Eqns. 1.2a and 1.2b) can be substituted into the Level 1 model (Equation 1.1) to result in a combined model

$$y_{it} = (\mu_\alpha + u_{\alpha i}) + a_t (u_\beta + \mu_{\beta i}) + \varepsilon_{it} \quad (1.3)$$

and simple rearrangement of terms results in

$$y_{it} = (\mu_\alpha + a_t \mu_\beta) + (u_{\alpha i} + a_t u_{\beta i} + \varepsilon_{it}) \quad (1.4)$$

Equation 1.4 clarifies that the observed repeated measures of y can be expressed as an additive combination of a *fixed* component of growth (e.g., $\mu_\alpha + a_t \mu_\beta$) and a *random* component of growth (e.g., $u_{\alpha i} + a_t u_{\beta i} + \varepsilon_{it}$). These parameters will combine to provide important information about our research questions of interest. The two fixed effects of growth are

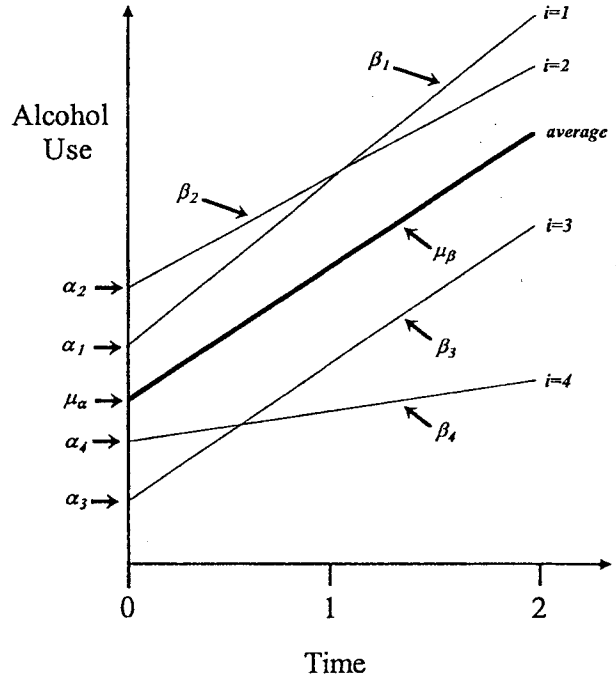

Fig. 1.3. Four fitted growth trajectories.
Note: Light lines represent individual trajectories and the heavy line represents the mean growth; α represents the individual intercepts, β represents the individual slopes, μ_α represents the mean intercept, and μ_β represents the mean slope.

μ_α (the mean intercept of the growth trajectory) and μ_β (the mean rate of change of the growth trajectory). The random effects of growth include the variance around the mean intercept ($var(u_{\alpha i}) = \sigma^2_\alpha$), the variance around the mean slope ($var(u_{\beta i}) = \sigma^2_\beta$), the covariance between the intercept and slope ($cov(u_{\alpha_i}, u_{\beta_i}) = \sigma_{\alpha\beta}$), and the time specific residual variance ($var(\varepsilon_{it}) = \sigma^2_\varepsilon$).

The model described in Eqn. 1.4 is sometimes referred to as an *unconditional* growth model (Bryk & Raudenbush, 1987); that is, we have estimated the mean starting point and mean rate of change of the developmental trajectories (the fixed effects) as well as the individual variability in both the starting point and rate of change (the random effects). Although this unconditional model allows for estimation of the general characteristics of individual differences in growth, we have

made no attempt to *predict* these individual differences. We do not know what factors might differentiate children who report drinking at a high rate at the initial assessment period from those who do not, or what factors might differentiate children who report steep increases in alcohol use over time from children who do not. These are questions of *inter*individual, or *between person*, differences in *intra*individual change over time. To explore what factors are predictive of individual differences in starting point and rates of change over time, we must move to a *conditional* growth model.

The conditional growth model incorporates Equation 1.1 as stated above, but Equations 1.2a and 2b are expanded to include explanatory variables such that

$$\alpha_i = \mu_\alpha + \gamma_1 x_{1i} + \gamma_2 x_{2i} + u_{\alpha i} \quad (1.5a)$$

$$\beta_i = \mu_\beta + \gamma_3 x_{1i} + \gamma_4 x_{2i} + u_{\beta i} \quad (1.5b)$$

where x_{1i} and x_{2i} are two explanatory variables of interest (say gender and parental alcoholism diagnosis), and γ_1, γ_2, γ_3, and γ_4 are fixed regression parameters that relate the explanatory variables to the intercept and slope components of growth. (These are fixed parameters because they do not vary across individual). Whereas the unconditional model allowed us to examine general characteristics of individual differences in development over time (needed to examine questions *Q1*, *Q3*, and *Q5*), the conditional model allows us to predict individual differences in development as a function of our set of explanatory variables (needed to examine questions *Q2*, *Q4*, and *Q6*).

Summary. Using our six theoretically and empirically derived research questions as a guide, we conceptually know precisely what type of information we would like to extract from our observed data. First, we would like to use the observed repeated measures of adolescent and peer alcohol use to estimate an underlying true growth trajectory for each individual in the sample. Second, we would like to compute summary statistics for these individual trajectories across the entire sample to better understand characteristics of individual differences in growth over time. Third, we would like to incorporate explanatory variables to examine predictors of individual differences in growth over time. Finally, we would like to build a multivariate model that will

estimate the interrelations between developmental trajectories of adolescent alcohol use and developmental trajectories of peer alcohol use over time.

Although we can clearly articulate the types of analytical models we require to optimally evaluate our research hypotheses, in the past it has been quite difficult to statistically estimate these types of models in an appropriate way. Fortunately, there are a variety of statistical modeling techniques that have recently become available that allow for the estimation of the fixed and random components of growth. Recent developments include latent curve analysis, hierarchical linear models, general estimating equations, general mixed models, and Bayesian growth models. A thorough comparison of these various techniques is beyond the scope of this chapter (but see MacCallum, Kim, Malarkey, & Kiecolt-Glaser, 1997, and Willett & Sayer, 1994, for excellent comparisons of several of these modeling techniques). I will focus explicitly on the use of latent curve analysis.

Latent Curve Analysis

Structural equation modeling (SEM) is a powerful analytic framework that allows for the simultaneous estimation of the relations between a set of observed variables and a smaller set of underlying latent constructs (the confirmatory factor or measurement model), as well as the relations among the latent constructs themselves (the structural model); (Bentler, 1980, 1983; Jöreskog, 1971a, 1971b; Jöreskog & Sörbom,1979). Latent curve analysis (LCA) is a highly structured type of structural equation model. Whereas SEM typically (but not necessarily) incorporates only information about the covariance structure of the observed measures, LCA uses information about both the covariance structure and the mean structure of the observed measures. Conceptually, LCA considers the observed repeated measures over time to be fallible indicators of an unobserved true growth trajectory. The observed repeated measures are thus used to define one or more underlying latent growth factors using the SEM framework. The fixed and random components of the growth trajectory are then estimated via the means and variances of the latent growth factors. These latent growth factors can then be regressed on one or more exogenous variables to examine predictors of individual differences in change over time. The technical developments of latent curve analysis are presented in McArdle (1988, 1989, 1991), McArdle &

Epstein (1987), Meredith & Tisak (1984, 1990), and Muthén (1991, 1993). Applied substantive examples of latent curve models include Curran, Muthén, & Harford (1998), Curran et al. (1997), Duncan, Duncan, Alpert & Hops (1997), MacCallum et al. (1997), and Stoolmiller (1994).

We will now use the latent curve models to estimate the fixed and random components of growth described above in order to address our six research questions of interest. Recall that the first research question (*Q1*) seeks to describe the characteristics of intra-individual differences in developmental trajectories in adolescent alcohol use. We will begin with the estimation of an unconditional growth model in which we examine the characteristics of the growth process prior to incorporating explanatory variables to predict growth. Using the structural modeling framework, two latent factors are defined to represent the intercept and slope of the alcohol use growth trajectory. The factor loadings relating the three observed alcohol use measures to the intercept factor are all fixed to 1.0 to define the starting point of the alcohol use growth trajectory. The factor loadings relating the observed repeated measures to the slope factor are a combination of fixed or free loadings that best capture the functional form of the growth trajectory over the three time points. The initial approach is typically to fix the factor loadings to 0, 1, and 2 to represent straight-line growth.[2]

The means of the two latent factors are freely estimated, but the intercepts of the observed repeated measures are fixed to zero. The observed mean structure among the repeated measures is thus reproduced completely through the means of the latent factors. The sample estimate of the mean of the latent intercept factor ($\hat{\mu}_\alpha$, denoted by a "hat" to indicate a sample estimate of a population parameter) represents the mean initial status of the alcohol use growth trajectory; the sample estimate of the variance of the latent intercept factor ($\hat{\sigma}^2_\alpha$) represents the

[2]One difficult issue to resolve in latent curve modeling is how to best define the factor loadings relating the observed repeated measures to the latent slope factor. In this example, I have fixed these loadings to values of 0, 1, and 2 to define linear growth. One advantage of the latent curve approach is that the adequacy of these loadings can be formally tested using nested group comparisons. However, given that only three assessment periods were available here, linear growth was the most complicated functional form of growth that could be estimated. A minimum of four time points is necessary to test higher order functions (e.g., quadratic polynomials). There are several approaches that can be used to define different growth functions, but a full discussion of these issues are beyond the scope of this chapter. McArdle (1989, 1991) and Willett & Sayer (1994) provide excellent discussions of these issues.

individual variability in initial levels of use. Similarly, the sample estimate of the mean of the latent slope factor ($\hat{\mu}_\beta$) represents the mean slope of the alcohol use trajectory, and the sample estimate of the variance of the latent slope factor ($\hat{\sigma}^2_\beta$) represents individual variability in rates of change in alcohol use over time.

Research Question *Q1*: Characteristics of Individual Differences in Developmental Trajectories of Adolescent Alcohol Use

To examine research question *Q1*, an unconditional latent curve model was estimated for adolescent alcohol use. Model 1 is presented in Fig. 1.4 and the computer code is presented in Appendix B.[3] To estimate this model, the three repeated measures of alcohol use were defined to load on both the latent intercept factor and the latent slope factor by fixing the factor loadings relating the observed measures to the underlying latent factors to prespecified values. The factor loadings relating the three adolescent alcohol use measures were fixed to 1.0 on the intercept factor, and 0, 1.0, and 2.0 on the slope factor. The means of the two latent growth factors (not shown in Fig. 1.4) were freely estimated whereas the means of the observed repeated measures were not. Finally, variances were estimated for both the observed measures and for the two latent growth factors. It is the variance in the growth factors that infers the presence or absence of individual differences in growth over time. The model shown in Fig. 1.4 fit the observed data well ($\chi^2(1, N = 363) = 2.1$, $p = .15$, TLI = .99, CFI = .99, RMSEA = .055 with 90CI = 0, .16). Together, these various measures suggest a close fit of the model to the observed data.

Once we determined that there is an adequate overall fit of the model to the observed data, we can now evaluate the eight estimated model parameters: two means (one for the intercept factor and one for the slope factor), five variances (three error variances for the repeated measures and two variances for the growth factors), and one covariance (between the intercept and slope factor). First, the means for the

[3]Although I used EQS to estimate all of the latent curve models here, any SEM software package that can estimate both mean and covariance structures can equivalently be used to estimate the latent curve models (e.g., Amos, CALIS, Mplus, LISREL, Mx, etc.).

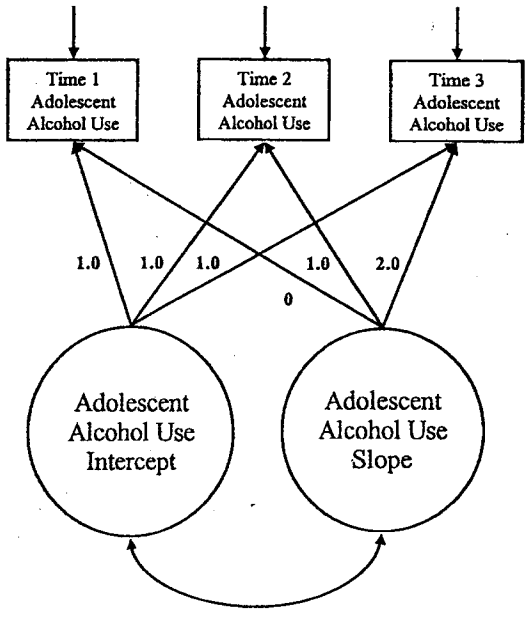

Fig. 1.4. Unconditional latent curve model for three repeated measures of adolescent alcohol use.
Note: Model fit was $\chi^2(1, N = 363) = 2.1$, $p = .15$, TLI = .99, CFI = .99, RMSEA = .055. Factor loadings are fixed to shown values; estimated parameters include five variances (one for each repeated measure, one for each latent factor), one covariance (between the two latent factors), and two means (one for each latent factor).

intercept and slope factors were estimated to be $\hat{\mu}_\alpha = 1.36$ and $\hat{\mu}_\beta = .88$, respectively, both of which significantly differed from zero ($p < .05$). This indicated that the estimated mean starting point of adolescent alcohol use was 1.36, and the estimated mean rate of true change was .88 units per year. Next, there were significant variance estimates for both the intercept ($\hat{\sigma}^2_\alpha = 7.9$) and slope ($\hat{\sigma}^2_\beta = 3.2$) factors, indicating that there was substantial individual variability around both the mean starting point and mean rate of change over time. Finally, there was a marginally significant ($p = .10$) estimate for the covariance between the intercept and slope factors ($\hat{\sigma}_{\alpha\beta} = -.54$)that was standardized to a correlation of $r = -.11$, indicating that adolescents reporting higher initial levels of alcohol use tended to report slightly smaller rates of increase in alcohol use over time.

In sum, the unconditional growth model indicated that the group reported a significant amount of alcohol use at the initial starting point, the group reported significant increases in alcohol use over the three time points, and there was significant individual variability in both the starting point and rates of change over time. These results are critical information needed to fully evaluate research question *Q1*. Note the greater depth and dynamism of information resulting from the latent curve results relative to those obtained from the ARCL stability coefficients estimated from the very same data. This reflects the additional information that can be extracted from the observed data using a growth modeling approach to the study of change over time.

Research Question *Q2*: Predicting Individual Differences in Developmental Trajectories of Adolescent Alcohol Use

Given the presence of variability in intraindividual differences in the developmental growth trajectories of adolescent substance use (that is, the significant variance estimates in the intercept and slope factors), we are now interested in trying to model this variance using additional explanatory variables. This allows us to explore predictors of individual developmental trajectories as was posited in research question *Q2*. To model the observed variability in the intercept and slope factors, we regress the two latent growth factors on three exogenous variables: age, gender, and parental alcoholism diagnosis. Model 2 is presented in Fig. 1.5 and the computer code is presented in Appendix C.

This conditional growth model was estimated and was found to fit the data quite well ($\chi^2(4, N = 363) = 5.5$, $p = .24$, TLI = .99, CFI = .99, RMSEA = .033 with 90CI = 0, .09). Of key interest here are the sample estimates of the regression parameters linking the three exogenous variables to the two growth factors because these estimates capture potential predictability of individual differences in development as a function of the explanatory variables. Examination of significant ($p < .05$) *z*-ratios indicated that older children and children of alcoholics reported higher initial levels of alcohol use and that older children, boys, and children of alcoholics reported steeper increases in alcohol use over time. Finally, even with the inclusion of the three explanatory variables, there remained significant variability in both the intercept and slope

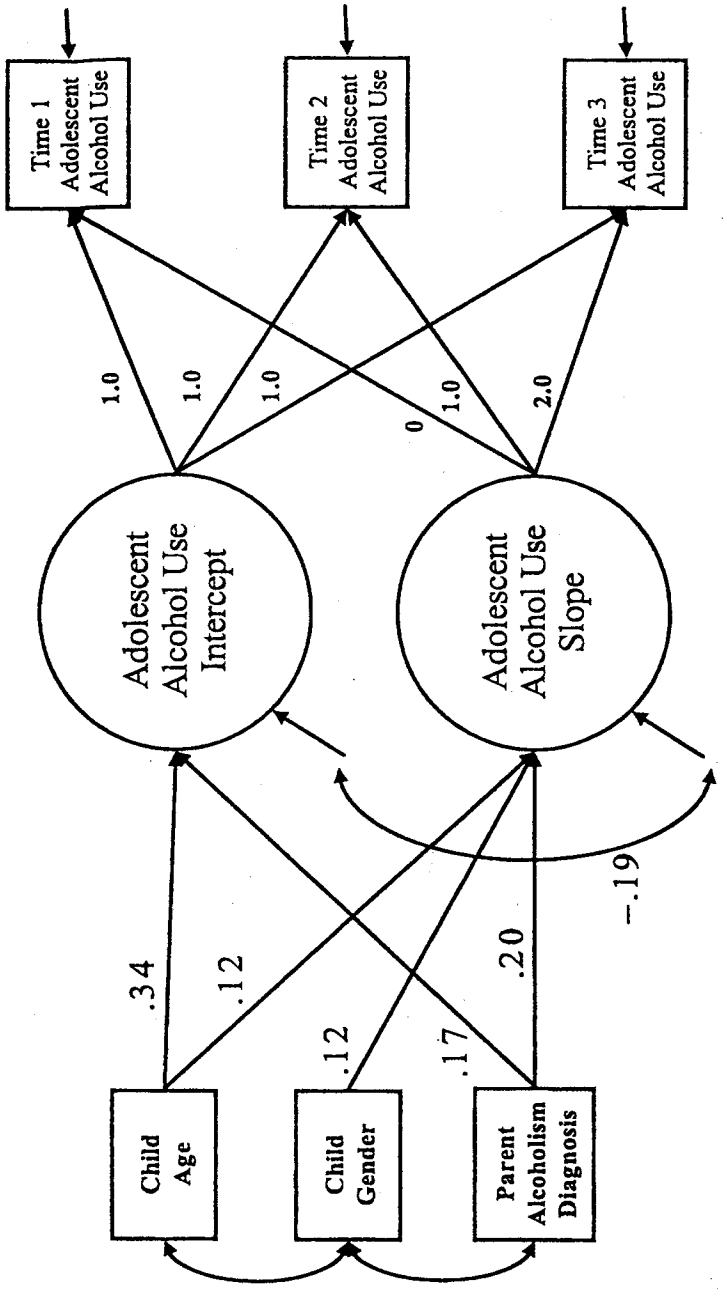

Fig. 1.5. Conditional latent curve model for adolescent alcohol use regressed on adolescent age, gender, and parental alcoholism diagnosis. Note: Model fit was $\chi^2(4, N = 363) = 5.5$, $p = .24$, TLI = .99, CFI = .99, RMSEA = .033. Only significant paths ($p < .05$) are shown. All parameter estimates are standardized.

factors, indicating that additional explanatory variables are necessary to fully model the observed individual differences in growth over time.

Thus far, we have learned a great deal about the characteristics of growth in adolescent alcohol use over the three time points, and in the prediction of growth in alcohol use from age, gender, and parental alcoholism diagnosis. Recall that one of the key goals of these analyses is to explore the relation of the child's peer's alcohol use and the child's own alcohol use (research questions *Q5* and *Q6*). An initial step might be to simply add the Time 1 measure of peer alcohol use to the conditional growth model containing the three exogenous variables we just estimated. Model 2 could be easily modified to incorporate Time 1 peer use as a fourth exogenous variable, and then the intercept and growth factors could be regressed on Time 1 peer use. However, our theories predict that peer use itself is developing systematically over time, so considering just the Time 1 measure of peer use in the presence of systematic change in peer use over time could be unnecessarily limiting. We thus must also examine growth in peer use prior to moving toward exploring research question *Q5*.

Research Question *Q3*: Characteristics of Individual Differences in Developmental Trajectories of Peer Alcohol Use

We need to retrace our steps and examine an unconditional growth model for peer alcohol use, which is identical in form to Model 1 but with the three peer alcohol use measures instead of the three adolescent alcohol use measures. This model was estimated and fit the observed data well ($\chi^2(1, N = 363) = 4.5$, $p = .03$, TLI = .97, CFI = .99, RMSEA = .09 with 90CI = .02, .19). A significant mean estimate for the intercept factor ($\hat{\mu}_\alpha = 1.3$) reflected that the estimated mean starting point for peer alcohol use was 1.3; a significant mean estimate of the slope factor ($\hat{\mu}_\beta = .55$) reflected that the estimated mean rate of change for peer alcohol use was .55 units per year. Further, large and significant variance estimates in both the intercept factor ($\hat{\sigma}^2_\alpha = 2.5$) and slope factor ($\hat{\sigma}^2_\beta = .40$) reflected the presence of individual variability in both mean starting point and mean rate of change over time. Finally, a significant correlation of r = .38 was found between intercept and slope, indicating that adolescents with lower initial levels of peer use tended to report steeper increases in peer use over time. Now we can extend this model to

examine potential predictors of interindividual differences in these intraindividual developmental trajectories.

Research Question *Q4*: Predicting Individual Differences in Developmental Trajectories of Peer Alcohol Use

To evaluate question *Q4* that posited predictors of individual differences in peer alcohol use, the unconditional peer use model was regressed on the three measures of age, gender and parental alcoholism diagnosis. This model fit the observed data well ($\chi^2(4, N = 363) = 12.2$, $p = .02$, TLI = .94, CFI = .98, RMSEA = .08 with 90CI = .03, .13); significant parameter estimates indicated that older children and children of alcoholics reported higher initial levels of peer alcohol use, and that younger children reported steeper increases in peer alcohol use over time. As was found with adolescent alcohol use, significant variability remained in both the intercept and slope factors after inclusion of the three explanatory variables suggesting additional variables are needed to fully understand individual differences in growth in peer alcohol use over time.

Research Question *Q5* & *Q6*: Multivariate Relations Between Individual Differences in Developmental Trajectories of Adolescent Alcohol Use and Developmental Trajectories of Peer Alcohol Use

The four latent curve models presented previously are considered *univariate* given that change in only one construct was considered, even though multiple measures were taken on that one construct. We now create a *multivariate* latent curve model by simultaneously estimating the two unconditional univariate growth models of adolescent alcohol use and peer alcohol use, and regressing both growth processes on the exogenous measures of age, gender, and parental alcoholism diagnosis. This allows us to examine research questions *Q5* (the relation between developmental trajectories of adolescent and peer alcohol use over time) and *Q6* (earlier levels on one construct predicting later developmental change on the other construct). It is important to note that the more complicated these growth models become, the greater variety of ways there are to approach the model building strategy. There is not necessarily a right or wrong way to build and probe these models, but whatever steps are taken should be clearly articulated, well justified, and closely guided by the theoretical questions of interest. I follow these guidelines in the construction of Model 3.

I define the covariance structure among the latent growth factors such th factor of adolescent alcohol use is regressed on the intercept factor of peer alcohol use, and the slope factor of peer alcohol use is regressed on the intercept factor of adolescent alcohol use. This model represents the initial a priori multivariate growth model. Lagrange multipliers are then examined for potential estimation of additional parameters in the prediction of the two slope factors. See MacCallum (1986) and Kaplan (1990) for thorough discussions of post hoc model modifications, and Curran et al. (1997) for further details about the model-building process.

Model 3 was estimated and found to fit the observed data moderately well ($\chi^2(16, N = 363) = 36.9$, $p = .001$, TLI = .97, CFI = .98, RMSEA = .06 with 90CI = .03, .08). Examination of significant Lagrange multipliers ($p < .01$) suggested the addition of two parameters: gender and COA status predicting the adolescent alcohol use slope factor. The inclusion of these two regression parameters were associated with a significant improvement in model fit and the resulting model fit the data well ($\chi^2(14, N = 363) = 25.4$, $p = .03$, TLI = .98, CFI = .98, RMSEA = .04 with 90CI = .01, .08; see Fig. 1.6 for the model diagram and Appendix D for the computer code). Results indicated that older children and children of alcoholics reported both higher initial levels of adolescent alcohol use and higher initial levels of peer alcohol use compared to younger children and children of nonalcoholics. In addition, boys and children of alcoholics reported steeper increases in adolescent alcohol use over time compared to girls and children of nonalcoholics. After the inclusion of the three explanatory variables, there remained large and positive correlations between both the intercept factors and both the slope factors. This suggests that a child's initial status on adolescent alcohol use was quite similar to his or her initial status on peer alcohol use, and a child's rate of change on adolescent alcohol use was quite similar to his or her rate of change on peer alcohol use. These findings provide insight into research question *Q5* and suggest that there is a rather high degree of similarity between individual differences in developmental trajectories of adolescent alcohol use and developmental trajectories of peer alcohol use over the three time points.

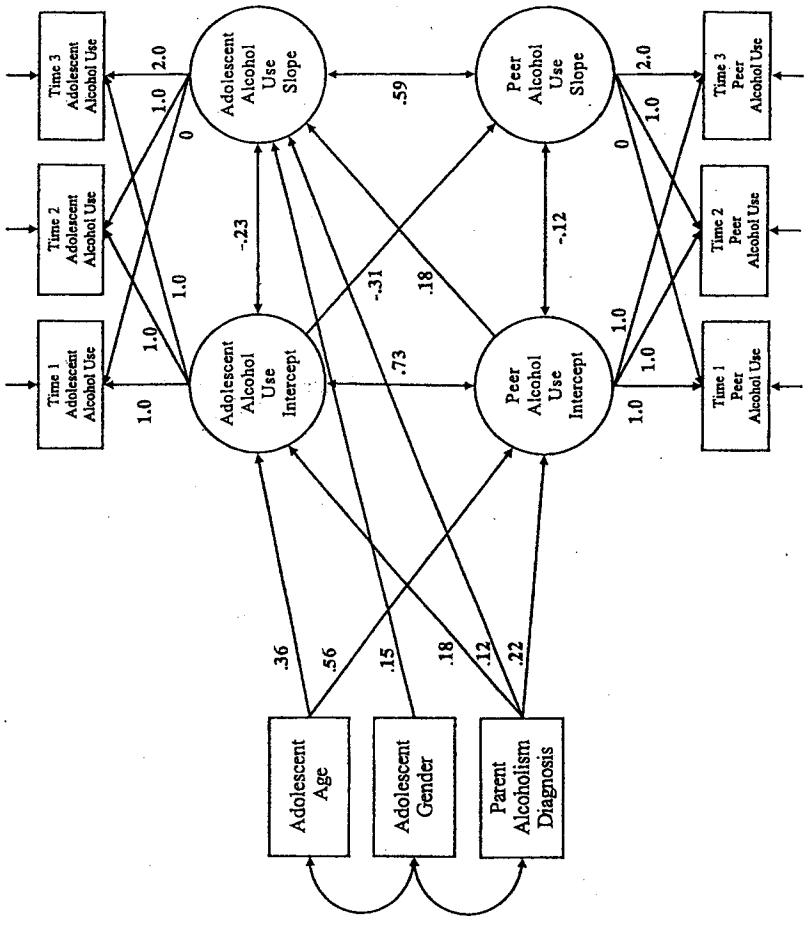

Fig. 1.6. Final multivariate conditional growth model between adolescent alcohol use and peer alcohol use regressed on adolescent age, gender, and parental alcoholism diagnosis.
Note: Model fit was $\chi^2(14, N = 363) = 25.4$, $p = .03$, TLI = .98, CFI = .98, RMSEA = .04. Only significant paths ($p < .05$) are shown. All parameter estimates are standardized. Covariances among the within time disturbances of the repeated measures are not shown.

The regression parameters between the intercept factor of adolescent alcohol use and the slope factor of peer alcohol use, and between the intercept factor of peer alcohol use and the slope factor of adolescent alcohol use allow us to examine the final research question *Q6* that inquired about the relation between earlier levels of one construct predicting later developmental trajectories of the other construct. Both of these regression parameters were found to significantly differ from zero ($p < .05$). The peer alcohol use intercept factor positively predicted the adolescent alcohol use slope factor indicating that higher initial levels of peer alcohol use were associated with steeper growth trajectories in adolescent alcohol use over the following 2 years. In comparison, the adolescent alcohol use intercept factor *negatively* predicted the peer alcohol use slope factor, indicating that higher initial levels of adolescent alcohol use was associated with *less* steep (but still positively increasing) growth trajectories in peer alcohol use over the following 2 years. It is extremely important to note that this negative relation in no way indicates *decreases* in peer alcohol use over time; instead, this implies that higher initial levels of adolescent alcohol use are associated with *smaller* rates of positive change in peer alcohol use.

To better understand these complex relations, it is helpful to probe the model implied growth trajectories on one construct as a function of initial status on the other construct. Conceptually, this is done in much the same way as we might probe a two-way interaction in multiple regression (e.g., Aiken & West, 1991). Although these techniques are well worked out for the regression model, it is not so clear how to best accomplish this for the latent curve model, and further work is needed in this area. The method I used here incorporates the final parameter estimates from the latent curve model to compute the model implied growth trajectories as a function of the predictor variables. The goal here is to compute the mean of the slope factor on one construct as a function of varying values of the intercept factor on the other construct. There is no strict definition for what is meant by varying values, but here I probed this relation using one standard deviation above and below the mean of each intercept factor. That is, I first used the final parameter estimates to provide the average model implied growth trajectories. I then added and subtracted the corresponding standard deviations to the intercepts of the growth factors to provide the model implied growth trajectories as a function of high versus low standing on the initial status factors. Finally, I plotted these means to probe the

nature of the relation among the latent growth factors. These trajectories are shown in Fig. 1.7.

Figure 1.7 reflects that, although all developmental growth trajectories are positive, there are differences in the *magnitudes* of the trajectories for both constructs. Positive growth in adolescent alcohol use was *accelerating* over time as a function of peer alcohol use; that is, higher initial levels of peer use were associated with steeper rates of positive growth in adolescent use. In comparison, positive growth in peer alcohol use was *decelerating* over time as a function of adolescent alcohol use; that is, higher initial levels of adolescent alcohol use were associated with less steep rates of positive growth in peer use. Thus, the initial status of both peer and adolescent alcohol use was predictive of later changes in the other construct, but the *magnitude* of the rate of positive change varied within each construct.

Summary of Findings

The series of latent curve models allowed us to explore all six of our research questions described at the beginning of the chapter. Growth in adolescent alcohol use was characterized by a positive linear trajectory over the three time periods, and there were large individual differences in both the starting point of the trajectory and the rate of change over time (*Q1*). Individual differences in developmental trajectories of adolescent alcohol use were systematically related to age, gender, and parental alcoholism diagnosis (*Q2*). Growth in peer alcohol use was also characterized by a positive linear trajectory with large individual differences in both the starting point of the trajectory and the rate of change over time (*Q3*). Individual differences in developmental trajectories of peer alcohol use were systematically related to age and parental alcoholism, but not gender (*Q4*). Individual differences in developmental trajectories of adolescent alcohol use were closely related to those of peer alcohol use. Results indicated that the intercepts of the two growth processes were strongly and positively correlated with one another, as were the rates of change of the two growth processes, thus reflecting that the characteristics of growth in adolescent alcohol use were similar to the characteristics of growth in peer alcohol use (*Q5*). Finally, higher initial levels of adolescent alcohol use were predictive of smaller positive changes in peer alcohol use, and higher initial levels of peer alcohol use were predictive of larger positive changes in adolescent alcohol use (*Q6*).

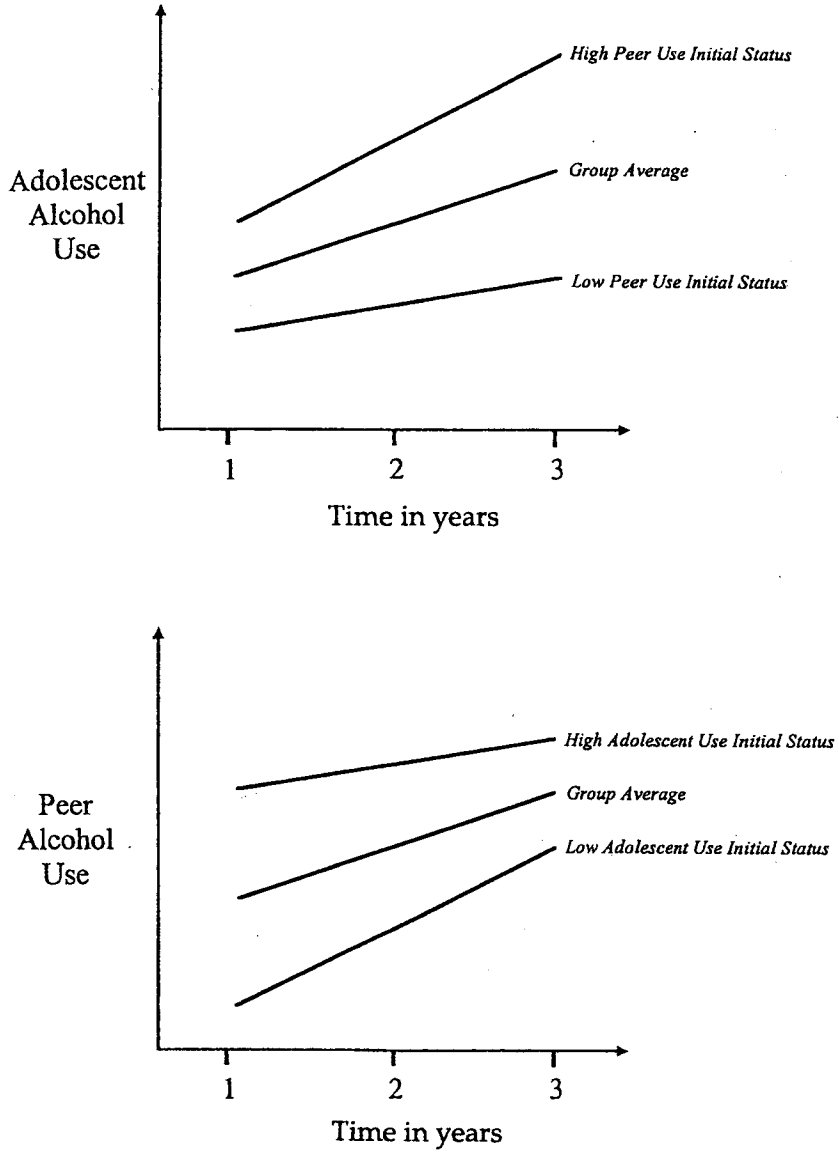

Fig. 1.7. Model implied growth trajectories for adolescent alcohol use as a function of initial status on peer alcohol use, and model implied growth trajectories for peer alcohol use as a function of initial status on adolescent alcohol use.
Note: *High* and *Low* defined as one standard deviation above or below the mean on the initial status growth factor.

Taken together, these results are most consistent with the *bidirectional peer influences* model of adolescent alcohol use, at least for adolescents similar in characteristics to those studied here.

Extensions of the Latent Curve Model

There are many interesting ways in which the latent curve models can be extended to test a variety of types of research hypotheses often encountered in studies of substance use and abuse. For example, whereas I used manifest variables for the repeated measures of alcohol use over time, the model can easily be extended to incorporate multiple indicator latent factors within each time period to estimate and remove the effects of measurement error (McArdle, 1988). The latent curve model can provide formal tests of both mediation and moderation across a variety of research designs (McArdle, 1988, 1989, 1991). The latent curve approach is particularly well suited for evaluating the efficacy of prevention and treatment programs targeted at altering normative developmental trajectories (e.g., deflecting substance use trajectories during adolescence; Curran & Muthén, in press; Muthén & Curran, 1997). Given an adequate number of repeated measures, curvilinear trajectories can be examined (McArdle & Epstein, 1987) as well as alternative functional forms of growth (Browne & Du Toit, 1993). These nonlinear growth models play a particularly important role when studying development in substance use during times of acceleration or deceleration when growth might not follow a linear trajectory. Finally, the latent curve model can be used to provide important estimates of statistical power and effect size to be used in the planning of future longitudinal studies of development and change (Muthén & Curran, 1997). In sum, latent curve analysis is a highly flexible technique that can be adapted to test many different types of questions about individual differences in change over time and is particularly well suited for the empirical study of the development of substance use across the life span.

Limitations of the Latent Curve Model

The previous analyses have highlighted a number of distinct advantages of the latent curve model for studying individual differences in change over time. There are, of course, a number of limitations as well. First, the sample used for the previous analyses was characterized by a 97% retention rate across the three time periods making missing data a

nonissue here. In general, this is not typically the case, especially in longitudinal studies of substance use and high risk behavior, and issues of missing data must be closely considered in analyses of this kind (Seltzer & Raudenbush, 1997). Although recent advances have been made in the incorporation of missing data structures within the structural equation modeling framework (e.g., Graham, Hofer, & MacKinnon, 1996; McArdle, 1994; Muthén, 1993; Muthén, Kaplan, & Hollis, 1987), this still poses a limitation in latent curve analysis. The latent curve model as typically estimated makes the further assumption that all subjects are assessed at the same time periods (so-called *time-structured* data; Willett & Sayer, 1994), and this can be a limitation in many types of substance use research settings. However, this limitation is one of current software implementation, and individually varying assessments can be appropriately modeled in a latent variable framework using certain software packages (e.g., Mx; Neale, 1994). Further, normal theory maximum likelihood (ML) estimation was used for all of the latent curve models here. ML estimation assumes that the observed data follow a multivariate normal distribution (Browne, 1984), and violations of this assumption are associated with both inflated test statistics (Curran, West, & Finch, 1996; Hu, Bentler, & Kano, 1992) and attenuated standard errors (Muthén & Kaplan, 1985, 1992). This is a significant issue in the empirical study of substance use given the severely non-normal distributions commonly encountered in practice. Alternative methods of estimation such as two-staged least-squares (Bollen, 1996) and pseudo maximum likelihood (Arminger & Schoenberg, 1985) that are less affected by non-normality are becoming increasingly available to the applied researcher, but further work is needed to better understand these estimators in applied settings. These limitations should be closely examined when considering whether latent curve analysis is an appropriate statistical model to be used in a given research application.

CONCLUSIONS

There is rarely a situation in which there exists one single statistical model that is the only "correct" choice for evaluating a given research hypothesis. There are a variety of powerful methods currently available to the applied researcher, each of which is characterized by certain advantages and certain disadvantages. The optimal method depends on the specific data characteristics and research hypotheses of the particu-

lar study at hand. The applied researcher will do well to strive to maintain flexibility in the selection of an analytic technique that best meets the needs of the particular research hypothesis. One never wants to be in the position of having a single statistical model in desperate search of a question. The old adage "When you only own a hammer, everything begins to look like a nail" rings especially true here.

It is also extremely important to stress that the selection of an appropriate statistical model is *not* tantamount to the selection of a particular software package. That is, the key analytic decision is not whether to use EQS or LISREL or HLM or PROC MIXED to evaluate the proposed research hypothesis. Instead, the statistical model that best corresponds to the theoretical model must first be selected, and only then will the choice of software logically follow. It is ultimately the responsibility of the individual researcher to ascertain the particular advantages and disadvantages of each of the available analytic techniques, and to make a fully informed decision about the selection of the statistical technique that is best suited to evaluate the given research hypothesis.

I have dedicated a large portion of this chapter to a discussion of matching the statistical model to the theoretical model. However, I also believe that the relation between theory and statistics can be a symbiotic one. That is, the statistical model is not merely a tool with which to empirically evaluate the theoretically derived research hypothesis. Although this is a critical role filled by the statistical model, it can also be used to prompt us to more clearly articulate our developmental theories and associated research hypotheses. Given the ever increasing complexity of the available statistical modeling techniques, there is a corresponding need to pay much greater attention to the specific details of the influences and mechanisms that we postulate in our theories. In the past, predictions about development could often be broadly discussed in general terms of "change," and there was not a pressing need to explicitly articulate precisely what *type* of change the theory was inferring. However, given that the selection of an appropriate statistical model depends directly on the type of change that we hypothesize to exist, we are in turn obligated to clearly enunciate these theoretical details to optimally guide our statistical analyses. The subsequent result of this increased focus of attention on the selection of an appropriate statistical model is the strengthening of both our developmental theories and our corresponding research hypotheses.

Appendix A

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/title
autoregressive crosslagged panel model for adolescent alcohol use and
peer alcohol use with age, gender and parental alcoholism as predictors
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v7=peer1; v8=peer2; v9=peer3;
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v5= *v4 + *v7 + e5;
v6= *v5 + *v8 + e6;
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v9 = *v8 + *v5 + e9 + *v2;
/variances
v1 to v3=*; e4 to e9=*;
/covariances
v3,v2=.003*; v3,v1=-.1*; v2,v1=-.04*;
e7,e4=.020*; e8,e5=.773*; e9,e6=.133*;
/matrix
1.968
-0.022  0.250
-0.104  0.001  0.246
1.238   0.019  0.164  7.906
1.750   0.110  0.331  7.634  15.828
1.532   0.239  0.478  6.726  12.917  23.013
1.179  -0.081  0.078  3.246   4.021   3.853  3.047
1.118  -0.022  0.107  2.691   4.749   4.555  2.077  3.321
0.822  -0.132  0.159  2.147   4.055   5.849  1.691  2.126  4.039
/end
```

Appendix B

```
/title
two factor unconditional latent curve model for
three repeated measures of adolescent alcohol use
/specifications
cases=363; variables=9; method=ml; matrix=cov; analysis=moment;
/labels
v1=age; v2=gen; v3=coa; v4=alc1; v5=alc2; v6=alc3;
v7=peer1; v8=peer2; v9=peer3;
```

```
f1=alcint; f2=alcslp;
/equations
v4= 1.0f1 + 0f2 + e4;
v5= 1.0f1 + 1f2 + e5;
v6= 1.0f1 + 2f2 + e6;
f1= *v999 + d1;
f2= *v999 + d2;
/variances
e4 to e6=*;
d1 to d2=*;
/covariances
d2,d1=*;
/means
12.9146 0.5152 0.5647 1.3636 2.1185 3.1846 1.3289 1.7543 2.4749
/matrix
1.968
-0.022 0.250
-0.104 0.001 0.246
1.238 0.019 0.164 7.906
1.750 0.110 0.331 7.634 15.828
1.532 0.239 0.478 6.726 12.917 23.013
1.179 -0.081 0.078 3.246 4.021 3.853 3.047
1.118 -0.022 0.107 2.691 4.749 4.555 2.077 3.321
0.822 -0.132 0.159 2.147 4.055 5.849 1.691 2.126 4.039
/end
```

Appendix C

```
/title
two factor conditional latent curve model for adolescent alcohol use
regressed on age, gender, and parent alcoholism diagnosis
/specifications
cases=363; variables=9; method=ml; matrix=cov; analysis=moment;
/labels
```



```
v1=age; v2=gen; v3=coa; v4=alc1; v5=alc2; v6=alc3;
v7=peer1; v8=peer2; v9=peer3;
f1=alcint; f2=alcslp;
/equations
v1= *v999 + e1;
v2= *v999 + e2;
v3= *v999 + e3;
v4= 1.0f1 + 0f2 + e4;
v5= 1.0f1 + 1f2 + e5;
v6= 1.0f1 + 2f2 + e6;
f1= *v999 + *v1 + *v2 + *v3 + d1;
f2= *v999 + *v1 + *v2 + *v3 + d2;
/variances
e1 to e6=*; d1 to d2=*;
/covariances
e3,e2=*; e3,e1=*; e2,e1=*; d2,d1=*;
/means
12.9146 0.5152 0.5647 1.3636 2.1185 3.1846 1.3289 1.7543 2.4749
/matrix
1.968
-0.022 0.250
-0.104 0.001 0.246
1.238 0.019 0.164 7.906
1.750 0.110 0.331 7.634 15.828
1.532 0.239 0.478 6.726 12.917 23.013
1.179 -0.081 0.078 3.246 4.021 3.853 3.047
1.118 -0.022 0.107 2.691 4.749 4.555 2.077 3.321
0.822 -0.132 0.159 2.147 4.055 5.849 1.691 2.126 4.039
/end0
```

Appendix D

```
/title
multivariate growth model between adolescent and peer alcohol use
regressed on age, gender, and parent alcoholism diagnosis
/specifications
cases=363; variables=9; method=ml; matrix=cov; analysis=moment;
/labels
```

```
v1=age; v2=gen; v3=coa; v4=alc1; v5=alc2; v6=alc3;
v7=peer1; v8=peer2; v9=peer3;
f1=alcint; f2=alcslp; f3=peerint; f4=peerslp;
/equations
v1= 12.9*v999 + e1;
v2= .515*v999 + e2;
v3= .554*v999 + e3;
v4= 1.0f1 + 0f2 + e4;
v5= 1.0f1 + 1f2 + e5;
v6= 1.0f1 + 2f2 + e6;
v7= 1.0f3 + 0f4 + e7;
v8= 1.0f3 + 1f4 + e8;
v9= 1.0f3 + 2f4 + e9;
f1= -7*v999 + .5*v1 + .5*v2 + .5*v3 + d1;
f2= -.14*v999 + *v2 + *v3 + *f3 + d2;
f3= -6.6*v999 + .5*v1 + .5*v2 + .5*v3 + d3;
f4= .5*v999 + *f1 + d4;
/variances
e1 to e9=*; d1 to d4=*;
/covariances
e3,e2=0*; e3,e1=0*; e2,e1=0*; e7,e4=.5*; e8,e5=.5*; e9,e6=.5*;
d2,d1=*; d4,d3=*; d4,d2=*; d3,d1=*;
/means
12.9146 0.5152 0.5647 1.3636 2.1185 3.1846 1.3289 1.7543 2.4749
/matrix
1.968
-0.022 0.250
-0.104 0.001 0.246
1.238 0.019 0.164 7.906
1.750 0.110 0.331 7.634 15.828
1.532 0.239 0.478 6.726 12.917 23.013
1.179 -0.081 0.078 3.246 4.021 3.853 3.047
1.118 -0.022 0.107 2.691 4.749 4.555 2.077 3.321
0.822 -0.132 0.159 2.147 4.055 5.849 1.691 2.126 4.039
/end
```

NOTES

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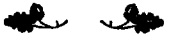

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to our colleagues in quantitative methods
for showing us the way;
to NICHD and NIDA
for supporting the journey;
and to our families
for allowing us the time to make the trip.

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