The Relation Between Adolescent Alcohol Use and Peer Alcohol Use: A Longitudinal Random Coefficients Model

Patrick J. Curran
Duke University

Eric Stice
Stanford University

Laurie Chassin
Arizona State University

Longitudinal latent growth models were used to examine the relation between changes in adolescent alcohol use and changes in peer alcohol use over a 3-year period in a community-based sample of 363 Hispanic and Caucasian adolescents. Both adolescent alcohol use and peer alcohol use were characterized by positive linear growth over time. Not only were changes in adolescent alcohol use closely related to changes in peer alcohol use, but the initial status on peer alcohol use was predictive of later increases in adolescent alcohol use and the initial status on adolescent alcohol use was predictive of later increases in peer alcohol use. These results are inconsistent with models positing solely unidirectional effects between adolescent alcohol use and peer alcohol use.

Of the variables found to be related to adolescent substance use, peer substance use is consistently one of the strongest predictors (Brook, Brook, Gordon, Whiteman, & Cohen, 1990; Hawkins, Catalano, & Miller, 1992; Newcomb & Bentler, 1986). 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); that is, adolescents who affiliate with substance-using friends are more likely to use substances themselves. However, an alternative explanation is that adolescents who initiate illicit substance use seek out a peer group that more closely matches their newly acquired behavior and attitudes (Farrell, 1994; Farrell & Danish, 1993). A third possibility is that a combination of these two processes exists 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).

Another alternative that has received much less attention is that the observed relation between peer substance use and ado-lescent substance use might be attributable to a third variable. Any potential third variable must be one that could theoretically account for both peer substance use and adolescent substance use. Although there are many plausible third variable correlates, one candidate is the adolescents' general tendencies toward non-conformity or rebelliousness. These characteristics have been shown to be related to both adolescent substance use and affiliations with substance-using peers (Brook, Gordon, & Whiteman, 1985; Brook, Whiteman, Gordon, & Cohen, 1989; Kandel, 1978; Stein, Newcomb, & Bentler, 1987). Adolescents who are particularly rebellious may be both more likely to select friends who use alcohol or drugs and also use these substances themselves. Thus, the observed relation between adolescent substance use and peer substance use might not be attributable to bidirectional influences between these two constructs but instead might be due to the shared influence of adolescent rebelliousness. If so, the relation between adolescent use and peer use will disappear when the effects of rebelliousness are controlled.

There are several reasons why it is important to better understand the nature of the relation between peer substance use and adolescent substance use. Most centrally, given the strong observed association between peer and adolescent substance use, peer group influences are leading prospects for manipulation in programs aimed at delaying the onset or escalation of adolescent substance use. If earlier peer group affiliation is predictive of later adolescent substance use (peer *socialization*), then the peer group is an important point to focus intervention efforts. However, if the direction of influence is reversed and adolescents first begin to use illicit substances and subsequently affiliate with substance-using peers (peer *selection*), then interventions focused on the peer group would be misplaced. Similarly, if the observed relation between peer use and adolescent use is due to a third variable cause, then attempting to manipulate peer influences would be futile.

Previous research has identified both cross-sectional (Kandel & Andrews, 1987; Pruitt, Kingery, Mirzaee, Heuberger, & Hurley, 1991; Swaim et al., 1989) and longitudinal (Bailey & Hubbard, 1991; Brook, Whiteman, Gordon, Nomura, & Brook, 1986; Duncan, Duncan, & Hops, 1994; Ellickson & Hays, 1991) support for the prediction of adolescent substance use from peer influences. In addition, several studies have examined the possible longitudinal reciprocal relations between these two constructs (Downs, 1987; Farrell, 1994; Farrell & Danish, 1993; Fisher & Bauman, 1988; Kandel, 1978; Stein et al., 1987). However, many of these studies have resulted in contradictory findings. For example, Fisher and Bauman (1988) found prospective support for both peer influence and peer selection processes for both adolescent alcohol use and smoking (in separate samples) and for both adolescent self-reported and peer-reported substance use. In contrast, Stein et al. (1987) failed to find any prospective relations between adolescent use and peer use over time.

Recent studies provided additional insight. Swaim et al. (1989) studied the cross-sectional relations between emotional distress, peer drug associations, and adolescent drug use and tested the *peer cluster theory* proposed by Oetting and Beauvais (1986, 1987). Peer cluster theory postulates that friend dyads or small peer subgroups (clusters) are the salient proximal link between more distal causal influences and adolescent substance use. Adolescent psychological characteristics, behaviors, attitudes, and socialization may all exert an influence on subsequent substance use, but these effects are thought to be primarily, if not solely, mediated through the peer cluster. Consistent with peer cluster theory, Swaim et al. (1989) found that peer substance use strongly predicted adolescent substance use. Additionally, peer use partially mediated the effects of emotional distress on adolescent drug use. Although intriguing, the cross-sectional nature of this study precludes any definitive statements about the direction of influence.

Farrell and Danish (1993) presented a rigorous longitudinal test of the relation between peer influences and adolescent drug use. Data were collected at three time points over an 18-month period from a large sample of middle-school students. Three competing models of drug use were tested across gender using manifest variable longitudinal structural equation modeling. Whereas adolescent drug use prospectively predicted later peer drug use, neither peer drug use nor peer pressure to use drugs prospectively predicted later adolescent drug use. Farrell and Danish (1993) concluded that "it appears that changes in the frequency of drug use precede changes in peer variables rather than vice-versa" (p. 332). Using this same sample, Farrell (1994) focused on the relations among anger, peer alcohol use, and adolescent alcohol use. Consistent with their previous findings, earlier peer alcohol use did not predict later adolescent alcohol use, whereas earlier adolescent alcohol use did predict later peer alcohol use.

Although the studies of Farrell and Danish (Farrell, 1994; Farrell & Danish, 1993) provide rather strong evidence against peer cluster theory, there are several key issues that should be considered. First, the Farrell and Danish (1993) sample was composed primarily of African Americans, and peer influences have been reported to be weaker for this ethnic group (Brannock, Schandler, & Oncley, 1990; Newcomb & Bentler, 1986). A sec-

ond issue is that the analyses of Farrell and Danish (1993) and Farrell (1994) consisted of traditional fixed-effects autoregressive (AR) structural equation models. Examination of the univariate means and variances reported in these studies suggest that peer and adolescent drug and alcohol use were growing over time (as evidenced by increasing means and increasing variances in both constructs over all three time points). Because traditional fixed-effects AR models are based on the between-wave covariance matrix and do not explicitly model the mean structure of the data, no information is provided about growth or individual differences in growth over time (Rogosa, 1987, 1988). AR models have thus been criticized when applied to the study of longitudinal processes that are systematically growing over time (Rogosa, 1987, 1988; Rogosa & Willett, 1985). Failing to consider these individual differences in growth does not take full advantage of all of the information available in the data and may also bias model parameters, which may lead to potentially misleading conclusions (Curran, Harford, & Muthén, 1996).

A recently emerging analytic technique that explicitly models growth and individual differences in growth over time is random coefficients latent growth (LG) modeling (McArdle & Epstein, 1987; Meredith & Tisak, 1984, 1990; Muthén, 1991, 1993; see also the 1994 special section, "Structural equation modeling in clinical research," of the *Journal of Consulting and Clinical Psychology*). LG models combine elements of repeated measures multivariate analysis of variance (MANOVA), confirmatory factor analysis, and structural equation modeling to analyze changes in a construct over time. LG models consider both the between-wave covariance matrix and the observed mean structure so that group growth parameters and individual variation in growth can be examined. Thus, random coefficient models offer many advantages over more traditional fixed-effects techniques for examining changes in adolescent and peer alcohol use over time.

The present study is guided by three primary goals. First, competing theoretical models are tested that examine the unidirectional and bidirectional relations between peer alcohol use and adolescent alcohol use over time. Second, few previous studies have explicitly investigated potential third variable influences that might explain the observed relation between peer and adolescent substance use. Accordingly, the present study incorporates a measure of adolescent rebelliousness to test whether this construct accounts for the relation between peer alcohol use and adolescent alcohol use. Finally, nearly all previous studies have used fixed-effects statistical models that are known to have certain limitations when applied to the analysis of processes that are systematically growing over time. The present study incorporates latent variable random coefficient growth models that are thought to be better suited for the examination of individual differences in growth and correlates of change. To further understand the potential differences in fixed- and random-effects models, a traditional fixed-effects model was estimated using the same data and compared with the final LG model.

Method

Participants

The total sample at Time 1 consisted of 454 adolescents aged 10.5 to 15.5 years ($M = 12.7$, $SD = 1.45$) and their parents who were

participants in a longitudinal study of adolescent substance use. Children of alcoholics (COAs; $n = 246$) had at least one biological and custodial alcoholic parent, and controls ($n = 208$) had no biological or custodial alcoholic parents. Of these 454 adolescents, 449 (99%) were interviewed at Time 2 and 445 (98%) were interviewed at Time 3. Of the 442 families with complete data across all three time points (on the constructs of interest to the present study), 74 families were dropped because the child reported no substance use and no peer use at any of the three time waves.[1] Initial analyses identified a small but influential subgroup of adolescents ($n = 5$) who reported extremely high Time 1 alcohol use and steep decreases in use over time. These participants reported alcohol use greater than 4 SD above the mean at Time 1 and reported subsequent decreases greater than 1 SD at each time point. Inclusion of these outliers resulted in unstable estimation of the LG models. Although an important subgroup, these five outlying cases were excluded from subsequent analyses because of the undue influence on the overall group growth parameters. The final sample used for the present analyses consisted of 363 families. The average age at Time 1 was 12.9 years, 56% were COAs, 48% were female, 25% were Hispanic, and 75% were Caucasian.

Recruitment Procedures

COA families were recruited using court records (full sample = 103), wellness questionnaires from a health maintenance organization (full sample = 22), and community telephone surveys (full sample = 120). Screening and recruitment were done by research team members (or by participating agencies when required because of confidentiality concerns). COAs had to meet the following criteria: Anglo or Hispanic ethnicity, Arizona residency, ages 10.5–15.5 years, English speaking, and having no cognitive limitations that would preclude interview (e.g., severe mental retardation or psychosis). Finally, direct interview data had to confirm that a biological and custodial parent met criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed.; *DSM–III;* American Psychiatric Association, 1980) for lifetime alcohol abuse or dependence diagnoses using the Diagnostic Interview Schedule, Version III (DIS; Robins, Helzer, Croughan & Ratcliff, 1981) or spousal report on the Family History Research Diagnostic Criteria, Version III (FH-RDC; Endicott, Andreason, & Spitzer, 1978) if the second parent was not interviewed. Demographically matched control families were recruited using telephone interviews. When a COA participant was recruited, reverse directories were used to locate families living in the same neighborhood. Families were screened to match the COA participant on ethnicity, family composition, child's age (within 1 year), and socioeconomic status (SES, using the property value code from the reverse directory). Direct interviews were used to confirm that neither biological nor custodial parents met *DSM–III* criteria for lifetime diagnoses of alcohol abuse or dependence. Recruitment biases because of selective contact with participants or participant refusals were minimal and are discussed in detail elsewhere, along with additional information about recruitment procedures (Chassin, Barrera, Bech, & Kossak, 1992; Chassin, Rogosch, & Barrera, 1991).

Data were collected through computer-assisted interviews with the adolescents and their parents at the family's home or the university research center. Trained interviewers read each item aloud, and all close-ended responses were entered directly into the computer. Family members were interviewed individually on the same occasion by different interviewers. Family members were interviewed in separate rooms when possible, and participants had the option of entering their responses directly on the computer keyboard to avoid verbal responses.

Measures

Demographic variables. Demographic variables were child age at Time 1, child gender (male, coded 1; female, coded 0) and child ethnicity

(Hispanic, coded 1; Caucasian, coded 0). A child was considered Hispanic if the child reported his or her own ethnicity as Hispanic and at least 1 parent reported their own ethnicity as Hispanic.

Parent alcoholism. Lifetime *DSM–III* diagnoses of alcohol abuse or dependence were obtained using a computerized version of the Diagnostic Interview Schedule (DIS, Version III; Robins, Helzer, Croughan, & Ratcliff, 1981). If one of the two parents was not directly interviewed (5% of mothers and 22% of fathers in the total sample), the interviewed parent reported on the noninterviewed parent using the FH-RDC. Parent alcoholism was a dichotomous variable reflecting the presence (coded 1) or absence (coded 0) of diagnosis in either or both parents.

Peer alcohol use. 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 using items adopted from the Monitoring the Future Study (Johnston, O'Malley, & Bachman, 1991). Response options ranged from 0 (*none*) to 5 (*all*). These two items were strongly correlated both within time (rs ranged from .77 to .80) and across time (rs ranged from .69 to .71), and within-time coefficient alphas ranged from .85 to .87. A single peer alcohol use score was calculated by summing the two items.

Adolescent alcohol use. Adolescents self-reported their frequency of consumption of beer–wine and hard liquor (two items), frequency of consumption of five or more drinks in a row (one item), and frequency of getting drunk (one item) in the past 12 months. Response options ranged from 0 (*not at all*) to 7 (*every day*). The four adolescent alcohol use items were strongly correlated both within time (rs ranged from .63 to .82) and across time (rs ranged from .56 to .75), and within-time coefficient alphas ranged from .86 to .91. A single alcohol use score was calculated by summing the four items.

Adolescent rebelliousness. Adolescent rebelliousness was measured using eight items developed by Smith and Fogg (1979). Adolescents endorsed a series of statements describing themselves (e.g., "Sometimes I enjoy seeing how much I can get away with" and "I feel guilty when I break a rule" [reverse coded]). Response options ranged from 1 (*strongly disagree*) to 5 (*strongly agree*). The eight adolescent rebellious items were strongly correlated both within time (rs ranged from .59 to .79) and across time (rs ranged from .64 to .75), and within-time coefficient alphas ranged from .79 to .81. A single rebelliousness score was calculated by summing the eight items.

Results

Table 1 presents the means, standard deviations, zero-order correlations and univariate skewness coefficients for the four predictors and the Time 1, Time 2, and Time 3 measures of adolescent alcohol use and peer alcohol use. Table 2 presents the means and standard deviations of adolescent alcohol use and peer alcohol use as a function of adolescent age and gender. Both the observed means and variances of adolescent alcohol use and peer alcohol use were increasing over time. Additionally, adolescent alcohol use and peer alcohol use were strongly positively correlated both within and across

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[1] These complete abstainers were not included in the analyses because of the focus on studying change over time in adolescent and peer alcohol use, and this subgroup of adolescents reported no use at any time point. However, all models were reestimated on the full sample including the abstainers. Although there were mean differences in adolescent and peer alcohol use, there were no differences in the pattern of findings between the two groups.

Table 1

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

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Time 1: Adolescent alcohol use	—										
2. Time 2: Adolescent alcohol use	.68	—									
3. Time 3: Adolescent alcohol use	.50	.68	—								
4. Time 1: Peer alcohol use	.66	.58	.46	—							
5. Time 2: Peer alcohol use	.53	.65	.52	.65	—						
6. Time 3: Peer alcohol use	.38	.51	.61	.48	.58	—					
7. Time 1: Rebelliousness	.30	.29	.32	.33	.31	.26	—				
8. Age	.31	.31	.23	.48	.44	.29	.14	—			
9. Gender	.01	.06	.10	−.09	−.02	−.13	.14	−.03	—		
10. COA status	.12	.17	.20	.09	.12	.16	.12	−.15	.00	—	
11. Ethnicity	.01	.00	−.05	.07	.05	.07	.00	.03	−.04	.06	—
M	1.36	2.12	3.18	1.33	1.75	2.48	2.58	12.91	0.52	0.56	0.24
SD	2.81	3.98	4.79	1.74	1.82	2.01	0.69	1.40	0.50	0.49	0.43
Skewness	3.23	2.73	2.07	1.57	1.16	0.66	0.01	−0.28	−0.06	−0.26	1.22

Note. Absolute values of correlations greater than $r = .19$ represent a per-test $p < .002$ and a familywise $p < .10$. Statistics are based on $n = 363$. COA = children of alcoholics.

waves of measurement. Age was positively correlated with both adolescent and peer alcohol use, indicating that older participants reported higher levels of alcohol and peer use. Because ethnicity was not found to be significantly related to any measure at any time point, this variable was not included in further analyses.

Examination of Growth Over Time

LG models were used to study change in the constructs over time (McArdle & Epstein, 1987; Meredith & Tisak, 1984, 1990; Muthén, 1991, 1993). Whereas hierarchical linear models (HLM) are regression-based multilevel analyses (Bryk & Raudenbush, 1992), LG 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). All LG models were estimated using EQS (Bentler, 1989) based on the observed covariance matrix and column vector of means. The first step in the LG analyses was to test for the presence of change in adolescent alcohol use and peer alcohol use over the three yearly assessments. Two LG models were estimated, one for adolescent alcohol use and one for peer alcohol use. The basic LG model comprises two latent

factors, with the repeated measures of the construct over time as the indicators (see Figure 1). Conceptually, this model can be viewed as a confirmatory factor-analytic model. 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 reflects the variation of each individual around the overall group growth parameters. The estimation of variability in individual change over time makes this a random coefficients model.

Adolescent alcohol use. A two-factor LG model as described above was estimated for the three repeated measures of adolescent alcohol use. This model was found to fit the observed data well, $\chi^2(1, N = 363) = 2.1, p = .15$; TLI = .99 (Tucker–Lewis fit index; TLI; Tucker & Lewis, 1973) = .99; comparative fit index (CFI; Bentler, 1990) = .99. A significant positive mean for the slope factor ($\mu = .88$) indicated that the overall group reported increases in adolescent alcohol use over time. The equally spaced factor loadings (0, 1, 2) reflected that this in-

Table 2

Observed Means (and Standard Deviations) of Adolescent and Peer Alcohol Use as a Function of Age and Gender

| Variable	Total ($N = 363$)	Ages 10–11 ($n = 66$)	Ages 12–13 ($n = 160$)	Ages 14–15 ($n = 137$)	Male ($n = 187$)	Female ($n = 176$)
Time 1 adolescent use	1.36 (2.81)	0.29 (0.67)	1.05 (2.09)	2.25 (3.77)	1.40 (3.08)	1.32 (2.50)
Time 2 adolescent use	2.12 (3.98)	0.65 (1.86)	1.53 (3.03)	3.52 (5.13)	2.33 (4.51)	1.89 (3.32)
Time 3 adolescent use	3.18 (4.79)	1.85 (4.16)	2.78 (4.22)	4.31 (5.46)	3.65 (5.33)	2.69 (4.12)
Time 1 peer use	1.33 (1.74)	0.34 (0.80)	0.95 (1.32)	2.25 (2.07)	1.17 (1.73)	1.49 (1.75)
Time 2 peer use	1.75 (1.82)	0.69 (1.11)	1.45 (1.59)	2.63 (1.96)	1.71 (1.89)	1.80 (1.76)
Time 3 peer use	2.48 (2.01)	2.37 (1.92)	2.30 (1.87)	3.04 (2.07)	2.22 (2.06)	2.75 (1.92)
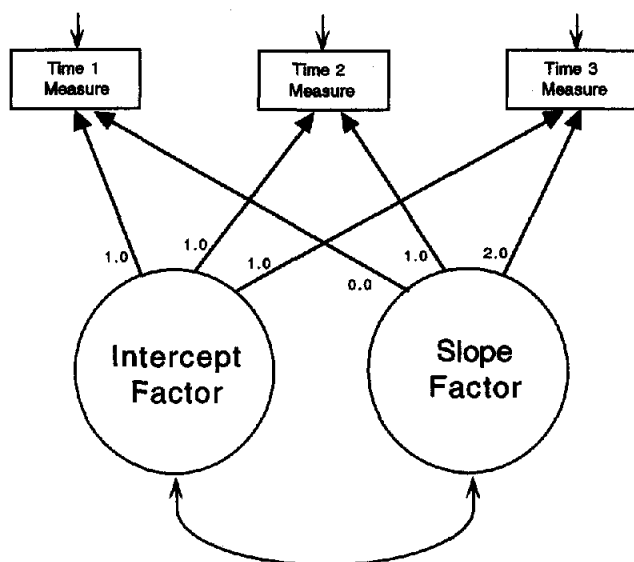
Figure 1. Basic two-factor, three time point latent growth model.

crease was linear.[2] A significant variance component in both the intercept (ψ = 7.9) and the slope (ψ = 3.2) factors indicated that there were significant individual differences in both initial levels and growth in adolescent alcohol use over time. Finally, a marginally significant negative correlation between the intercept and slope factors ($r = -.11, p < .10$) indicated that there was an inverse relation between initial status and change over time (i.e., individuals who reported lower levels of alcohol use at Time 1 tended to report steeper increases in use over time).

Peer alcohol use. A second two-factor LG model was estimated for the three repeated measures of peer alcohol use, and this model also fit the observed data well, $\chi^2(1, N = 363) = 4.52, p = .03$; TLI = .97; CFI = .99. The results for peer alcohol use were similar to those found for adolescent alcohol use. A significant positive mean for the slope factor ($\mu = .55$) indicated increasing trajectories in peer alcohol use over time. The equally spaced factor loadings (0, 1, 2) reflected that this increase was linear (see Footnote 2). A significant variance component in both the intercept (ψ = 2.5) and the slope (ψ = .40) factors indicated that there were significant individual differences in both initial levels and growth over time in peer alcohol use. Finally, a significant negative correlation between the intercept and slope factors ($r = -.38$) indicated that there was an inverse relation between initial status and change over time.[3]

Combined LG Models

The two LG models presented above indicated that there was positive linear growth in both adolescent and peer alcohol use and there were significant individual differences in growth over time. To further explore the individual variation around the group growth curves, the LG models for adolescent alcohol use and peer alcohol use were estimated simultaneously and regressed on adolescent age, gender, and COA status.

Hypothesized LG model. An initial a priori model was esti-

mated so that age, gender, and COA status predicted both the intercept and slope factors for adolescent alcohol use and peer alcohol use. In addition, correlations were estimated between the adolescent and peer alcohol use intercept factors and between the adolescent and peer alcohol use slope factors, as well as correlations between the residuals of the repeated measures within the three time periods between the two constructs (e.g., the Time 1 adolescent alcohol use residual was correlated with Time 1 peer alcohol use residual). Finally, structural parameters were estimated so that the intercept factor of adolescent alcohol use predicted the slope factor of peer alcohol use and the intercept factor of peer alcohol use predicted the slope factor of adolescent alcohol use. These structural paths between the intercept and slope factors represent longitudinal prospective prediction over time and test whether earlier information about one construct is predictive of later changes in the other construct. This a priori model was estimated and fit the observed data well, $\chi^2(16, N = 363) = 36.9, p = .002$; TLI = .96; CFI = .98. Lagrange multipliers were examined to determine whether there were any model misspecifications related to the control variables. This was done because if the model is misspecified, the tests of specific path coefficients may be incorrect (see e.g., MacCallum, 1986). Although this procedure capitalizes on chance in estimating the effects of the control variables, it provides a stringent test of the theoretical variables of interest. Based on the Lagrange multiplier statistics ($p < .01$), two paths were freely estimated: COA status and gender were allowed to predict the adolescent alcohol use slope factor. There were no remaining indications of significant model misspecification.

Final LG model. The final model fit the observed data well, $\chi^2(14, N = 363) = 25.4, p = .03$; TLI = .98; CFI = .99 and is presented in Figure 2.[4] Older children reported significantly ($p < .05$) higher initial levels of both adolescent alcohol use and peer alcohol use, but age was not associated with change over time in either of these constructs. COAs reported higher initial levels of alcohol use and peer alcohol use, and both male

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[2] A nonsignificant nested chi-square test indicated that the third factor loading could be fixed to 2.0 to reflect linear change in both constructs over time.

[3] The present sample size was not large enough to compare the full LG models across gender. However, simplified multiple-group LG models were estimated to test for gender differences in the shape of adolescent and peer alcohol use growth trajectories. Nested chi-square tests indicated that the functional form of growth over time in adolescent alcohol use and peer alcohol use was linear for both male and female participants. Further research with larger samples is needed to examine potential gender differences in the predictors of these similarly shaped growth trajectories.

[4] There was a concern that the generalizability of these findings might be limited due to the oversampling of COA families. To further examine this possibility, the final model was reestimated using a smaller subsample ($n = 240$) selected so that the prevalence of parent alcoholism reflected a normative random community sample that does not oversample COAs (see Colder, 1990, for further details). Although this sample size is too small, given the model complexity to draw firm conclusions, the bidirectional prospective relations were also found in this more normative subsample. This suggests that the bidirectional effects were not due to the high-risk nature of the sample.
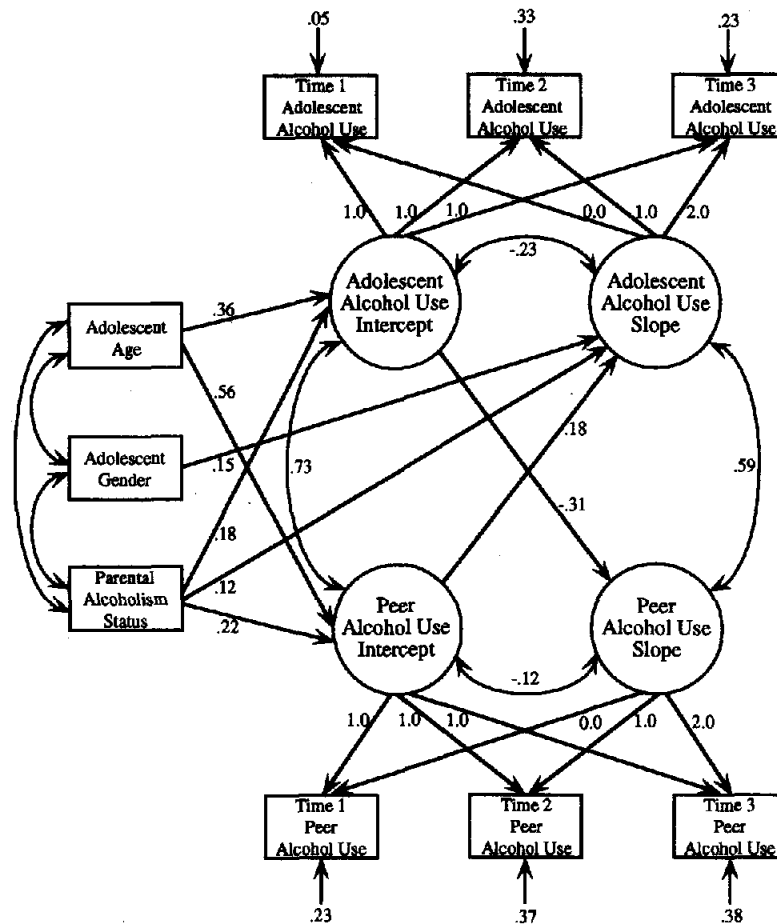
Figure 2. Final latent growth model of adolescent and peer alcohol use. Final model, $\chi^2(14, N = 363)$ = 25.4, p = .03. All parameter values are standardized with the exception of the fixed factor loadings. All parameters shown are $p < .05$.

participants and COAs reported steeper increases in alcohol use over time. Of key interest is the bidirectional prospective prediction of changes on one construct as a function of initial status on the other construct. Initial status with respect to adolescent alcohol use was negatively predictive of changes in peer alcohol use over time, indicating that, although the entire group was increasing in peer alcohol use over time, adolescents who reported lower Time 1 alcohol use tended to increase at a steeper rate in peer alcohol use, compared with those reporting higher Time 1 alcohol use. In comparison, initial status with respect to peer alcohol use was positively predictive of changes in adolescent alcohol use over time, indicating that, although the entire group was increasing in adolescent alcohol use over time, adolescents who reported higher Time 1 peer alcohol use tended to increase at a steeper rate in adolescent alcohol use, compared with adolescents who reported lower Time 1 peer alcohol use.

It is important to note that the negative relation between the initial status of adolescent alcohol use and later change in peer use does not mean that the group experienced a decrease in peer alcohol use over time; rather, the findings indicate that higher levels of initial adolescent alcohol use were associated with

smaller rates of positive growth in peer alcohol use over the three time points and that lower levels of initial adolescent alcohol use were associated with steeper rates of positive growth in peer alcohol use over time. This relation was probed by plotting the model-implied growth trajectories of each construct as a function of initial status on the second construct (1 *SD* above and below the mean of initial status; see Figure 3). It can be seen that, although all growth curves were positive, growth in adolescent alcohol use was accelerating as a function of peer alcohol use whereas growth in peer-alcohol use was decelerating as a function of adolescent alcohol 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 differed within each construct.

Inclusion of rebelliousness. The previous model suggested that not only were changes in adolescent alcohol use and changes in peer alcohol use closely related to one another but the initial status on one construct prospectively predicted subsequent changes on the second construct. To test for a possible third variable influence that accounted for the prospective relation between these two variables, we reestimated the final model (presented in Figure 2)
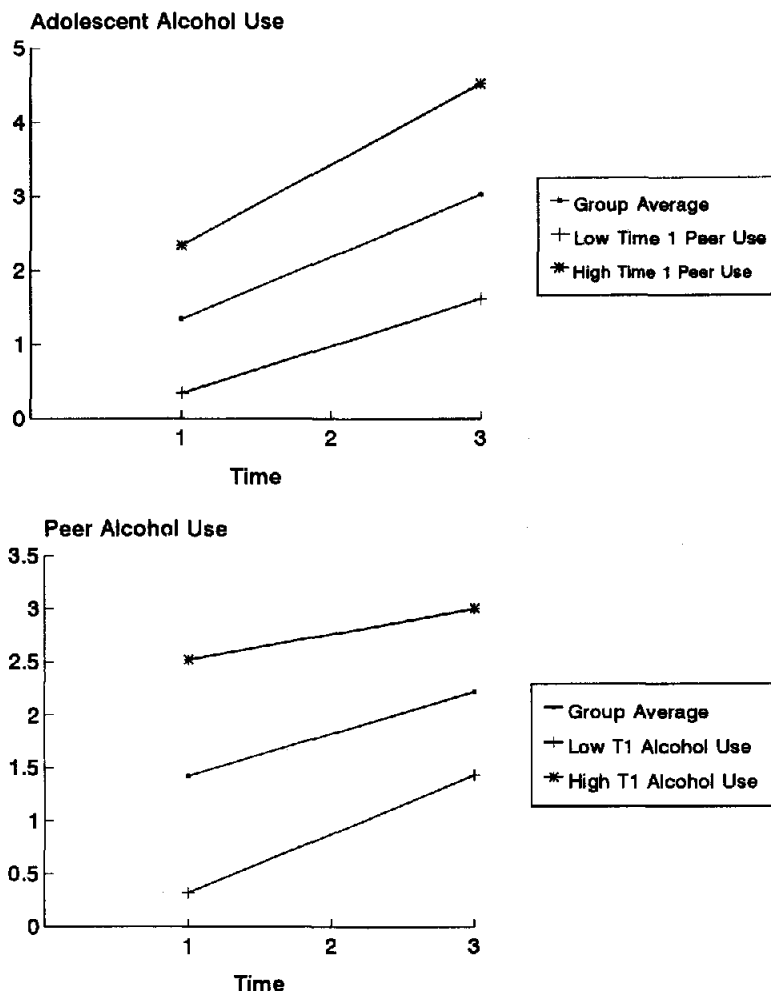
Adolescent Alcohol Use

Peer Alcohol Use

Figure 3. Model implied growth trajectories as a function of high, medium, and low initial levels of peer alcohol use (top graph) and adolescent alcohol use (bottom graph). Low = 1 SD below the mean; High = 1 SD above the mean.

with the inclusion of the Time 1 manifest variable measure of rebelliousness as an exogenous predictor.[5] The model was estimated exactly as described before, with the exception that Time 1 rebelliousness was added as a fourth predictor variable. This model also fit the data well, $\chi^2(18, N = 363) = 29.1, p = .05$; TLI = .98; CFI = .99. Time 1 rebelliousness was significantly and positively related to the two intercept factors such that adolescents reporting higher levels of Time 1 rebelliousness also reported higher initial levels of alcohol use and peer alcohol use. However, Time 1 rebelliousness was not related to either slope factor, and the prospective relations between the intercept and slope factors for adolescent alcohol use and peer alcohol use remained strong and significant even in the presence of rebelliousness. This indicates that rebelliousness did not account for the observed relation between adolescent and peer alcohol use.

AR Cross-Lagged Panel Design

For comparative purposes, we estimated a repeated measures AR cross-lagged panel model (Dwyer, 1983) using these same

data. The same participants and measures were used for the AR model as were used in the LG model (excluding Time 1 rebelliousness). The key difference was that the LG factors were not estimated, and change in the construct over time was modeled using the stability coefficients between time-adjacent measures of adolescent alcohol use and peer alcohol use (e.g.,

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[5] Because preliminary LG models revealed that rebelliousness did not systematically grow over the three time points, only the Time 1 measure was used as an exogenous predictor. In addition, given the ratio of subjects to estimated parameters in the final model, rebelliousness could not be stably estimated as a multiple indicator latent factor. To examine the potential attenuation of the regression coefficients that is due to unmodeled measurement error in the manifest measure of rebelliousness, we reestimated the final model with the measurement error of rebelliousness set to 1 minus coefficient alpha multiplied by the variance of the measure (see Bollen, 1989). There were no changes in the substantive interpretations of the model parameters after having corrected for measurement error in rebelliousness.

Time 1 alcohol use predicted Time 2 alcohol use, and Time 2 alcohol use in turn predicted Time 3 alcohol use).

A baseline model was estimated so that age, gender, and COA status were allowed to predict the Time 1 measures of adolescent alcohol use and peer alcohol use, and time-adjacent stability coefficients were estimated within each construct. This model was estimated and, as expected, fit the data poorly, $\chi^2(23, N = 363) = 412.8$, $p = .000$. Next, the three within-time residual correlations were added, which led to a significant improvement in model fit. Next, the two prospective paths from adolescent alcohol use predicting later peer alcohol use were added (Time 1 predicting Time 2, and Time 2 predicting Time 3), which also led to a significant improvement in model fit. Then the two prospective paths were added from peer alcohol use predicting later adolescent alcohol use (Time 1 predicting Time 2, and Time 2 predicting Time 3), which also led to a significant improvement in model fit. Finally, Lagrange multipliers were consulted to test for possible model misspecifications related to the control variables ($p < .01$), and two paths were added: one from age to Time 2 peer alcohol use, and one from gender to Time 3 peer alcohol use.

The final AR model fit the data well, $\chi^2(14, N = 363) = 29.2$, $p = .01$; TLI = .97; CFI = .99 (see Figure 4). Older adolescents and COAs reported higher Time 1 adolescent alcohol use and higher Time 1 peer alcohol use. Further, older adolescents reported higher Time 2 peer alcohol use and male adolescents reported lower Time 3 peer alcohol use. Of greatest interest were the large and significant bidirectional positive prospective paths between adolescent alcohol use and peer alcohol use. These prospective paths were replicated across both time lags. Adolescents who reported higher levels of alcohol use at one time period tended to report higher levels of peer alcohol use at the following time period, and vice versa. These prospective effects accounted for unique variability beyond that attributed to the stability of the construct over time.

Discussion

This study was guided by three primary goals. The first was to test competing models of the longitudinal relations between adolescent alcohol use and peer alcohol use. The second was to test whether adolescent rebelliousness served as a third variable correlate in explaining the observed relation between adolescent alcohol use and peer alcohol use. The final goal was to compare the results of the random-effects LG models with more traditional fixed-effects models to identify potential advantages and disadvantages of these two longitudinal analytic techniques.

The LG models suggested that both adolescent and peer alcohol use were growing systematically over time, the functional form of this growth was linear, and there were significant individual differences in initial status and change over time. Earlier levels of adolescent alcohol use were strongly related to later changes in peer alcohol use, and earlier levels of peer alcohol use were strongly related to later changes in adolescent alcohol use. This bidirectional prospective relation remained even after the inclusion of adolescent rebelliousness. A more traditional AR model fitted to the same data similarly reflected that earlier levels of adolescent alcohol use were predictive of later levels of peer alcohol use and vice versa. However, the AR models were much more limited in that no inferences could be made about growth or predictors of individual differences in change over time. Overall, this study presents consistent support for the existence of both peer selection and peer socialization processes in the prediction of adolescent and peer alcohol use over time.

Unlike Farrell and Danish (Farrell, 1994; Farrell & Danish, 1993), who found support only for the unidirectional prediction of later peer substance use from earlier adolescent substance use, the present results are supportive of a prospective bidirectional relation between peer alcohol use and adolescent alcohol use. This bidirectional relation was supported by both random-effects growth models and fixed-effects AR models. These findings have potentially important implications for treatment and preventive intervention programs designed to delay the onset and escalation of adolescent substance use. Whereas the unidirectional findings of Farrell and Danish (Farrell, 1994; Farrell & Danish, 1993) suggest that earlier peer group affiliation does not influence later adolescent alcohol use, the current bidirectional findings suggest that peer group affiliation may be a useful candidate for treatment manipulation, at least for the subgroup
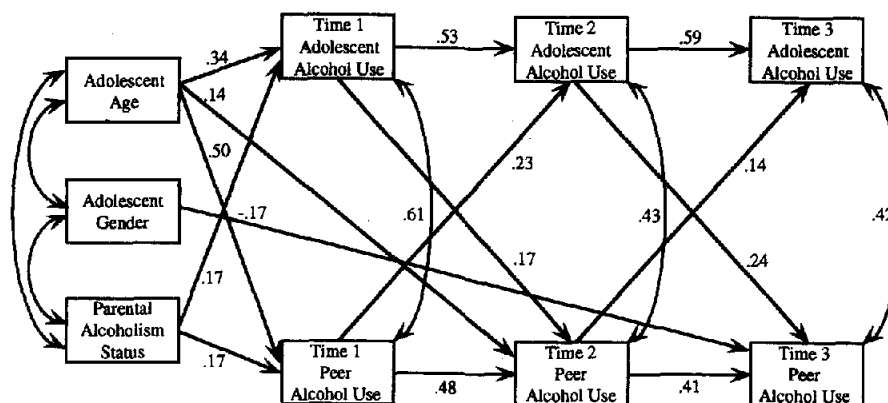

Figure 4. Autoregressive cross-lagged model of adolescent and peer alcohol use. Final model, $\chi^2(14, N = 363) = 29.3$, $p = .01$. All parameter values are standardized. All parameters shown are $p < .05$.

of adolescents who are similar in characteristics to those studied here.

One reason why our findings may have differed from those of Farrell and Danish (1993) and Farrell (1994) is that 92% of their sample were African American, whereas the present sample consisted of Hispanic and Caucasian adolescents. African American high school students tend to report later onset and lower overall levels of alcohol and drug use, compared with Caucasian youths (Johnston et al., 1991). Newcomb and Bentler (1986) found weaker cross-sectional correlations between adolescent alcohol use and peer alcohol use for African Americans, compared with Caucasians. Similarly, Brannock et al. (1990) found that the relation between alcohol use and preference for alcohol using peers was stronger for Caucasians than for African Americans, and Caucasians reported that their drinking was influenced by peers significantly more often than did African Americans. Thus, there may be weaker relations between peer influences and substance use for African American adolescents, compared with other ethnic groups, although the specific reason for this remains unclear. An important implication is that peer group intervention programs may be more beneficial for Hispanic or Caucasian youths.

The second goal of our study was to test for the possibility that the bidirectional relation observed between adolescent alcohol use and peer alcohol use was spurious and instead might be attributable to a common third variable influence. Although not often examined, it is important to consider because if the observed relation is actually spurious, then any attempts to manipulate one construct in hopes of producing a change in the second construct would be misplaced. It was hypothesized that rebelliousness might simultaneously influence adolescents to use alcohol themselves and associate with alcohol-using peers. Although adolescent rebelliousness was strongly related to the initial status of both peer alcohol use and adolescent alcohol use, rebelliousness was not related to later change over time in either construct. More importantly, the observed prospective bidirectional relation between peer alcohol use and adolescent alcohol use remained strong and consistent even after the inclusion of rebelliousness. Although there are many other potential third variable correlates that were not examined here, the present analyses suggest that the observed bidirectional relation between peer use and adolescent use cannot be attributed to the common third variable influence of adolescent rebelliousness.

The third goal of our study was to compare the results of the latent growth model with those of the more traditional fixed-effects AR model. The first comparison of interest was that, in this particular application, the AR and LG models resulted in rather similar conclusions about the basic theoretical questions of interest; that is, both models indicated that earlier adolescent alcohol use predicted later peer alcohol use, and earlier peer alcohol use predicated later adolescent alcohol use.[6] Despite the congruence in basic substantive conclusions, a key difference between the two approaches arises when considering what the AR model cannot conclude; that is, only the LG models could make inferences about growth over time. The LG models showed that peer and adolescent alcohol use was increasing over time, and that growth in peer use was decelerating as a function of adolescent use, whereas growth in adolescent use was accelerat-

ing as a function of peer use. In comparison, the AR model was not able to examine this complex growth process but instead only allowed inferences about the participants' standing relative to the group mean. For example, the positive prospective relations derived from the AR model indicated that their standing relative to the mean at Time 1 on one construct tended to be similar to their standing relative to the mean at Time 2 on the other construct (i.e., participants who were above the mean on adolescent use at Time 1 tended to be above the mean on peer use at Time 2). However, this measure of relative standing does not provide any information about a participant's continuous growth trajectory over all three time periods but only reflects that the participant maintained his or her rank relative to the mean between the two time points. Although both the LG and AR models resulted in somewhat similar conclusions about the basic questions of interest to the present study, the LG models allowed for richer and more dynamic types of conclusions to be drawn from the same data. Because of these advantages, we recommend that LG models be closely considered when studying behavioral change over time.

There are complex dimensions of peer groups, affiliations, and influences that we were not able to address in this study. For example, no distinction was made between the influences of peer cliques versus the influences of peer crowds (Brown, 1990). Because information about changes in peer group membership over time was not available, it was not possible to determine whether the same peer group was influencing the adolescent's behavior, or if the adolescent changed allegiance to a more substance-using group of friends over time. Additionally, the adolescents reported on both their own alcohol use as well as the alcohol use of their peers. It is possible that the observed relation between adolescent and peer alcohol use was overestimated because of the projection of the adolescent's own behavior on to that of their peers and, thus, might have inflated the prospective bidirectional relations between these two constructs (Bauman & Ennett, 1994; but see Donaldson, 1995). Finally, the present findings and subsequent conclusions are based solely on passive observational data. A true experimental design in which a preventive intervention is used to decrease an adolescent's alcohol use or peer's alcohol use would greatly increase the strength of the causal inferences that can be drawn.

The present study provides evidence supporting the bidirectional relation between peer alcohol use and adolescent alcohol use, at least for adolescents who have characteristics similar to those of the sample considered here. Despite the importance of supporting the existence of this effect, these analyses do not illuminate the mechanisms that underlie this effect (e.g., availability, modeling, self-medication, cognitive expectancies). Continued identification of the specific factors that mediate the overall process of influence is important for the design and implementation of preventive intervention and treatment programs that target adolescent substance use (Ennett & Bauman, 1991). In addition to consideration of mediating factors, it is

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[6] Note, however, that the degree of similarity in conclusions drawn from AR and LG models varies from sample to sample, and it is possible that quite different conclusions can be drawn from these two types of models that are fitted to the same data (Curran et al., 1996).

also important to explore influences that might moderate the relation between peer use and adolescent use. Examples might include the quality of the adolescent–parent relationship, adolescent individuation and autonomy, or adolescent depressive symptomatology. Further exploration of both mediating and moderating influences will help better understand the complex relation between the behavior of the peer group and that of the individual.

References

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

Bailey, S. L., & Hubbard, R. L. (1991). Developmental changes in peer factors and the influence on marijuana initiation among secondary school students. *Journal of Youth and Adolescence, 20,* 339–360.

Bauman, K. E., & Ennett, S. T. (1994). Peer influence on adolescent drug use. *American Psychologist, 49,* 820–822.

Bentler, P. M. (1989). *EQS: Structural equations program manual.* Los Angeles: BMDP Statistical Software.

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin, 107,* 238–246.

Bollen, K. A. (1989). *Structural equations with latent variables.* New York: Wiley.

Brannock, J. C., Schandler, S. L., & Oncley, P. R. (1990). Cross-cultural and cognitive factors examined in groups of adolescent drinkers. *Journal of Drug Issues, 20,* 427–442.

Brook, J. S., Brook, D. W., Gordon, A. S., Whiteman, M., & Cohen, A. (1990). The psychosocial etiology of adolescent drug use: A family interactional approach. *Genetic, Social, and General Psychology Monographs, 116,* 111–267.

Brook, J. S., Gordon, A. S., & Whiteman, M. (1985). Stability of personality during adolescence and its relationship to stage of drug use. *Genetic, Social, and General Psychology Monographs, 111,* 319–330.

Brook, J. S., Whiteman, M., Gordon, A. S., & Cohen, P. (1989). Changes in drug involvement: A longitudinal study of childhood and adolescent determinants. *Psychological Reports, 65,* 707–726.

Brook, J. S., Whiteman, M., Gordon, A. S., Nomura, C., & Brook, D. W. (1986). Onset of adolescent drinking. A longitudinal study of intrapersonal and interpersonal antecedents. *Advances in Alcohol and Substance Use, 5,* 91–110.

Brown, B. B. (1990). Peer groups and peer cultures. In S. Feldman & G. Elliott (Eds.), *At the threshold: The developing adolescent* (pp. 171–196). Cambridge, MA: Harvard University Press.

Bryk, A. S., & Raudenbush, S. W. (1992). *Hierarchical linear models: Applications and data analysis methods.* Newbury Park, CA: Sage.

Chassin, L., Barrera, M., Bech, K., & Kossak, J. (1992). Recruiting a community sample of adolescent children of alcoholics: A comparison of three subject sources. *Journal of Studies on Alcohol, 53,* 316–320.

Chassin, L., Rogosch, R., & Barrera, M. (1991). Substance use and symptomatology among adolescent children of alcoholics. *Journal of Abnormal Psychology, 100,* 449–463.

Colder, C. R. (1990). *Social bonding and stress and negative affect: One step beyond two theories of substance use.* Unpublished master's thesis, Arizona State University.

Curran, P. J., Harford, T., & Muthén, B. O. (1996). The relation between heavy alcohol use and bar patronage: A latent growth model. *Journal of Studies on Alcohol, 57,* 410–418.

Donaldson, S. I. (1995). Peer influence on adolescent drug use: A perspective from the trenches of experimental evaluation research. *American Psychologist, 50,* 801–802.

Downs, W. R. (1987). A panel study of normative structure, adolescent

alcohol use and peer alcohol use. *Journal of Studies on Alcohol, 48,* 167–175.

Duncan, T. E., Duncan, S. C., & Hops, H. (1994). The effects of family cohesiveness and peer encouragement on the development of adolescent alcohol use: A cohort-sequential approach to the analysis of longitudinal data. *Journal of Studies on Alcohol, 55,* 588–599.

Dwyer, J. H. (1983). *Statistical models for the social and behavioral sciences.* New York: Oxford University Press.

Ellickson, P. L., & Hays, R. D. (1991). Antecedents of drinking among young adolescents with different alcohol use histories. *Journal of Studies on Alcohol, 52,* 398–408.

Endicott, J., Andreason, N., & Spitzer, R. L. (1978). *Family History–Research Diagnostic Criteria (FH-RDC).* New York: New York State Psychiatric Institute.

Ennett, S. T., & Bauman, K. E. (1991). Mediators in the relationship between parental and peer characteristics and beer drinking by early adolescents. *Journal of Applied Social Psychology, 21,* 1699–1711.

Farrell, A. D. (1994). Structural equation modeling with longitudinal data: Strategies for examining group differences and reciprocal relationships. *Journal of Consulting and Clinical Psychology, 62,* 477–487.

Farrell, A., & Danish, S. (1993). Peer drug associations and emotional restraint: Causes or consequences of adolescents' drug use? *Journal of Consulting and Clinical Psychology, 43,* 522–527.

Fisher, L. A., & Bauman, K. E. (1988). Influence and selection in the friend-adolescent relationship: Findings from studies of adolescent smoking and drinking. *Journal of Applied Social Psychology, 18,* 289–314.

Hawkins, J. D., Catalano, R. F., & Miller, J. Y. (1992). Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention. *Psychological Bulletin, 112,* 64–105.

Johnston, L. D., O'Malley, P. M., & Bachman, J. G. (1991). Drug use among American high school seniors, college students and young adults, 1975–1990: Vol. 1. High school seniors (DHHS Publication No. ADM 91-1813). Rockville, MD: U.S. Government Printing Office.

Kandel, D. B. (1978). Homophily, selection, and socialization in adolescent friendships. *American Journal of Sociology, 84,* 427–436.

Kandel, D. B. (1985). On processes of peer influences in adolescent drug use: A developmental perspective. *Advances in Alcohol and Substance Abuse, 4,* 139–163.

Kandel, D. B., & Andrews, K. (1987). Processes of adolescent socialization by parents and peers. *International Journal of the Addictions, 22,* 319–342.

MacCallum, R. (1986). Specification searches in covariance structure modeling. *Psychological Bulletin, 100,* 107–120.

McArdle, J. J., & Epstein, D. (1987). Latent growth curves within developmental structural equation models. *Child Development, 58,* 110–133.

Meredith, W., & Tisak, J. (1984). *"Tuckerizing" curves.* Paper presented at the annual meeting of the Psychometric Society, Santa Barbara, CA.

Meredith, W., & Tisak, J. (1990). Latent curve analysis. *Psychometrika, 55,* 107–122.

Muthén, B. (1991). Analysis of longitudinal data using latent variable models with varying parameters. In L. M. Collins & J. L. Horn (Eds.), *Best methods for the analysis of change: Recent advances, unanswered questions, future directions* (pp. 1–17). Washington, DC: American Psychiatric Association.

Muthén, B. (1993). Latent variable modeling of growth with missing data and multilevel data. In C. M. Cuadras & C. R. Rao (Eds.), *Multi-*

variate analysis: Future directions 2 (pp. 199–210). Amsterdam, The Netherlands: North Holland.

Newcomb, M. D., & Bentler, P. M. (1986). Substance use and ethnicity: Differential impact of peer and adult models. *Journal of Psychology, 120,* 83–95.

Oetting, E. R., & Beauvais, F. (1986). Peer cluster theory: Drugs and the adolescent. *Journal of Counseling and Development, 65,* 17–22.

Oetting, E. R., & Beauvais, F. (1987). Peer cluster theory, socialization characteristics and adolescent drug use: A path analysis. *Journal of Counseling Psychology, 34,* 205–213.

Pruitt, B. E., Kingery, P. M., Mirzaee, E., Heuberger, G., & Hurley, R. S. (1991). Peer influence and drug use among adolescents in rural areas. *Journal of Drug Education, 21,* 1–11.

Robins, L. N., Helzer, J. E., Croughan, J., & Ratcliff, K. S. (1981). National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. *Archives of General Psychiatry, 38,* 381–389.

Rogosa, D. R. (1987). Causal models do not support scientific conclusions: A comment in support of Freedman. *Journal of Educational Statistics, 12,* 185–195.

Rogosa, D. R. (1988). Myths about longitudinal research. In K. W. Schaie, R. T. Campbell, W. Meredith, & S. C. Rawlings (Eds.), *Methodological issues in aging research* (pp. 171–209). New York: Springer.

Rogosa, D., & Willett, J. B. (1985). Understanding correlates of change by modeling individual differences in growth. *Psychometrika, 50,* 203–228.

Smith, G. M., & Fogg, C. P. (1979). Psychological antecedents of teenage drug use. *Research in Community and Mental Health, 1,* 87–102.

Stein, J. A., Newcomb, M. D., & Bentler, P. M. (1987). An 8-year study of multiple influences on drug use and drug use consequences. *Journal of Personality and Social Psychology, 53,* 1094–1105.

Stoolmiller, M., Duncan, T., Bank, L., & Patterson, G. (1993). Some problems and solutions in the study of change: Significant patterns in client resistance. *Journal of Consulting and Clinical Psychology, 61,* 920–928.

Swaim, R. C., Oetting, E. R., Edwards, R. W., & Beauvais, F. (1989). Links from emotional distress to adolescent drug use: A path model. *Journal of Consulting and Clinical Psychology, 57,* 227–231.

Tucker, L. R., & Lewis, C. (1973). A reliability coefficient for maximum likelihood factor analysis. *Psychometrika, 38,* 1–10.

Willett, J. B., & Sayer, A. G. (1994). Using covariance structure analysis to detect correlates and predictors of change. *Psychological Bulletin, 116,* 363–381.