CHAPTER 4

The Best of Both Worlds

Combining Autoregressive and Latent Curve Models

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There are various approaches to both the theoretical conceptualization and the statistical analysis of panel data. Two analytic approaches that have received a great deal of attention are the *autoregressive model* (or "fixed effects Markov simplex model") and random coefficient *growth curve models*. Researchers have attempted to identify the conditions under which the growth curve and autoregressive approaches do or do not provide useful results when applied to empirical longitudinal data (see, e.g., Bast & Reitsma, 1997; Curran, 2000; Kenny & Campbell, 1989; Marsh, 1993; and Rogosa & Willett, 1985). This critical comparative approach has tended to foster a polarization of views that has led many proponents of one modeling approach to reject the methods of the other, and vice versa.

However, what has become increasingly apparent is that there is not necessarily a "right" or "wrong" approach to analyzing repeated-measures data over time. The proper choice of a statistical model varies as a function of the theoretical question of interest, the characteristics of the empirical data, and the researcher's own philosophical beliefs about issues such as causation and change. Despite the more tempered view that different analytic approaches can reveal different things about the same data, the autoregressive and growth curve modeling approaches remain competing analytic viewpoints. A moderate position sees these two models as equally viable options in which the autoregressive model is more appropriate under some conditions and the growth

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curve model works best under other conditions. Although this approach tends to be less adversarial than the correct–incorrect distinction, the result still remains an either–or scenario; that is, one adopts the autoregressive approach or the growth modeling approach, but not both.

Given that the autoregressive and growth curve models are each associated with certain key advantages and disadvantages, it seems logical to work toward synthesizing these approaches into a more unified general framework. If successful, this would allow for drawing on the strengths of both approaches that might provide even greater information than either approach taken alone. Our goal is to work toward developing such a synthesized longitudinal model of change.

In this chapter, we present an extended empirical example to illustrate our ongoing efforts to synthesize these models. Although we provide the basic equations and assumptions for the models that we estimate, our emphasis here is on the application of these techniques to an empirical example. A more technical treatment of our models is presented elsewhere (Bollen & Curran, 1999, 2000). We open this chapter with a description of a theoretical substantive question that motivates the development of the synthesized model. This is followed by a brief introduction to the data for the empirical example. We then present a review of the univariate and bivariate autoregressive simplex models followed by a general description of the univariate and bivariate latent curve models. In the next section, we propose the synthesis of the simplex and latent curve model for both the univariate and bivariate cases. The simplex, latent curve, and synthesized models are then systematically applied to the empirical data set to evaluate a series of questions relating to the developmental relation between antisocial behavior and depressive symptomatology in children over time. We conclude with model extensions on which we are currently working as well as directions for future research.

Developmental Relation Between Antisocial Behavior and Depressive Symptomatology

There has been a great deal of interest in the developmental relation between antisocial behavior and depressive symptomatology over time, both in terms of predictors of change in these constructs and potential bidirectional relations between them over time. Better understanding of these complex developmental processes are important not only for establishing the etiology of these disorders but also for helping inform prevention and intervention programs targeted at internalizing and externalizing symptomatology. Recent empirical evidence suggests that antisocial behavior and depressive symptomatology in childhood are related to one another, both cross-sectionally (e.g., Capaldi, 1991) and longitudinally (e.g., Capaldi, 1992). Despite these important findings, the specific nature of this developmental relation remains unclear. Specifically, it is not clear if the continuous underlying developmental trajectories of these constructs are related to one another or if instead the underlying trajectories are rather independent of one another but the time-specific levels of symptomatology are related over time.

For example, it may be that a steeply increasing developmental trajectory of antisocial behavior across time may influence the corresponding underlying trajectory of depressive symptomatology. Thus, the time-specific measures of these behaviors do not relate directly to one another but instead the relation is solely at the level of the continuous trajectory. Alternatively, these two underlying developmental trajectories may be relatively independent, but an elevated level of antisocial behavior at a particular time point might be associated with a subsequent elevation of depressive symptomatology at a later time point. In this case, there are two sources of influence on the repeated measures over time. The first is the influence from the underlying growth trajectory for that particular construct (e.g., antisociality), and the second is the influence from the time-specific preceding measures on the other construct (e.g., depression). So the time-specific observed measures of antisocial behavior are due to a combination of the continuous underlying developmental trajectory of antisociality and time-specific influences of depressive symptomatology.

Although it is rather straightforward to hypothesize a theoretical model such as this, current statistical methods are not well suited for empirically evaluating this model using sample longitudinal data (Curran & Hussong, in press). It is ironic that there are two well-developed analytic approaches that can be used to examine one component of the theoretical model or of the other but not of both. The Markov simplex modeling approach is well suited for examining the time-specific relations between two constructs over time, and the growth modeling approach is well suited for examining relations in individual differences in continuous developmental trajectories over time. At this point, there is no well-developed strategy for examining both of these components simultaneously (but see chapter 5, by McArdle and Hamagami, in this volume, for an important alternative approach to dealing with a similar type of problem). The development of such a model serves two key purposes. First, this technique allows for a comprehensive empirical evaluation of the developmental relation between antisocial behavior and depressive symptomatology over time. Second, this technique can be generalized and applied to many other types of longitudinal settings to evaluate similar types of questions.

Data for an Applied Example

The empirical data come from the National Longitudinal Survey of Youth (NLSY). The original 1979 panel included a total of 12,686 respondents, 6,283 of whom were women. Beginning in 1986, an extensive set of assessment instruments was administered to the children of the original NLSY female respondents and was repeated every other year thereafter. The data used here are drawn from the children of the NLSY female respondents, and three key criteria determined inclusion in the sample. First, children must have been 8 years of age at the first wave of measurement, a sampling design that helps control for developmental heterogeneity. Second, children must have data on all measures we use for all four waves of measurement. Finally, the sample includes only one biological child from each mother. On the basis of these three criteria, the final sample consisted of 180 children (57% were male).

Although there are a variety of powerful options currently available for estimating models with missing data (e.g., Arbuckle, 1996; Graham, Hofer, & MacKinnon, 1996; Little & Rubin, 1987; B. O. Muthén, Kaplan, & Hollis, 1987; L. K. Muthén & Muthén, 1998), for purposes of simplicity we ignore this complication to better focus on the proposed models. Of the initial 282 cases that met the selection criteria with valid data at Time 1, 29 (10%) were missing at Time 2; 76 (27%) were missing at Time 3; 79 (28%) were missing at Time 4; and 102 (36%) were missing one or more assessments at Times 2, 3, and 4. Thus, the final sample consisted of 180 (64%) of those children eligible at Time 1 and with complete data at Times 2, 3, and 4, and subsequent modeling results should be interpreted with this in mind.

Children's antisocial behavior and children's depressive symptomatology are the two constructs we consider. Antisocial behavior was operationalized using the mother's report on six items that assessed the child's antisocial behavior as it had occurred over the previous 3 months. The three possible response options were "not true" (scored 0), "sometimes true" (scored 1), or "often true" (scored 2). We summed these six items to compute an overall measure of antisocial behavior that ranged from 0 to 12. Depressive symptomatology was operationalized using the mother's report on five items that assessed the child's internalizing and depression symptoms having occurred over the previous 3 months using the same response options as for antisocial behavior. We summed the five items to compute an overall measure of depressive symptomatology with a range from 0 to ten. The means, standard deviations, and correlations for the four repeated measures of antisocial behavior and depressive symptomatology are presented in Table 4.1.

The Longitudinal Markov Simplex Model

One of the most important approaches developed for the analysis of panel data is the autoregressive or Markov simplex model. Its earliest development dates back to the seminal work of Guttman (1954), who proposed a model to examine the simplex structure of correlations derived from a set of ordered tests.

TABLE 4.1

Means, Variances, Covariances, and Correlations for Four Repeated Measures of Antisocial Behavior and Four Repeated Measures of Depressive Symptomatology

MEASURE	1	2	3	4	5	6	7	8
1. Time 1 antisocial	2.926	1.390	1.698	1.628	1.240	0.592	0.929	0.659
2. Time 2 antisocial	0.394	4.257	2.781	2.437	0.789	1.890	1.278	0.949
3. Time 2 antisocial	0.466	0.633	4.536	2.979	0.903	1.419	1.900	1.731
4. Time 4 antisocial	0.402	0.499	0.591	5.605	1.278	1.004	1.000	2.420
5. Time 1 depression	0.405	0.214	0.237	0.301	3.208	1.706	1.567	0.988
6. Time 2 depression	0.173	0.458	0.333	0.212	0.477	3.994	1.654	1.170
7. Time 3 depression	0.287	0.327	0.471	0.223	0.462	0.437	3.583	1.146
8. Time 4 depression	0.202	0.241	0.426	0.535	0.289	0.306	0.317	3.649
M	1.750	1.928	1.978	2.322	2.178	2.489	2.294	2.222

Note. Correlations are below the diagonal, covariances are above the diagonal, and variances are on the diagonal. All statistics are based on $N = 180$.

Anderson (1960), Humphreys (1960), Heise (1969), and Jöreskog (1970, 1979) further developed these univariate panel data models. The key characteristic of the simplex model is that correlations decrease in magnitude as a function of distance from the diagonal of the correlation matrix. When applied to longitudinal data, this means that later measures have progressively lower correlations with earlier measures as a function of increasing time. Furthermore, change in the construct over time is an additive function of the influence of the immediately preceding measure of the construct plus a random disturbance. The path diagram for the model is presented in Figure 4.1. The equation for the measured variable y at initial time period $t = 1$ is

$$y_{i1} = \alpha_1 + \varepsilon_{i1}, \tag{4.1}$$

and for subsequent time periods is

$$y_{it} = \alpha_t + \rho_{t,t-1} y_{i,t-1} + \varepsilon_{it}, \tag{4.2}$$

where $E(\varepsilon_{it}) = 0$ for all i and t and $\mathrm{COV}(\varepsilon_{it}, y_{i,t-1}) = 0$ for all i and $t = 2, 3, \ldots, T$. Furthermore, the variance of the measured y for all i at the initial time period is

$$V(y_{i1}) = \theta_{\varepsilon_1} \tag{4.3}$$

and at subsequent time periods is

$$V(y_{it}) = \rho^2_{t,t-1} V(y_{i,t-1}) + \theta_{\varepsilon_t}, \tag{4.4}$$

with the expected value for the initial time period

$$E(y_{i1}) = \alpha_1 \tag{4.5}$$

and for subsequent time periods

$$E(y_{it}) = \alpha_t + \rho_{t,t-1}\alpha_{t-1}. \tag{4.6}$$

Each measure is only a function of the immediately preceding measure plus a random disturbance. This is the source of the term *autoregressive*—the measure at each time point is regressed onto the same measure at the previous time point. Variables assessed at times earlier than the immediately prior time have no direct impact on the current value. An implication of this model is that the correlation between time t and time $t + 2$ is zero when controlling for the effects of time $t + 1$; the influence of the measure at time t on the measure at time $t + 2$ is entirely mediated by the measure at time $t + 1$.

Another term for this autoregressive model is the *univariate simplex model* because of the focus on only a single variable. This model can be directly extended to the multivariate case with two or more distinct variables over time. These panel data models that include additional explanatory variables received considerable attention and development from several sources (e.g., Bohrnstedt, 1969; Campbell, 1963; O. D. Duncan, 1969; Heise, 1969; Jöreskog, 1979). We extend Equation 4.2 to include both the autoregressive parameters and the *crosslagged* coefficients that allow for influences across constructs (see Figure 4.2). These crosslags represent the longitudinal prediction of one construct from the other above and beyond the autoregressive prediction of that construct from itself. The initial measures remain as before, but subsequent measures on y are

$$y_{it} = \alpha_{yt} + \rho_{yt,yt-1} y_{i,t-1} + \rho_{zt,zt-1} z_{i,t-1} + \varepsilon_{y_{it}}, \tag{4.7}$$

indicating that the measure of y at time t is a function of an intercept, the weighted influence of y at time $t - 1$, the weighted influence of z at time $t - 1$, and a random time-specific error, $\varepsilon_{y_{it}}$, that has a mean of zero and is uncorrelated with $y_{i,t-1}$ and $z_{i,t-1}$. An analogous equation holds for z_{it}, and the disturbances for these two equations are allowed to correlate. The substantive interpretations of the crosslagged parameter is that an earlier measure of z

FIGURE 4.1
Univariate Markov simplex model.

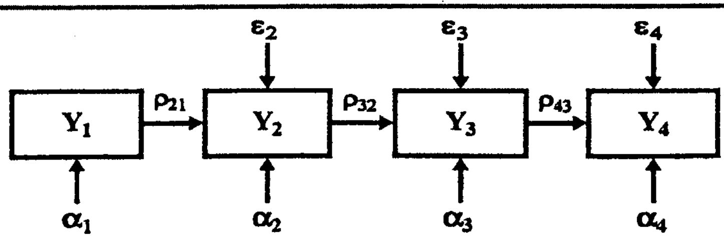
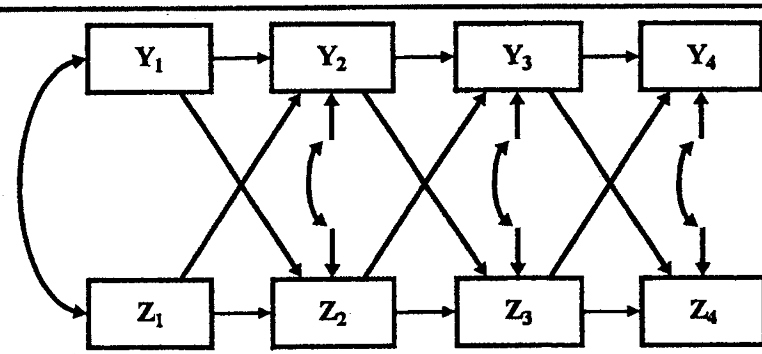


FIGURE 4.2
Bivariate Markov simplex model with correlated disturbances.



predicts a later measure of y above and beyond the previous measure of y. This is often referred to as an *autoregressive crosslagged* model.

Latent Curve Analysis

The preceding autoregressive univariate and bivariate models consider change over time in terms of each variable depending on its immediately prior value but not on its values for earlier periods. In addition, the autoregressive and crosslagged effects are the same for each individual. Although advantageous in many settings, this approach can be somewhat limiting when studying theoretical questions about individual differences in continuous developmental trajectories over time. Growth models approach the question of change from a different perspective. Instead of examining the time-adjacent relations of antisocial measures, we use the observed repeated measures to estimate a single underlying growth trajectory for each person across all time points. We can think of this as fitting a short time series trend line to the repeated measures for each individual. The x variable is time (where x equals 0, 1, 2, 3 in the case of four waves), the y variable is antisocial behavior, and we consider only 1 participant at a time. This line of best fit is an estimate of the individual's *growth trajectory* of antisociality over time. When a trajectory is fit to each individual in the sample, a researcher can compute an average intercept and average slope (sometimes called *fixed effects*) as well as the variability around these averages (sometimes called *random effects*).

Such developmental trajectories have long been hypothesized from substantive theory, but it has historically been quite difficult to properly estimate these trajectories statistically. There are several different approaches available for the estimation of these types of models, and one important example is latent curve analysis. Latent curve analysis is a direct extension of the structural equation model (SEM) that is common in the social sciences. The SEM approach simultaneously estimates relations between observed variables and the corresponding underlying latent constructs, and between the latent constructs themselves (Bentler, 1980, 1983; Jöreskog, 1971a, 1971b; Jöreskog & Sörbom, 1978). However, unlike the standard SEM approach, latent curve analysis explicitly models both the observed mean and covariance structure of the data (McArdle, 1986, 1988, 1989, 1991; McArdle & Epstein, 1987; Meredith & Tisak, 1984, 1990; B. Muthén, 1991).

From the SEM framework, the factor analytic model relates the observed variables y to the underlying latent construct η such that

$$y = v + \Lambda\eta + \varepsilon, \tag{4.8}$$

where v is a vector of measurement intercepts, Λ is a matrix of factor loadings (or measurement slopes), and ε is a vector of measurement residuals. The latent variable equation is

$$\eta = \alpha + \beta\eta + \zeta, \tag{4.9}$$

where α is a vector of structural intercepts, β is a matrix of structural slopes, ζ is a vector of structural residuals, and $V(\zeta) = \Psi$ represents the covariance structure among the latent factors. The model-implied mean structure is given as

$$E(y) = \mu = \nu + \Lambda(I - \beta)^{-1}\alpha, \tag{4.10}$$

and the covariance structure is given as

$$V(y) = \hat{\Sigma} = \Lambda(I - \beta)^{-1}\Psi(I - \beta)^{-1\prime}\Lambda' + \Theta. \tag{4.11}$$

Given that latent curve models are a direct extension of SEMs, one can use standard software such as AMOS, EQS, LISREL, or MPlus to estimate these models.

To estimate the variance components associated with the random growth coefficients, the latent curve analysis imposes a highly restricted factor structure on η through the Λ matrix. Consider an example in which there are $T = 4$ yearly measures of antisocial behavior collected from a sample of children. Two latent factors are estimated, one representing the intercept of the antisocial behavior growth trajectory (η_α), and the second representing the slope (η_β). This model is presented in Figure 4.3. The factor loadings relating the four antisocial measures to the intercept factor are fixed to 1.0 to define the intercept of the antisocial growth trajectory. The factor loadings relating the observed repeated measures to the slope factors are a combination of fixed and free loadings that best capture the functional form of the growth trajectory over the four time points. The initial approach is to fix the factor loadings to 0, 1, 2, and 3 to represent straight-line growth. The estimated mean of the intercept factor (μ_α) represents the initial status of the antisocial growth trajectory averaged across all individuals; the estimated variance of the intercept factor (ψ_α) represents the individual variability in initial levels of antisociality. Similarly, the estimated mean of the slope factor (μ_β) represents the slope of the antisocial trajectory averaged across all individuals, and the estimated variance of the slope factor (ψ_β) represents individual variability in rates of change in antisociality over time. Finally, the covariance between the intercept and slope factors is denoted $\psi_{\alpha\beta}$. Thus, the observed repeated measures are expressed as

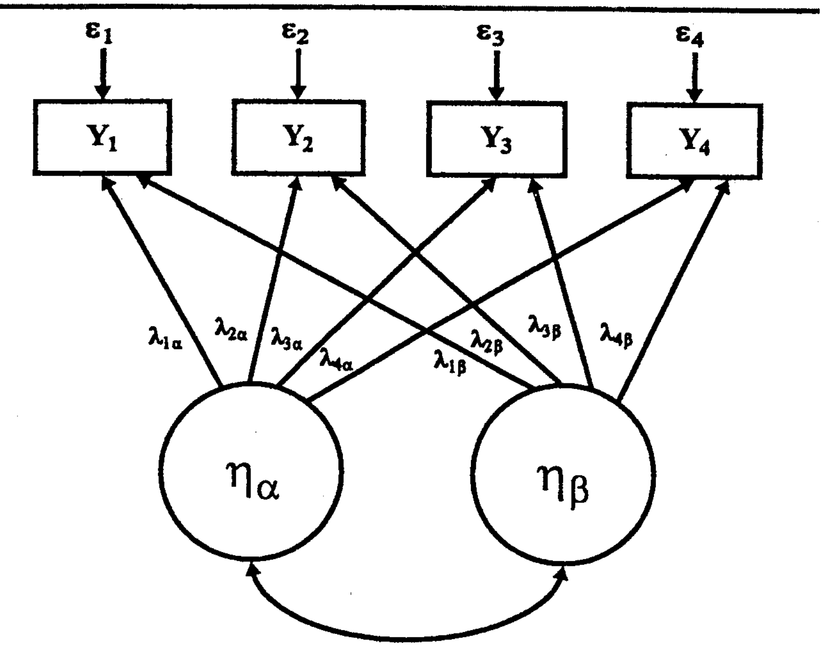
$$y_{it} = \eta_{\alpha_i} + \lambda_t\eta_{\beta_i} + \varepsilon_{it}, \tag{4.12}$$

where $\lambda_t = 0, 1, 2, 3$ and

$$\eta_{\alpha_i} = \mu_\alpha + \zeta_{\alpha_i} \tag{4.13a}$$

$$\eta_{\beta_i} = \mu_\beta + \zeta_{\beta_i}. \tag{4.13b}$$

FIGURE 4.3
Univariate latent curve model.



Substituting Equations 13a and 13b into Equation 12 leads to

$$y_{it} = (\mu_{\alpha} + \lambda_t \mu_{\beta}) + (\zeta_{\alpha_i} + \lambda_t \zeta_{\beta_i} + \varepsilon_{it}), \qquad (4.14)$$

where the first parenthetical term represents the fixed effect and the second term represents the random effect. The variance and expected value can then be expressed as

$$V(y_{it}) = \psi_{\alpha} + \lambda_t^2 \psi_{\beta} + 2\lambda_t \psi_{\alpha\beta} + \theta_{\varepsilon_t} \qquad (4.15)$$

$$E(y_{it}) = \mu_{\alpha} + \lambda_t \mu_{\beta}. \qquad (4.16)$$

The latent curve model described above is considered *univariate,* given that growth in a single construct is considered. However, this model can easily be extended to a *multivariate* situation to consider change in two or more constructs over time. Technical details of this procedure were presented by MacCallum, Kim, Malarkey, and Kiecolt-Glaser (1997) and McArdle (1989), and sample applications include Curran and Hussong (in press); Curran, Stice, and Chassin (1997); S. C. Duncan and Duncan (1996), and Stoolmiller (1994). Conceptually, the multivariate growth model is simply the simultaneous estimation of two univariate growth models. A researcher estimates growth factors for each construct, and typically the relation between changes in the construct over time is modeled at the level of the growth factors. That is, we allow covariances among the factors across constructs, or, alternatively, one growth factor might be regressed onto another growth factor to examine unique predictability across constructs. Regardless of how an analyst estimates these, it is important to note that the relations across constructs are typically evaluated at the level of the growth trajectories, not at the level of the repeated measures over time.

Each of these modeling approaches is uniquely suited to examining a particular form of change over time. The autoregressive simplex explicitly models the time-specific relations within and between repeated measures of one or more constructs, whereas the latent curve model explicitly models these relations strictly at the level of the continuous trajectory believed to underlie these same repeated measures. It would be valuable in many areas of applied research to be able to simultaneously take advantage of the strengths of each of these approaches. Furthermore, it also would be useful to know whether the autoregression, the latent curve model, or some combination of these models best describes the data. To address these issues, we now work toward combining the autoregressive simplex and latent curve modeling strategies into a single comprehensive model of change over time.

Combined Autoregressive Latent Curve Model

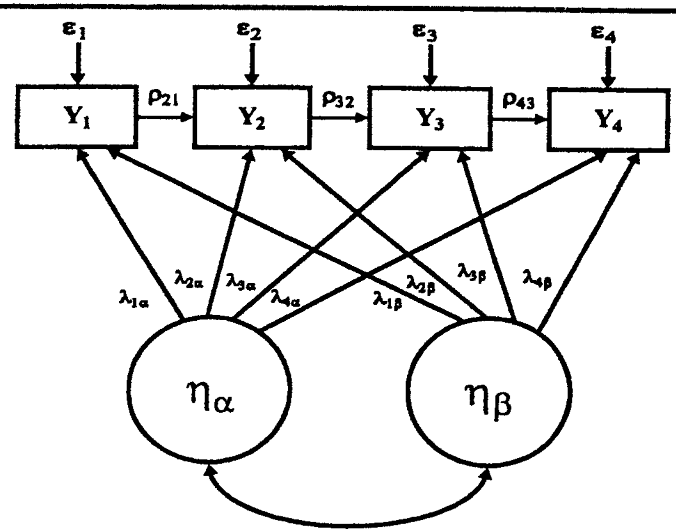
This synthesis proceeds in a straightforward manner, and we begin with the univariate case presented in Figure 4.4. The model includes a random intercept and slope factor from the latent curve model to capture the continuous underlying growth trajectories over time. It also incorporates the standard autoregressive simplex parameters to allow for the time-specific influences between the repeated measures themselves. Whereas the means and intercepts are part of the repeated measures in the simplex model, the mean structure enters solely through the latent growth factors in the synthesized model. This parameterization results in the expression of the measure of construct y for individual i at time point t as

$$y_{it} = \eta_{\alpha_i} + \lambda_t \eta_{\beta_i} + \rho_{t,t-1} y_{i,t-1} + \varepsilon_{it}, \qquad (4.17)$$

which highlights that the time-specific measure of y is an additive function of the underlying intercept factor, the underlying slope factor, a weighted contribution of the prior measure of y, and a time-specific random error term that has a mean of zero and that is uncorrelated with the righthand side variables. Viewing the model from this equation one sees that the simplex and latent curve models are not necessarily in competition as to which is proper or im-

FIGURE 4.4

Univariate simplex latent curve model.



proper, but instead each is a restricted variation of a more comprehensive model.

Some implications of Equation 4.17 that are not immediately obvious concern the "factor loadings" of y_{i1} on $\eta_{\alpha i}$ and $\eta_{\beta i}$. In the usual latent curve model these loadings are fixed to 1 and 0, respectively. However, in the presence of an autoregressive structure for y, this is no longer true. The reason is that implicit in this model is that y_{i1} depends on y_{i0}, which in turn depends on $y_{i,-1}$, on back to the earliest possible value of y. Furthermore, each of these earlier (unavailable) ys would be influenced by $\eta_{\alpha i}$ and $\eta_{\beta i}$. Figure 4.5 represents these omitted earlier measures of y and their positions in the model in gray and the positions of the observed measures in black. As a result of these omitted ys, the factor loadings of y_{i1} on $\eta_{\alpha i}$ and on $\eta_{\beta i}$ depart from their values in a standard latent curve model. More specifically, the factor loading for y at time $t = 1$ on $\eta_{\alpha i}$ is

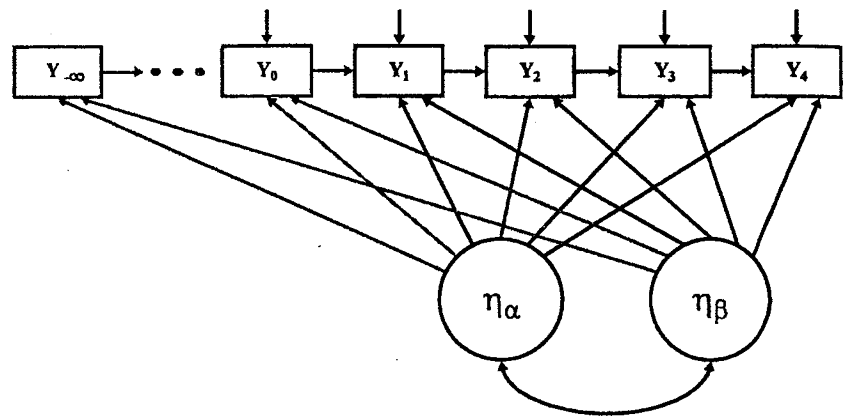
$$\lambda_{1\alpha} = \frac{1}{1 - \rho}, \qquad (4.18)$$

and the factor loading for y at time $t = 1$ on $\eta_{\beta i}$ is

$$\lambda_{1\beta} = -\left(\frac{\rho}{(1 - \rho)^2}\right), \qquad (4.19)$$

FIGURE 4.5

Univariate simplex latent curve model with omitted measures preceding Time 1.



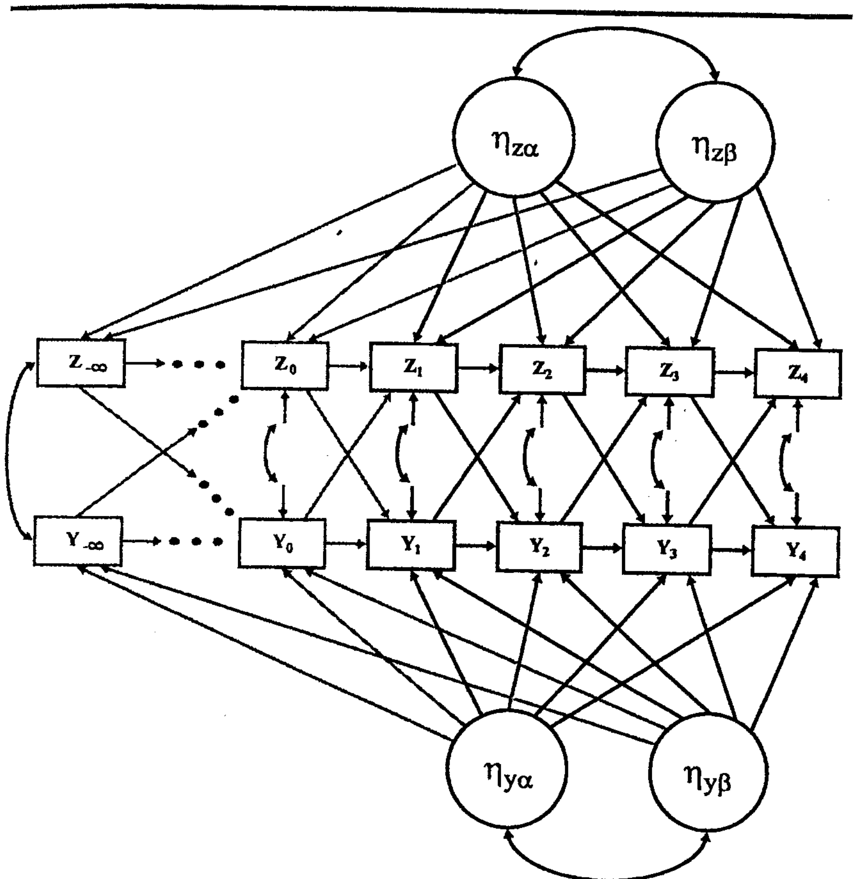
for which we assume that the autoregressive parameter is equal for all t ($\rho_{t,t-1} = \rho$) and that $|\rho| < 1$. As $\rho \to 0$, then $\lambda_{1\alpha} \to 1$ and $\lambda_{1\beta} \to 0$, which corresponds precisely to the values imposed in the standard latent curve model. However, as the value of ρ departs from zero, then fixing these factor loadings to 1.0 and 0 becomes increasingly restrictive and likely leads to bias elsewhere in the model. The technical developments that lead to these results are presented in Bollen and Curran (2000) in which we also propose a form of this model that treats the y_{i1} as "predetermined" so that these nonlinear constraints are not needed.

We can extend this univariate combined model to the multivariate case to examine these relations both within and across constructs. Here, the measure of y at time t for individual i is composed of the influence from the growth factors underlying y, the prior measure of y, and now the prior measure of, say, z. This leads to

$$y_{it} = \eta_{\alpha_i} + \lambda_t \eta_{\beta_i} + \rho_{yt,yt-1} y_{i,t-1} + \rho_{zt,zt-1} z_{i,t-1} + \varepsilon_{it}. \qquad (4.20)$$

This combined bivariate autoregressive latent curve model is presented in Figure 4.6, in which the omitted lagged measures described above are portrayed in gray and the observed measures are portrayed in black. This model highlights that a given measure of y is an additive combination of the continuous growth process underlying y, the weighted influence of the preceding measure of y,

FIGURE 4.6
Multivariate simplex latent curve model with omitted measures preceding Time 1.



the weighted influence of the preceding measure of z, and a time-specific random disturbance.[1] The model simultaneously and explicitly incorporates the strengths of both the autoregressive simplex and the latent curve model and

[1]Several colleagues have suggested that instead of modeling autoregressive structure among the observed measures we instead model these effects directly among the time-specific residuals. We do not pursue this strategy given our desire to more explicitly combine the autoregressive (simplex) and growth curve modeling traditions. See Goldstein, Healy, and Rasbash (1994) for an example of autoregressive structures among residuals.

allows for a more comprehensive evaluation of change in one or more constructs over time.

To demonstrate this approach, we will now apply a series of simplex and latent curve models to an empirical data set to evaluate the developmental relation between antisocial behavior and depressive symptomatology over time.

An Applied Example of the Autoregressive, Latent Curve, and Synthesized Models

We now incrementally illustrate the univariate and multivariate models that we presented in the previous sections. We apply these to the relation between antisocial behavior and depressive symptomatology in the sample of N = 180 eight-year-old children.

Tests of Equality of Means Over Time: Antisocial Behavior

Although the summary statistics presented in Table 4.1 suggest that both the means and variances of antisocial behavior are increasing as a function of time, a more formal test of this relation is necessary. There are a variety of methods for executing such a test (e.g., paired t test, repeated-measures analysis of variance), but we evaluate the mean structure using an SEM approach. The advantage of this technique is that an extension of this mean difference model allows the estimation of both the simplex model and the latent curve model. We fit this model of equal means to the four antisocial-behavior measures. We did not constrain the variances and covariances of the repeated measures, and we placed no equality constraints on the means of the four measures. This model is just identified and thus has a chi-square value of zero. Next we imposed equality constraints on the four means, which resulted in $\chi^2(3, N = 180) = 11.1$, $p = .011$; incremental fit index (IFI) = .96, root mean square error of approximation (RMSEA) = .12, 90% confidence interval (CI) = .05, .20 (see Steiger & Lind, 1980, and Browne & Cudeck, 1993, for a description of the RMSEA; and Bollen, 1989, for a description of the IFI). On the basis of this poor model fit (e.g., although the IFI exceeded .95, this was in the presence of a significant chi-square and an RMSEA exceeding .10), the null hypothesis that all means are equal over time is rejected. In a moment, we will use a latent curve model to examine the patterning of these means as a function of time.

The Simplex Model With Means: Antisocial Behavior

We now fit the univariate simplex model to the four repeated measures of antisocial behavior. Given the findings of the mean difference model, we begin by including means in the simplex model. Although in the traditional simplex modeling approach the mean structure is usually omitted, this is not necessary. The baseline simplex model has each variable at a given time regressed onto its immediately preceding variable value. We estimate the mean and variance for the Time 1 measure and the intercepts and disturbance variances for the Times 2, 3, and 4 measures without any equality constraints. The model fit the data quite poorly, $\chi^2(3, N = 180) = 29.04$, $p < .001$; IFI = .88; RMSEA = .22; 90% CI = .15, .30. Next we include a series of equality constraints starting with the three autoregressive parameters, then on the variances of the three disturbances and, finally, on the three intercepts. None of these constraints led to statistically significant decrements in model fit relative to the baseline model, yet the final model still fit the observed data poorly, $\chi^2(9, N = 180) = 40.58$, $p < .001$; IFI = .86; RMSEA = .14; 90% CI = .10, .19. These results strongly suggest that the simplex model does not provide an acceptable reproduction of the observed covariances and mean structure of antisocial behavior over time.

The One-Factor Latent Curve Model: Antisocial Behavior

Given the clear rejection of the autoregressive simplex structure of the relations among the four antisocial measures over time, we turn to a one-factor random intercept model. This one-factor model is an intercept-only latent curve model and is functionally equivalent to a one-factor repeated-measures analysis of variance with random effects (Bryk & Raudenbush, 1992). Unlike the simplex model, in which each later measure is influenced only by the immediately preceding measure, the random-intercept model hypothesizes that all repeated measures are equally influenced by a single underlying latent factor and that it is this shared influence that is responsible for the observed covariance and mean structure. This model also implies that there is a stable component underlying the repeated measures over time that is not changing as a function of time. Given the earlier rejection of the equal-means model, we do not expect this model to fit well. Consistent with this prediction, the one-factor intercept model fit the observed data poorly, $\chi^2(8, N = 180) = 41.8$, $p < .001$; IFI = .85; RMSEA = .15; 90% CI = .11, .20). The latent intercept was characterized by both a significant mean ($\hat{\mu}_\alpha = 1.96$) and variance ($\hat{\psi}_\alpha = 2.12$), suggesting an important underlying stable component of the four measures. However, given the poor model fit, additional components of growth are likely necessary.

The Two-Factor Latent Curve Model: Antisocial Behavior

We re-estimated the random intercept model with the addition of a second latent factor to account for potential systematic change as a function of time. This second factor is a *slope* factor in latent curve analytic terms. The addition of this second factor led to a significant improvement in model fit over the one-factor model, $\chi^2(6, N = 180) = 14.8$, $p = .022$; IFI = .96; RMSEA = .09; 90% CI = .03, .15. The intercept and slope factors had significant means ($\hat{\mu}_\alpha = 1.73$ and $\hat{\mu}_\beta = .17$, respectively) and variances ($\hat{\psi}_\alpha = 1.67$ and $\hat{\psi}_\beta = .20$, respectively), indicating that there not only is evidence for a meaningful starting point and positive rate of linear change in antisocial behavior over time but also substantial individual variability in these growth factors.

The Two-Factor Latent Curve Model With Autoregressive Parameters: Antisocial Behavior

Up to this point, we have treated the autoregressive simplex and latent curve models as independent approaches to modeling the relations among the repeated measures of antisocial behavior over time. However, given that both modeling strategies analyze the observed covariance matrix and mean vector, it seems logical to expect that these apparently separate models may share a common parameterization. As a first step in working toward the synthesizing of the autoregressive and latent curve model, we estimated the two-factor latent curve model with the inclusion of the autoregressive parameters between the time-adjacent measures of antisocial behavior. This model is meant to simultaneously capture two components of change over time. The latent variable parameters represent individual variability in continuous rates of change over time, whereas the autoregressive parameters represent group-level influences present at the prior time point. We freely estimated the factor loadings between the Time 1 measure of antisocial behavior and the intercept and slope factors because of the possibility that the loadings may take on values other than 0 and 1 as described in Equations 4.18 and 4.19.[2] The estimated model with the autoregressive parameters did not result in a significant improvement in model fit, $\chi^2(1, N = 180) = 3.12$, $p = .08$; IFI = .99; RMSEA = .11; 90% CI = 0, .25, beyond that for the latent curve model only. Equality constraints on all three autoregressive parameters did not significantly degrade model fit, $\chi^2(3, N = 180) = 3.3$, $p = .35$; IFI = .99; RMSEA = .02; 90% CI = 0, .13, and none of the individual autoregressive estimates significantly differed from zero ($p > .10$). On the basis of these results, we concluded that the observed covariance and mean structure are best captured with the two-factor latent curve model without autoregressive structure.

[2]In the presence of a significant ρ parameter, a more formal evaluation of these factor loadings would include the imposition of a nonlinear constraint on λ as a function of ρ. Given the near-zero estimates of ρ, we did not proceed with the imposition of these nonlinear constraints.

Tests of Equality of Means Over Time: Depressive Symptomatology

Before estimating the full multivariate model, we must repeat the above univariate analyses for the four repeated measures of depressive symptomatology. We begin by testing for the equality of the means of depressive symptomatology across the four time periods using the same model we used for antisocial behavior. The first model had all parameters freely estimated and fit the data perfectly given the model is just identified. A re-estimated model had an equality constraint placed on the observed means over the four time points. Unlike antisocial behavior, the restriction of equal means for the four depression measures was not rejected, $\chi^2(3, N = 180) = 4.89$, $p = .18$; IFI = .99; RMSEA = .06; 90% CI = 0, .15, indicating a single mean estimate for all four time points.

The Simplex Model With Means: Depressive Symptomatology

Next, we estimated the autoregressive simplex model. The baseline model had no imposed equality constraints and fit the observed data poorly, $\chi^2(3, N = 180) = 28.7$, $p < .001$; IFI = .80; RMSEA = .22; 90% CI = .15, .29. Equality constraints on the regression parameters, then on the disturbances and finally on the intercepts, did not result in a significant decrement in model fit, although the final model still fit the data poorly, $\chi^2(9, N = 180) = 37.7$, $p < .001$; IFI = .77; RMSEA = .13; 90% CI = .09, .18. So even though the means of depressive symptomatology were equal over time, the simplex model still resulted in a poor fit to the observed data.

The One-Factor Latent Curve Model: Depressive Symptomatology

Given the poor fit of the simplex model, we then tested a one-factor latent variable model in which the factor mean and variance were freely estimated but all factor loadings were fixed to 1. This model fit the data quite well, $\chi^2(8, N = 180) = 11.51$, $p = .17$; IFI = .97; RMSEA = .05; 90% CI = 0, .11, suggesting that the observed covariance and mean structure are well replicated given the presence of a single random-intercept factor. There was a significant mean of the latent factor, suggesting meaningful levels of depressive symptomatology in the sample, and there was a significant variance, suggesting meaningful individual variability in these levels of depression.

The Two-Factor Latent Curve Model: Depressive Symptomatology

To test if an additional factor was necessary to account for systematic change over time, we added a linear slope factor to the above model. The addition of this factor did not significantly improve the overall model fit, and the mean and variance of the slope factor did not significantly differ from zero. This suggests that although there is a random-intercept component underlying the four depressive symptomatology measures, there is no corresponding random-slope component. Indeed, this finding is consistent with the initial equal-means model that suggests that the means did not vary as a function of time. Thus, there is no evidence to retain the linear slope factor.[3]

The Bivariate Autoregressive Crosslagged Model: Antisocial Behavior and Depressive Symptomatology

The motivating goal of these analyses is to empirically examine the relation between antisocial behavior and depressive symptomatology over time. Now that we better understand the characteristics of stability and change within each construct, we can proceed to the simultaneous evaluation of these constructs across the four time periods. We start by combining the two simplex models described above; this allows for the introduction of the important crosslagged parameters across construct and across time. Although this model was built in a series of sequential steps (see Curran et al., 1997, for more details), only the final model is presented here, which was found to fit the data poorly, $\chi^2(26, N = 180) = 95.1$, $p < .001$; IFI = .86; RMSEA = .12; 90% CI = .10, .15. The model includes all imposed equality constraints with the exception of equalities on the intercepts within each construct at Times 2, 3, and 4. Findings indicate that there were large and significant positive regression parameters between time-adjacent measures within each construct. Furthermore, there were positive significant covariances of the disturbances within each time across the two constructs. Finally, whereas earlier depressive symptomatology did not predict later antisocial behavior, earlier antisocial behavior did significantly and positively predict later depressive symptomatology. Both of these crosslagged effects are evident even after controlling for the previous measure of each construct. For example, Time 1 antisocial behavior predicted Time 2 depressive symptomatology above and beyond the effects of Time 1 depressive symptomatology.

These results thus suggest that there is a relation between depressive symptomatology and antisocial behavior over time, but only in that earlier antisocial behavior predicts later depressive symptomatology, not vice versa. However, two important issues remain. First, although these crosslagged parameters were significant, these are drawn from a model that fits the observed data quite poorly, and biased parameter estimates and standard errors are likely (e.g., Kaplan, 1989). Second, previous analyses indicated that both antisocial behavior

[3]Given the pattern of means that first increased and then decreased as a function of time, we estimated an additional model that included three growth factors: an intercept, a linear slope, and a quadratic slope. However, there was a nonsignificant mean and variance for the quadratic factor, indicating that there was not a meaningful curvilinear component to changes in depression over time.

and depressive symptomatology are characterized by one or more random growth parameters, influences that are not incorporated into this bivariate fixed-effects simplex model. To address these issues, we will now turn to a bivariate latent curve model.

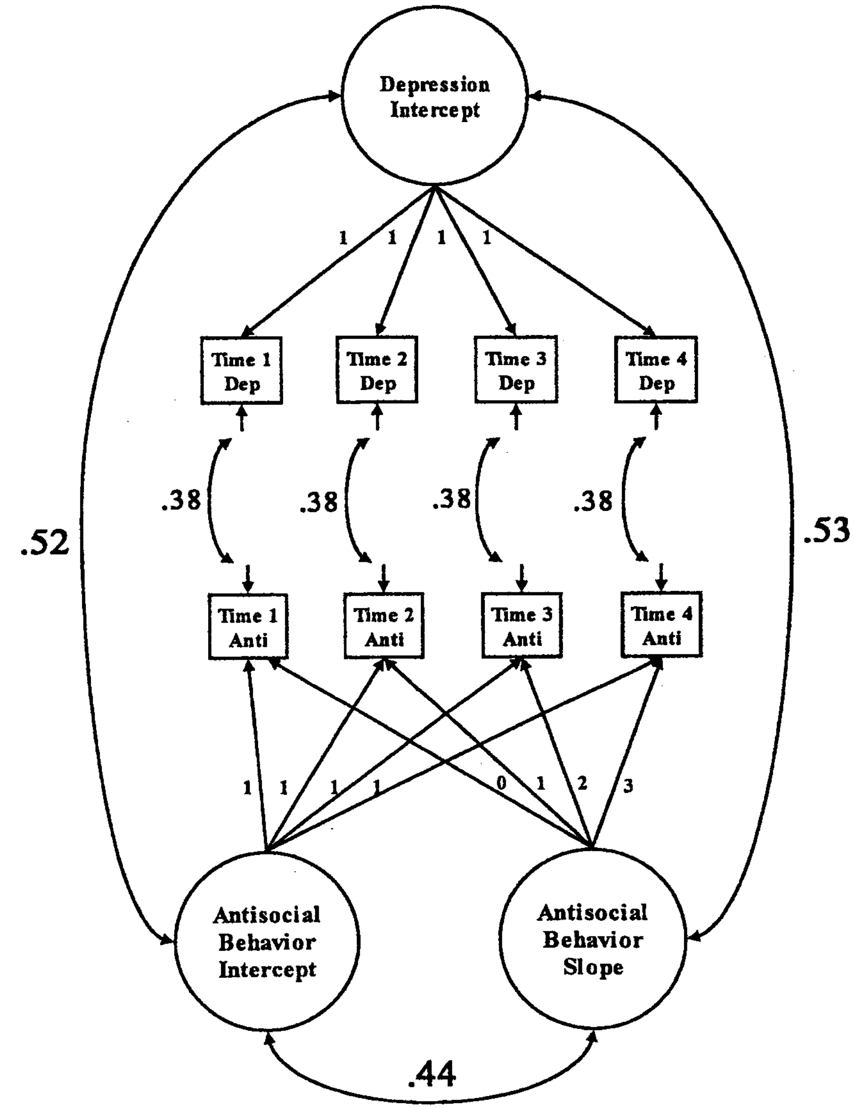
The Bivariate Latent Curve Model: Antisocial Behavior and Depressive Symptomatology

We first estimated our baseline bivariate latent curve model that consisted of the combination of the two univariate latent trajectory models from above (see Figure 4.7). Additional parameters included the three covariances among the latent growth factors and within time covariances for the time-specific residuals. As expected, this baseline model did not reflect adequate fit, $\chi^2(23, N = 180) = 54.45$, $p < .001$; IFI = .94; RMSEA = .09; 90% CI = .06, .12. We introduced a series of equality constraints on the variances of the disturbances across time and within construct, as well as the covariances within time and across construct. None of these equality constraints resulted in a significant deterioration in model fit, and the final model fit the data moderately well, $\chi^2(32, N = 180) = 62.9$, $p < .001$; IFI = .94; RMSEA = .07; 90% CI = .05, .10. Parameter estimates indicated that the three latent factors were positively and significantly correlated with one another (correlations ranged between .44 and .53), indicating that there was meaningful overlapping variability in the components of growth underlying the repeated measures of antisocial behavior and depressive symptomatology. Of greatest interest was the significant positive relation between the depressive symptomatology intercept and the antisocial slope. This correlation suggests that individual differences in the stable component of depressive symptomatology are positively associated with increases in antisociality over time. That is, on average, children with a higher stable component of depressive symptomatology tended to report steeper increases in antisocial behavior relative to children who reported lower stable levels of depressive symptomatology.

This finding has direct implications for our research hypotheses of interest. Namely, there does appear to be a relation between antisocial behavior and depressive symptomatology over time. However, this finding highlights one of the limitations of this model. Although empirical evidence suggests these two constructs are related over time, this relation holds only for the stable continuous component underlying antisociality and depression over time—that is, it is difficult to infer temporal ordering or a possible direction of influence; we can only observe that these two constructs are related in a potentially important way. To allow for the simultaneous influence of both the random underlying components of change with the time-specific fixed components of change, we now combine the crosslagged effects from the simplex model with the growth factors of the latent trajectory model.

FIGURE 4.7

Standard bivariate latent curve model without lagged effects between indicators. Dep = depression; Anti = antisocial behavior.



The Bivariate Crosslagged Latent Curve Model: Antisocial Behavior and Depressive Symptomatology

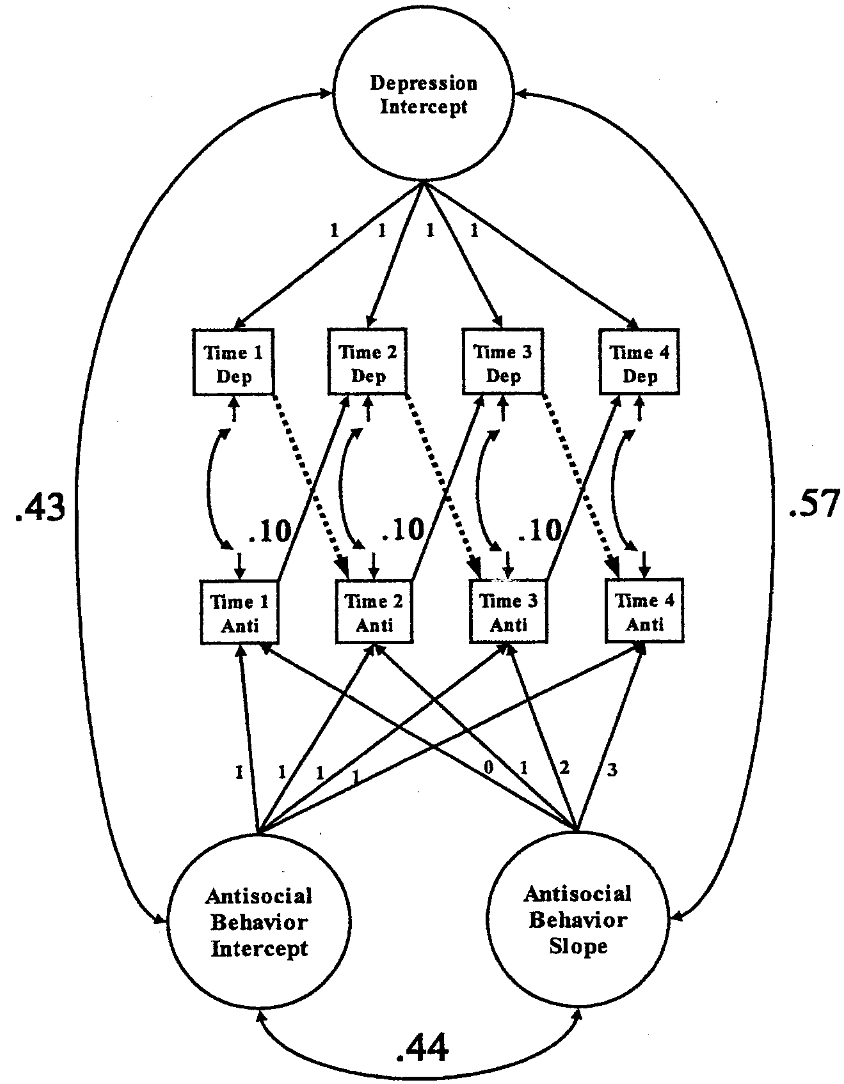
We extended the latent curve model by adding crosslagged effects in which we regressed a later measure of one variable onto the prior measure of the other variable. These parameters resulted in a significant improvement in model fit, $\chi^2(26, N = 180) = 54.9$, $p < .001$; IFI = .94; RMSEA = .08; 90% CI = .05, .11. Before interpreting the final model, we imposed additional equality constraints on the lagged effects, and none of the constraints resulted in a significant decrement to model fit. The final model fit the data moderately well, $\chi^2(30, N = 180) = 55.3$, $p = .003$; IFI = .95; RMSEA = .07; 90% CI = .04, .10, and is presented in Figure 4.8. All three growth factors were positively and significantly correlated with one another. Furthermore, although earlier measures of depression did not predict later levels of antisocial behavior, earlier levels of antisocial behavior did significantly predict later levels of depressive symptomatology. Note that this prospective lagged prediction is evident after the influences of the underlying latent growth processes have been partialed out. Thus, this bivariate latent trajectory model with lagged effects allows for the estimation of both the stable component of development over time (as captured in the latent factors) and time-specific differences in antisocial behavior or depressive symptomatology at any given time point. This is an extremely important combination of influences that neither the simplex nor the latent trajectory model allows when taken alone.

Summary of Substantive Findings Relating to Antisocial Behavior and Depressive Symptomatology

The series of simplex and latent curve models provides a great deal of insight into the relations between antisocial behavior and depressive symptomatology over an 8-year period in this sample of children. First, we found developmental changes in antisocial behavior to be positive and linear for the overall group. In addition, there was a significant amount of variability in both the starting point and the rate of change of antisociality over time; some children were increasing more steeply, some less steeply, and some not at all. Second, a similar systematic developmental trajectory over time did not exist for depressive symptomatology. There was a stable component of depressive symptomatology that was characterized by significant individual variability indicating that some children were reporting higher levels of depression over time whereas others were reporting lower levels or none at all. However, there was not a significant relation between depressive symptomatology and time. Third, both modeling approaches indicated that antisocial behavior and depressive symptomatology

FIGURE 4.8

Bivariate simplex latent curve model including lagged effects between indicators. Dep = depression; Anti = antisocial behavior.



were related over time in important ways. The latent curve model suggests that children who report higher baseline levels of depression tend to report steeper increases in antisocial behavior over time. However, the crosslagged models suggest that earlier levels of antisocial behavior are associated with later levels of depression but not vice versa.

It was only the synthesized autoregressive latent curve model that allowed for a simultaneous estimation of both of these stable and time-specific effects. The latent curve component of the model estimated the portion of variability in the repeated measures that was associated with a continuous underlying developmental trajectory of antisocial behavior or depressive symptomatology. At the same time, the simplex crosslagged effects indicated that, after controlling for the variability associated with the developmental trajectories, earlier antisociality predicted later depression, but earlier depression did not predict later antisociality. Taken together, these models provide important information that helps further our understanding about these complicated developmental issues.

Extensions of the Crosslagged Latent Curve Model

The proposed modeling strategy is expandable in a variety of ways. For example, one could regress the latent growth factors onto exogenous explanatory variables to better understand individual differences in change over time. In analyses not presented here because of space constraints, we regressed the growth factors defining antisocial behavior and depressive symptomatology onto family-level measures of emotional and cognitive support in the home (see Curran & Bollen, 1999, for details). The results suggest intriguing relations between these home support measures and individual differences in developmental trajectories over time. Additional explanatory variables could be incorporated to model variability both in the latent growth factors as well as directly in the repeated measures over time.

A second important extension uses the strength of the SEM framework for analyzing interactions as a function of discrete groups. An important example of this would be the examination of potential interactions between a child's gender and the relation between antisociality and depression over time. Again, in additional analyses not reported here, we evaluated gender differences in these models using a multiple-group estimation procedure, and the results suggest that the relation between antisociality and depression may interact as a function of gender (see Curran & Bollen, 1999). These techniques could be extended further by combining the synthesized models discussed here with the analytic methods proposed by Curran and Muthén (1999) and B. O. Muthén and Curran (1997), which would allow for the evaluation of whether two developmental processes could be "unlinked" from one another over time because of the implementation of a prevention or treatment program.

A third extension would be to further capitalize on the strengths of the SEM approach and to use multiple-indicator latent factors to define the constructs of interest within each time point. For example, instead of using a single scale score to measure antisocial behavior or depressive symptomatology, analysts could use latent factors to estimate these constructs and would thus be theoretically free from measurement error. Given the difficulty of measuring many constructs in the social sciences, incorporating the presence of measurement error is an important aspect in any modeling approach; Sayer and Cumsille (chapter 6, this volume) explore this issue.

Finally, although we found that earlier antisocial behavior was related to later depressive symptomatology, little is known about precisely why this effect exists. To better understand the relation between these two constructs over time, it would be very important to include potential mediators that might account for this observed effect. For example, it may be that higher levels of antisocial behavior are associated with greater rejection from positive social groups, and this social rejection is associated with greater isolation and depression. These models could be directly extended to include the influences of social rejection given the availability of appropriate data.

Conclusion

The simplex model and the latent curve model are both important tools for understanding change over time. However, each approach is limited in key ways that preclude drawing comprehensive inferences about change and development from observed empirical data. Although these limitations are difficult to overcome when considering only one modeling approach or the other, we believe that significant improvements are possible by combining elements drawn from each analytic approach to create a more general model of development and change. Of course, there are a variety of situations in which the simplex model or the latent curve model taken alone is well suited to evaluate the particular research hypotheses at hand. An advantage of the proposed framework is that under such conditions, the synthesized model directly simplifies to either the standard simplex model or the latent curve model (Bollen & Curran, 2000). However, under conditions in which there is interest in both continuous underlying trajectories and time-specific influences across constructs, we believe that the proposed modeling approach provides a powerful and flexible tool to help elucidate these complex relations over time.

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