chapter five

Testing turning points using latest growth curve models: Competing models of substance abuse and desistance in young adulthood

Andrea M. Hussong
University of North Carolina at Chapel Hill

Patrick J. Curran
University of North Carolina at Chapel Hill

Terrie E. Moffitt
University of London and University of Wisconsin–Madison

Avshalom Caspi
University of London and University of Wisconsin–Madison

Contents

Introduction

Multiple terms denote the twists and turns that mark the life course of youth as they move from risk to resilience or maladjustment. From Rutter (1987), we have vulnerabilities and protective factors that denote mechanisms turning these adaptive trajectories away from or toward resilience, respectively. Among those youth who may not start from a clear point of risk, however, evidence of psychopathology clearly indicates the potential for turning points to lead away from an expected course of healthy adjustment. Within the study of antisocial behavior, theoretical writings and empirical studies recognized these mechanisms as influencing trajectories of problem behaviors. The concept of *snares*, offered by Moffitt (1993), fills a unique niche in this literature and refers to those mechanisms responsible for prolonging what might otherwise be a developmentally normative pattern of desisting antisocial behavior. Snares thus define a turning point away from an expected course of adaptation. Given the pattern of desistance that typifies the course of antisocial behavior in young adulthood, factors that serve to prolong antisocial behavior during this developmental period inform our search for ensnaring mechanisms. The current chapter examines the role of a potential snare, substance abuse, in interfering with the normative pattern of desistance from antisocial behavior during young adulthood.*

Testing snares

The test of ensnaring mechanisms presents several methodological and analytic challenges. One set of guidelines for conducting these tests came from Rutter (1996), based on the premise that snares constitute one form of turning points. In this framework, methodological considerations include the need (1) to measure intraindividual change over time, (2) to relate that change to specific circumstances, (3) to focus on a relevant segment of the population in which there has been an opportunity for the hypothesized variable to operate, and (4) to rule out heterotypic continuity. In this chapter, we provide one example of how this framework might be used to guide the test of snares that have an impact on trajectories of antisocial behavior in young adulthood.

The first consideration is the need to measure intraindividual change over time, such that turning points are references with respect to individual

* This chapter expands on previously reported analyses in the work of Hussong, Curran, Moffit, Caspi, and Carrig (2005).

rather than group patterns of change. Multilevel modeling (also known as hierarchical linear modeling and mixed modeling) and latent growth curve (LGC) modeling (within the structural equation modeling tradition) are both powerful methods for modeling interindividual differences in intraindividual change over time. Although these two methods have substantial overlap in their capabilities, they also have some notable areas of difference (e.g., Bauer, 2003; Curran, 2003; Willett & Sayer, 1994). Through the inclusion of either time-varying or time-invariant predictors within either of these models, we can address the second consideration of testing turning points, namely, the need to relate intraindividual change to specific circumstances. To retain focus in our demonstration, we adopt the latent curve-modeling approach to permit us greater flexibility in model testing and comparison.

The third consideration of testing turning points is the need to evaluate the hypothesis within a relevant population. To examine the ensnaring mechanism of substance abuse within antisocial behavior, we must study (1) those who are likely to show normative decrements in antisocial behavior over time in the absence of the snare and (2) those who are likely to evidence the snare and thus to have the opportunity to show deviations from the expected pattern of desistance. Two consistent findings help us define a population of interest meeting these requirements. First, robust support for the age–crime curve indicates that both incidence and prevalence of crime are highest during late adolescence and begin to drop off only in young adulthood (Blumstein, Cohen, & Farrington, 1988; Farrington, 1986). However, several theorists posit that this population curve comprises interindividual differences in intraindividual change over time (Laub & Sampson, 2001; Moffitt, 1993), and emerging evidence supports this contention (Bushway, Piquero, Broidy, Cauffman, & Mazerolle, 2001; Piquero, Blumstein, et al., 2001). Second, within this developmental period, gender differences in rates of substance abuse indicate that men are more likely than women to evidence alcohol and substance disorders (Hanna & Grant, 1997; Robins & Reiger, 1991). Together, this evidence suggests that men passing through the period of young adulthood form an ideal population within which to test the snares hypothesis.

The fourth consideration in testing turning points is the need to rule out heterotypic continuity. To broaden this consideration within the realm of classic methodological design (Shadish, Cook, & Campbell, 2001), evidence for the snares hypothesis is strongest if we are able to rule out other alternative relations between substance abuse and antisocial behavior. To meet this objective, we first must clearly define the concept of developmental snares as it may be tested within the LGC framework. In this

regard, we propose two hypotheses in which substance abuse acts as a snare that serves to entrench young adult men in prolonged patterns of antisocial behavior during a period of normative desistance.

The first, captured by the "launch" hypothesis, posits that substance abuse early in young adulthood may both identify young men who are on a *long-term* course of elevated antisocial behavior and set men on such a course. According to this mechanism, substance abuse on entry into young adulthood defines different trajectories of antisocial behavior over time. Perhaps the most common method for examining individual development over time, the launch method, is "analogous to a catapult, in which the initial forces of the contextual antecedent are the major determinants of the shape of the curve of the outcome" (Kinderman & Skinner, 1992, p. 166). In such models, launching factors serve as distal predictors of change over time under the assumption that such time-lagged influences are more salient predictors of course than are time-varying or contextual factors. The role of such distal factors, though often described in causal terms, may also be one of early identification, which belies the effects of selection resulting from prior developmental processes. In either case, when applied to the study of crime desistance and substance abuse in young adulthood, this model posits that early signs of substance abuse predict maintenance of elevated antisocial behavior over young adulthood. This prediction is thus concerned with individual differences in the intercepts and slopes characterizing the trajectories of antisocial behavior over time (see Figure 5.1).

Previous studies showed support for the launch model as an explanation for antisocial behavior during adolescence, when such trajectories reflect a rise in antisocial behavior. For example, Munson, McMahon, and Spieker (2001) showed that greater maternal depression predicted steeper escalations in children's externalizing symptoms over time, especially among children with avoidant insecure attachments. However, to our knowledge, the role of substance abuse as a launching factor in young adulthood, when the expected pattern is desistance, has yet to be examined.

The second, for which we retain the term *snares hypothesis*, posits that substance abuse acts through a series of proximal influences on crime desistance such that *short-term* alterations in the course of antisocial behavior are impacted by substance abuse. Snares may then be defined in reference to protective factors as studied in the work of developmental criminologists and life course researchers (Laub & Sampson, 2001; Moffitt, 1993). "Protective" factors hasten the process of desistance among men at risk for continued antisocial behavior. Supporting the role of protective factors in crime desistance, several studies suggested that reduced involvement in antisocial behavior coincides with entry into good

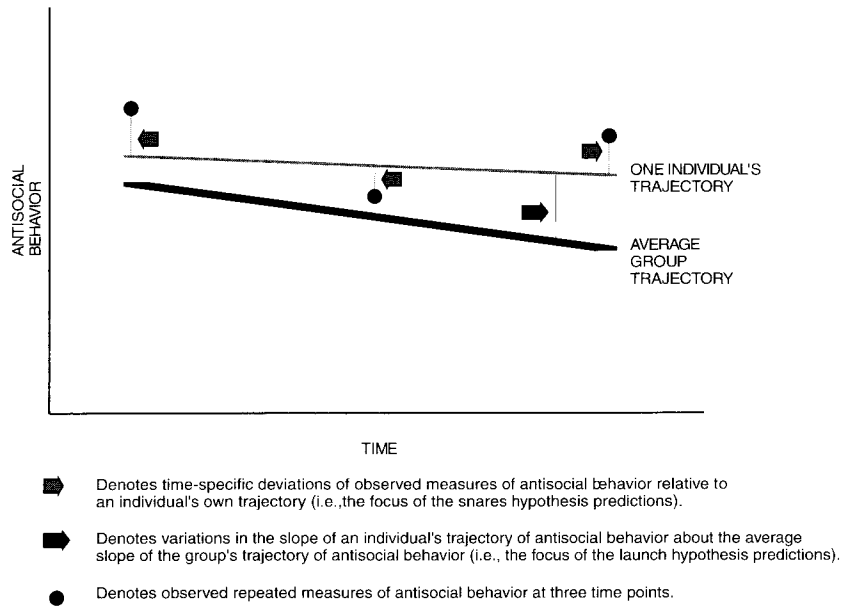

Figure 5.1 Contrast of snares versus launch hypotheses.

marriages and good jobs during young adulthood (Horney, Osgood, & Marshall, 1995; Laub, Nagin, & Sampson, 1998; Quinton, Pickles, Maughan, & Rutter, 1993). In contrast, "ensnaring" factors interfere with the normative deceleration of antisocial behavior that is observed in the population. As defined here, snares exert a contemporaneous or short-term effect on antisocial behavior, such that the local effects of snares alter the normative course of antisocial behavior when they or their sequelae are present. Unlike protective factors, the importance of snares in the maintenance of antisocial behavior has rarely been empirically evaluated (though see Piquero, Brame, Mazerolle, & Haapanen, 2001).

Ensnaring factors and protective factors are thought to play different roles in modifying antisocial behavior during young adulthood (Rutter, 1987). While the protective influences offered by a good marriage or a good job may serve to actively promote desistance during young adulthood, snares may serve to actively retard desistance during young adulthood. As such, a snare is posited to be more than merely the opposite of a protective factor. By distinguishing between these two influences, we are able to differentiate how the presence of various factors has a direct impact on young adults' lives. This distinction also has important potential implications for interventions. For example, desistance research

that focuses on protective factors in marriage and at work necessarily suggests that interventions should focus on acquiring and promoting new adult roles and responsibilities. In contrast, research that focuses on snares suggests that, if snares can be identified, interventions should focus on removing those barriers to crime desistance that are likely to impede a healthy transition to adulthood.

Despite their potential importance for interventions targeting antisocial behavior, to our knowledge, little research has examined such protective factors, and no research has directly tested the snares hypothesis. The paucity of studies focusing on these factors is expected given the relatively recent introduction of the constructs to the literature and the lack of data sets that can meet demands for studies of desistance to include self-report data assessed repeatedly during young adulthood. Nonetheless, substance abuse has been hypothesized to be a potent snare (Moffitt, 1993). Several mechanisms may account for the ensnaring role of substance abuse within trajectories of antisocial behavior.

First, substance abuse has been associated with difficulties in conventional adult roles, the same protective factors that have been found to precede desistance in antisocial and criminal behavior (e.g., good marriages; Bachman, Wadsworth, O'Malley, Schulenberg, & Johnston, 1997; Leonard & Rothbard, 2000). Second, substance abuse has been associated with interrupted education and incarceration (Sher & Gotham, 1999; Vaillant, 1995), both of which have been proposed as additional snares forestalling normative desistance. Third, substance abuse may reflect a physiological dependence that motivates antisocial behavior necessary to purchase, obtain, and use substances. Fourth, the social nature of substance abuse during young adulthood may serve to maintain common activities and ties with a deviant peer context. And, fifth, the disinhibiting properties of alcohol and other drugs may increase the odds that poor judgment and impulsivity will lead to antisocial activities.

Each of these pathways may result in greater antisocial behavior for those who abuse substances during a developmental period in which most individuals are curbing their involvement in deviant behavior. Across these pathways, substance abuse may serve as either a marker variable for a process influencing substance abuse or as either a direct or indirect causal factor. Our goal here is not to distinguish these roles of substance abuse but rather to examine whether there is support for substance abuse to function in any one of these roles based on its prediction of crime desistance.

In contrast to the launch hypothesis, this basic prediction of the snares hypothesis is concerned with time-varying deviations in antisocial behavior away from the expected pattern of desistance over time. Whereas snares are expected to alter time-specific variation in antisocial behavior

within the course of desistance, launching factors provide a more global prediction in which substance abuse alters the actual trajectory of antisocial behavior (see Figure 5.1). However, the launch and snares hypotheses are not necessarily incompatible; for example, substance abuse early in young adulthood may both decelerate an individual's overall pattern of crime desistance relative to others during this period (a launch prediction) and increase the likelihood of antisocial behavior within certain points in young adulthood relative to that individual's expected level of antisocial behavior (a snares prediction).

In light of these two definitions of snares, we now define an alternate hypothesis that addresses Rutter's fourth consideration for appropriately testing turning points, namely, ruling out the potential for heterotypic continuity. Both the launch and snares hypotheses posit that, during the transition from adolescence to adulthood, substance abuse serves to perpetuate continuity in antisocial behavior over time. An alternate, more parsimonious, explanation for the observed relation between substance abuse and antisocial behavior over time is offered by "general deviance" or "common propensity" hypotheses based on a sociogenic approach. These theories take as their starting point the empirical observation that problem behaviors in adolescence and adulthood are positively correlated (e.g., Elliott, Huizinga, & Menard, 1989; Osgood, Johnston, O'Malley, & Bachman, 1988). The fact that multiple problem behaviors tend to cluster among the same persons has led psychologists, sociologists, and criminologists to theorize that correlated problem behaviors (e.g., alcohol abuse, drug abuse, criminal participation) may have a common etiology. For example, problem behavior theory (Donovan & Jessor, 1985; Jessor & Jessor, 1977) posits that many different deviant behaviors form a "syndrome" that is caused by an underlying latent trait called *psychosocial proneness*. Similarly, Gottfredson and Hirschi (1990) hypothesized that participation in various correlated antisocial behaviors is caused by the same latent propensity factor at every age across the life span. Rather than substance abuse serving to influence continuity in antisocial behavior over time, general deviance or common propensity theories suggest that continuity in both substance abuse and antisocial behavior is a manifestation of an underlying propensity to engage in deviant activities.

General deviance models offer testable hypotheses about the structure of correlations among problem behaviors during adolescence and young adulthood. For example, these models predict that elevated substance abuse is an index of a heightened propensity toward deviancy that is similarly manifested in elevated antisocial behavior within time. Likewise, general deviance models predict that changes in substance abuse and antisocial behavior over time will mirror one another in synchrony because they are both thought to be developmental manifestations of the

same underlying propensity toward problem behaviors. This prediction thus defines substance abuse and antisocial behavior as multiple indicators of a single trajectory. We test this model using a multiple-indicator, second-order LGC model. Because a general theory is the most parsimonious model, it is a compelling alternate explanation to the snares hypothesis (Osgood & Rowe, 1994).

Chapter aims

The aims of this chapter are thus to demonstrate the use of LGC techniques for testing turning points, defined as the ensnaring role of substance abuse within the expected pattern of desisting antisocial behavior in young adulthood. Reflecting Rutter's (1996) guidelines for conducting such tests, we adopted statistical techniques that model intraindividual change over time as applied to a longitudinal data set able to reflect these patterns of change; we defined our sample as men passing through young adulthood, a developmental period when desistance is normative and thus the role of substance abuse as a snare is more obvious; we considered several alternate hypotheses, including that mirroring the assumption of heterotypic continuity. Specifically, our analyses tested (1) the launch hypothesis, in which substance abuse is tested as a time-invariant predictor of intraindividual differences in the intercepts and slopes defining trajectories of antisocial behavior over time; (2) the snares hypothesis, in which substance abuse is tested as a time-varying predictor of elevated antisocial behavior against an expected pattern of desistance over time; and (3) a general deviance hypothesis, in which substance abuse and antisocial behavior are multiple indicators of a single construct characterized by an underlying trajectory of problem behavior spanning young adulthood.

Sample and measures

Participants are members of the Dunedin Multidisciplinary Health and Development Study, a longitudinal investigation of health and behavior in a complete birth cohort (Silva & Stanton, 1996). The study members were born in Dunedin, New Zealand, between April 1972 and March 1973. Of these, 1,037 children (91% of eligible births; 52% males) participated in the first follow-up assessment at age 3, and they constitute the base sample for the remainder of the study. Cohort families represent the full range of socioeconomic status in the general population of New Zealand's South Island and are primarily white; fewer than 7% self-identified at age 18 as Maori or Pacific Islanders. Assessments have been conducted at ages 3 (n = 1,037), 5 (n = 991), 7 (n = 954), 9 (n = 955), 11 (n = 925), 13 (n = 850), 15 (n =976), 18 (n = 993), 21 (n = 961), and at age 26 (n = 980, 499 males; 96%

of living cohort members). The current study focused on self-report data gathered from men at ages 18, 21, and 26. Rates of diagnosed conduct disorder, substance dependence, and self-reported delinquent offending in New Zealand were similar to those obtained for surveys of same-age epidemiological samples in the United States; for documentation supporting generalization from the Dunedin cohort to other settings, see the work of Moffitt, Caspi, Rutter, and Silva (2001).

For the current study, men with incomplete data at age 18 (n = 64) or who were missing data at both ages 21 and 26 (n = 10) were omitted from analyses (final n = 461 of 535 male respondents at age 18, including 438 with completed data and 23 with partially missing data). The t tests showed no significant differences between retained and omitted cases, where available, on antisocial behavior, alcohol symptoms, or marijuana symptoms at ages 18, 21, or 26. Detailed analyses comparing groups of study members who did not take part in assessments versus those who did on a variety of family and individual characteristics have revealed no group differences (as reported in Moffitt et al., 2001).

Alcohol abuse and marijuana abuse were assessed by symptoms from the Diagnostic Interview Schedule (DIS; Robins, Helzer, Cottler, & Goldring, 1989). The DIS was administered to participants at ages 18 (DIS-IIIR), 21 (DIS-IIIR), and 26 (DIS-IV). Antisocial behaviors were assessed via the self-report offending interview, which ascertains illegal behaviors and conduct problems (Moffitt, Silva, Lynam, & Henry, 1994). Antisocial behaviors and substance abuse symptoms were ascertained on the same day but in separate, counterbalanced sessions conducted by interviewers who were blind to the other assessment. Because we were interested in examining changes in both the mean and variance of behavior over time, continuity in item content for each scale was very important. For this reason, parallel items were selected from each assessment age to measure antisocial behavior, alcohol abuse, and marijuana abuse.

We used eight parallel items assessing conduct disorder (*Diagnostic and Statistical Manual of Mental Disorders* [*DSM-IV-TR*], American Psychiatric Association, 1994) to create a variety score for antisocial behavior within each period. Variety scores index the total number of different forms of antisocial behavior in which a participant has engaged as opposed to, for example, the total frequency of antisocial acts. Previous studies suggested that variety scores may better reflect the extent or severity of antisocial involvement, and these scores are consistent with a diagnostic approach to assessing conduct problems (Gottfredson & Hirschi, 1995; Robins, 1978). Our variety scores were the total number of forms of antisocial behavior in which each participant had engaged over the past 12 months. Eight forms of antisocial behavior were assessed: breaking and entering; destroying property (illegal acts of vandalism); fighting (simple assault, aggravated

assault, or gang fighting); setting fires (arson); lying (criminal fraud); stealing with confrontation (robbery); stealing without confrontation (criminal theft); and carrying or using a weapon. Psychometric properties of the resulting variables are reported in Table 5.1.

Nineteen items from the DIS assessed symptoms of alcohol abuse and dependence, and 10 items assessed symptoms of marijuana abuse and dependence across the three assessments. These symptoms largely reflect those for substance abuse and dependence as stated in the *DSM-IV-TR* (e.g., unable to stop using, tolerance, continued use despite health or social problems; American Psychiatric Association, 2000). Each symptom was coded as present or absent within the previous year. The total number of symptoms endorsed for each scale served as the alcohol abuse and marijuana abuse scores, respectively, for the current study. Table 5.1 contains psychometric properties for these variables.

Analytic strategy

To test our hypotheses, we examined a series of LGC models. LGC, also referred to as growth curve analyses or random effects modeling, extends latent variable analyses within the structural equation modeling framework to provide a flexible tool for testing hypotheses of change over time and of predictors of such change (McArdle, 1988; Meredith & Tisak, 1984, 1990). First, we estimated an unconditional linear growth model to examine whether the characteristics of individual trajectories of antisocial behavior varied across men. Second, we tested the launch hypothesis through a conditional latent trajectory model in which substance abuse at age 18 served as an exogenous predictor of change over time in antisocial behavior. Third, we tested the snares hypothesis through a time-varying covariate LGC that considered the repeated measures of substance abuse as time-varying covariates to test their time-specific influences on antisocial behavior above and beyond the influence of each individual's underlying trajectory of antisocial behavior. The time-varying covariate LGC allowed for a direct test of our hypothesis about developmental snares given the simultaneous estimation of (1) variability across men in individual trajectories of antisocial behavior and (2) the association of substance abuse with time-specific deviations away from this predicted trajectory for each man's antisocial behavior within time (see, e.g., Bryk & Raudenbush, 1992, p. 151; Curran & Hussong, 2002; Curran, Muthén, & Harford, 1998). Fourth, we tested the general deviance hypothesis using a second-order LGC as described by Bollen and Curran (2005) and McArdle (1988).

To avoid bias due to the limited attrition in the sample, we estimated all models using the direct maximum likelihood procedure available in Mplus (Muthén & Muthén, 1998) and thus included all cases who had

Table 5.1. Correlation Matrix

	Variable	1	2	3	4	5	6	7	8	9
1	Antisocial behavior at 18	—								
2	Antisocial behavior at 21	.56	—							
3	Antisocial behavior at 26	.51	.53	—						
4	Marijuana symptoms at 18	.55	.37	.36	—					
5	Marijuana symptoms at 21	.50	.54	.46	.55	—				
6	Marijuana symptoms at 26	.42	.40	.49	.42	.57	—			
7	Alcohol symptoms at 18	.53	.37	.35	.46	.42	.31	—		
8	Alcohol symptoms at 21	.44	.48	.34	.34	.53	.36	.52	—	
9	Alcohol symptoms at 26	.34	.32	.40	.20	.30	.48	.34	.46	—
	M	1.95	1.57	1.50	0.63	1.07	1.08	2.32	3.54	2.78
	SD	1.59	1.71	1.46	1.61	2.02	1.98	2.85	3.68	3.23
	Reliability	.67	.74	.67	.86	.86	.85	.82	.86	.84
	n	461	451	455	461	446	456	461	451	456

Note: Due to missing data, n = 440–461 across correlations reported above; all correlations are significant at $p < .0001$. M, mean; SD, standard deviation.

complete data at age 18 and at least one subsequent time point (final $n = 461$). The adequacy of model fit was evaluated using the likelihood ratio test (i.e., model χ^2) and associated p value. Given that our large sample size might lead to excessive power of the χ^2 test to detect even small misspecifications (MacCallum, 1990), we also used two incremental fit indices that are less dependent on sample size (Comparative Fit Index [CFI]; Bentler, 1990; Incremental Fit Index [IFI]; Bollen, 1989).

Trajectories of antisocial behavior in young adulthood

To examine the fixed and random components of growth in antisocial behavior, we estimated an unconditional LGC for the repeated measures of antisocial behavior reported at ages 18, 21, and 26. Two latent factors were estimated: one to define the intercept of the developmental trajectory of antisocial behavior (with all factor loadings fixed to 1.0) and one to define the linear slope of the trajectory (with factor loadings set to 0, 3, and 8 to define an annual metric of time). A mean was estimated for the intercept and slope factors, and these values represented the mean model-implied developmental trajectory pooled over all individuals. A variance was also estimated for the intercept and slope factors, representing the degree of individual variability in trajectories around the group mean values. The covariance between the two factors represented the covariation between initial level and rate of change. Larger variance estimates imply greater individual variability in the starting point and the rate of change over time. Finally, residual variances were estimated for each repeated measure, and these values represented variability in the time-specific measures not accounted for by the underlying random trajectories.

The unconditional LGC was estimated and found to fit the observed data well, with $\chi^2(1) = 9.31$, $p = .002$, IFI = .98, and CFI = .98. The means of the latent factors showed that the model-implied trajectory for the group was characterized by a significant intercept of 1.90 different types of antisocial behavior at the first time period ($t = 26.33$, $p < .001$) and a significantly decreasing slope of 0.05 units per year ($t = -5.84$, $p < .001$; see Figure 5.2). Thus, the model-implied mean rate of antisocial behavior significantly decreased from 1.90 to 1.50 types of behavior over the period of study. Further, significant variance estimates for both the intercept ($\hat{\psi} = 1.77$, $t = 8.96$, $p < .001$) and slope ($\hat{\psi} = 0.02$, $t = 2.69$, $p < .01$) factors indicated substantial interindividual variability in intraindividual developmental trajectories of antisocial behavior. Finally, the negative correlation between the intercept and slope factors ($r = -0.44$, $t = -3.20$, $p < .01$) indicated that higher initial values were associated with steeper decreases over time.

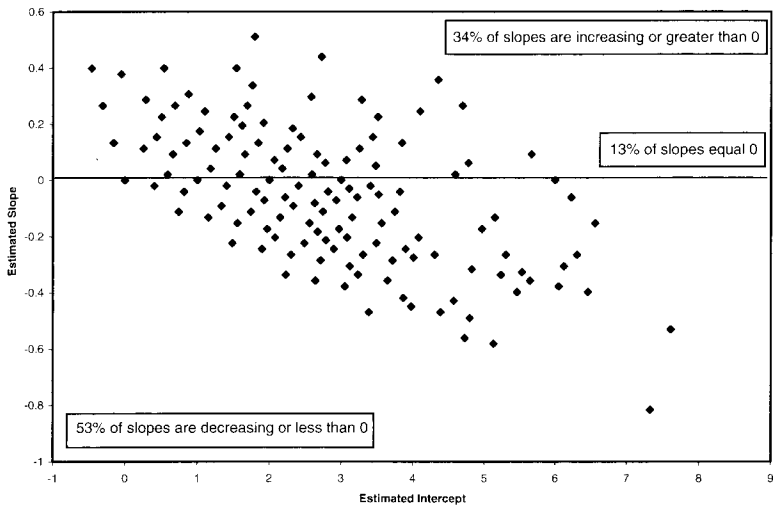

Figure 5.2 Pattern of trajectory parameters based of regression analyses within individual.

To examine whether men who were incarcerated during the 12-month periods before assessments at ages 21 and 26 accounted for this pattern of desistance (Piquero, Blumstein, et al., 2001), we reestimated these models by dropping the 14 men who had been incarcerated for more than 1 month prior to either assessment point. No meaningful changes in the findings occurred. We also reestimated these models to explore whether cases that showed a notable drop in antisocial behavior at age 21 relative to ages 18 and 26 served as influential outliers. Again, no meaningful changes in the findings occurred.

Overall, these results indicate that the mean developmental trajectory of antisocial behavior for the sample was significantly decreasing over time, consistent with previous findings on the age–crime curve. However, we also found that there were substantial individual differences in both the initial level and rate of change over time. Figure 5.2 depicts such variation by plotting the intercept and slope values for each participant's estimated trajectory against one another. (Note that these individual case-by-case estimates are for descriptive visualization purposes only.) These trajectories were estimated by conducting separate regression models within each case with complete data (see Carrig, Wirth, & Curran, 2004, for further details). As indicated, 53% of participants showed decreasing trajectories over time (i.e., slopes greater than 0), 13% showed no change, and 34% showed increasing slopes. These results further underscore the notable variation in individual trajectories. Although the growth trajectories

explained 70%, 49%, and 78% of the variance in the time-specific indicators of antisocial behavior at ages 18, 21, and 26, respectively, significant residual variances remained at each age. Thus, the underlying trajectory process is accounting for only a portion of the observed variability in antisocial behavior within each time period.

Test of the launch hypothesis

We next estimated a conditional LGC that tested the hypothesis that substance abuse at age 18 predicts a slowed or dampened pattern of desistance in the overall developmental trajectory of antisocial behavior over young adulthood. In other words, this model tested whether the magnitude of intercepts and slopes underlying antisocial behavior varied as a function of age 18 substance abuse. Both marijuana and alcohol abuse at age 18 were included as exogenous predictors of the intercept and slope factors defining the trajectories of antisocial behavior over ages 18, 21, and 26 (see Figure 5.3). The resulting model provided a good fit to the data, with $\chi^2(3) = 11.33$, $p = .01$, CFI = .99, and IFI = .99. Greater alcohol and marijuana abuse at age 18 both significantly predicted higher intercepts of the trajectories of antisocial behavior ($\hat{\beta} = .44$, $t = 9.63$, $p < .001$ and $\hat{\beta} = .40$, $t = 8.81$, $p < .001$, respectively). Both marijuana ($\hat{\beta} = -.21$, $t = -3.24$, $p < .001$) and alcohol ($\hat{\beta} = -.19$, $t = -2.86$, $p < .001$) abuse were also negatively related to the slope of the antisocial behavior trajectories, meaning that higher age 18 substance abuse predicted lower or increasingly negative slope values. Because such negative predictions may reflect a variety of relations, we further probed this effect by plotting model-implied trajectories of antisocial behavior one standard deviation above and below the mean of the predictor (i.e., substance abuse). This procedure is similar in many respects to probing interactions in multiple regression and formally recognizes the interaction inherent in these models between time and substance abuse, reflected in the growth factor prediction (Curran, Bauer, & Willoughby, 2004).

Results indicated that men with the highest substance symptoms at age 18 also showed steeper negative slopes in their trajectories of antisocial behavior ($M = -.03$, $-.02$, and $-.01$ for those high, medium, and low, respectively, in substance abuse at age 18), although this finding reflects a change of less than one type of antisocial acts difference between each of the three groups over the 8-year period. Importantly, probing of this relation by recoding the trajectory factors such that the intercept factor represents average antisocial behavior at age 26 also revealed that men who reported greater substance abuse at age 18 showed greater antisocial behavior at even the final time points ($\beta = .17$, $.09$; $z = 5.13$, 4.80, $p < .001$ for marijuana and alcohol abuse, respectively). Taken together, these results indicate that

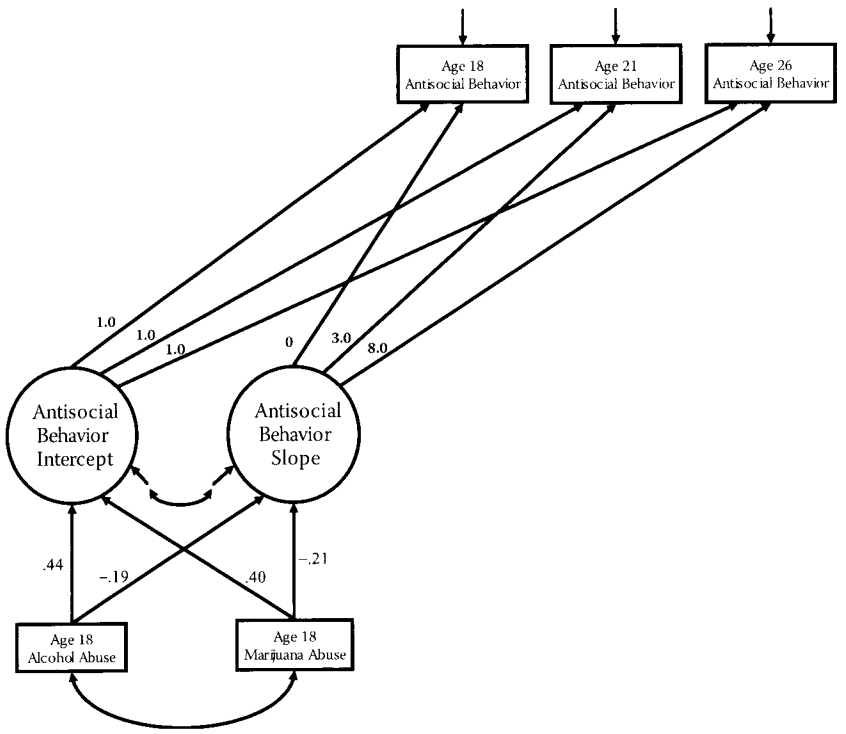

Figure 5.3 Conditional latent growth curve (LGC) testing launch hypothesis.

men elevated in substance abuse at age 18 reported higher initial levels of and steeper decreases in antisocial behavior over time but were significantly elevated in antisocial behavior across all periods of observation.

Test of the snares hypothesis

The extent to which substance abuse symptoms accounted for time-specific elevations in antisocial behavior over young adulthood was examined through a time-varying covariate model in which indicators of substance abuse (e.g., alcohol and marijuana abuse) at ages 18, 21, and 26 served as predictors of within-time individual variability in antisocial behavior that was not accounted for by the underlying individual trajectories of such behavior (see Figure 5.4 and Curran et al., 1998, for more detail). This strategy evaluated whether higher levels of substance abuse uniquely predicted a time-specific elevation or "shock" in antisocial behavior above and beyond what was expected based on the individual-specific underlying trajectory of antisocial behavior (Curran & Bollen,

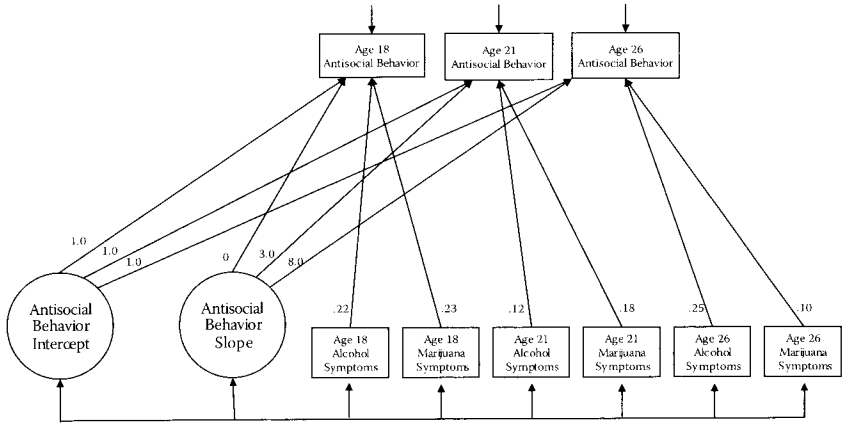

Figure 5.4 Time-varying covariate latent growth curve (LGC) testing snares hypothesis.

2001). In other words, significant prediction of time-specific measures of antisocial behavior, above and beyond the decreasing individual trajectories, from the measures of substance abuse indicate that substance abuse maintained a higher level of antisocial behavior than would be expected for that individual given his overall pattern of antisocial behavior during young adulthood. In this manner, the time-varying covariate model examined whether substance abuse was either a marker variable for a causal process or a causal variable itself in relation to antisocial behavior.

The hypothesized model with the time-varying effects of alcohol and marijuana abuse fit the observed data well, with $\chi^2(1) = 10.59$, $p = .001$, CFI = .99, and IFI = 1.0. At the age 18 and 21 assessment periods, men with more symptoms of alcohol or marijuana abuse reported significantly higher levels of antisocial behavior than would be expected based on their individual trajectories alone (at age 18 $\hat{\beta} = .22$, $t = 2.93$, $p < .001$ and at age 21 $\hat{\beta} = .12$, $t = 2.58$, $p < .001$ for alcohol; at age 18 $\hat{\beta} = .23$, $t = 2.92$, $p < .001$ and at age 21 $\hat{\beta} = .18$, $t = 3.25$, $p < .001$ for marijuana). At the age 26 assessment, this effect of alcohol abuse was marginally significant ($\hat{\beta} = .25$, $t = 1.83$, $p = .07$), and this effect for marijuana abuse was nonsignificant ($\hat{\beta} = .10$, $t = 0.68$, $p > .10$). These results suggest that, during the periods when these young men experienced more symptoms of substance abuse, they did not decline in their antisocial behavior to the extent that we would expect based on their antisocial behavior throughout young adulthood. Rather, substance abuse appeared to ensnare these young men within elevated patterns of antisocial behavior. This effect became weaker as men aged through this period of crime desistance.

To examine whether the snaring effects of substance abuse persisted over the subsequent measurement interval, we modified our LGC model to include (1) covariances (rather than structural pathways) between substance abuse indices and antisocial behavior within each measurement period that were constrained to be equal within time (e.g., the age 18 covariance between marijuana abuse and antisocial behavior was equated with the age 18 covariance between alcohol abuse and antisocial behavior) and (2) structural pathways from substance abuse at ages 18 and 21 predicting subsequent time-specific variations in antisocial behavior at ages 21 and 26, respectively. To identify this model, these paths were constrained to be equal within time (e.g., the path between age 18 marijuana abuse and age 21 antisocial behavior was equated with the path between age 18 alcohol abuse and age 21 antisocial behavior). The resulting model fit the data well, with $\chi^2(2) = 9.50$, $p = .01$, CFI = .99, and IFI = .99. All lagged predictions of time-specific deviations in antisocial behavior above and beyond the influences of the underlying trajectory process, and the covariances among substance abuse and antisocial behavior were nonsignificant ($\hat{\beta} = -.07$, $t = -1.86$ from ages 18 to 21; $\hat{\beta} = -.06$, $t = -0.95$). These results suggest that, as predicted by the snares hypothesis, substance abuse exerted a contemporaneous, rather than a lagged, effect on time-specific deviations away from individual trajectories of antisocial behavior.

Test of the general deviance hypothesis

A competing hypothesis is that substance abuse is not uniquely related to antisocial behavior, but instead antisocial behavior and substance abuse are interrelated over time due to a single underlying shared factor. Although we cannot directly observe such an underlying influence, we can estimate a model that would be consistent with this general deviance hypothesis. We estimated a second-order LGC in which alcohol abuse, marijuana abuse, and antisocial behavior each served as indicators on latent factors representing deviance proneness traits at ages 18, 21, and 26. Consistent with the work of Bushway et al. (2001) and Duncan, Duncan, Strycker, Li, and Alpert (1999), we then modeled growth in these three latent factors as a function of a higher-order intercept (with loadings set to 1.0) and slope (with loadings set to 0, 3, and 8) factor, reflecting individually varying trajectories of deviance proneness over time (see Figure 5.5).

We estimated this model in two steps. First, we tested just the longitudinal measurement model in which the three indicators of antisocial behavior, alcohol abuse, and marijuana abuse each loaded on a single underlying latent factor within each of the three time periods (Sayer & Cumsille, 2001). To identify this model, we set the factor loadings for the antisocial behavior indicator equal to 1.0 for each deviance proneness

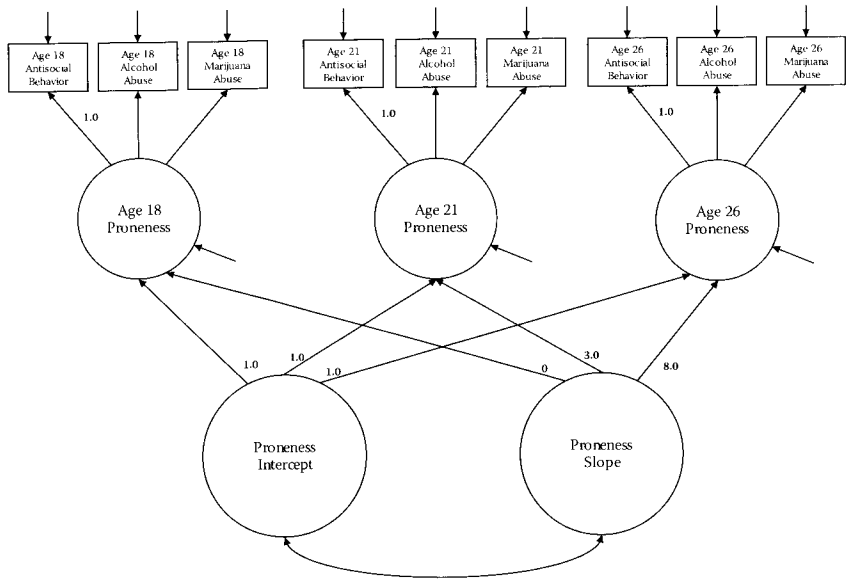

Figure 5.5 Second-order latent growth curve (LGC) testing general deviance hypothesis.

factor and fixed the intercept of this item to zero (Bollen & Curran, 2005). We also estimated means for each latent factor within time and freely correlated errors of measured variables across like indicators over time (e.g., errors for alcohol abuse at ages 18, 21, and 26 were correlated). The resulting model provided an adequate fit to the data, with $\chi^2(15) = 28.14$, $p = .02$, CFI = .99, and IFI = .99.

We extended this measurement model within the LGC framework to test whether individuals varied from one another in their trajectories of deviance proneness over time. As described by Bollen and Curran (2005), such second-order LGCs require that factor loadings within the measurement model be constrained to be equal over time for like constructs (e.g., factor loadings for marijuana abuse at ages 18, 21, and 26 were constrained to be equal). This constraint is necessary so that changes over time captured through the latent growth model are not confounded with changes over time in the contributions of individual indicators to the construct for which growth is modeled over time (see Sayer & Cumsille, 2001). After imposing these constraints, latent trajectory factors were initially estimated to reflect the intercept and slope of the trajectory underlying the three deviance proneness factors estimated within each assessment period. As before, errors of measured variables were correlated across like

indicators over time. The mean and variance of each trajectory factor were initially estimated. However, serious estimation problems indicated that the variance of the slope factor was near zero, suggesting no individual variability in the slope of general deviance over time. Following recommendations based on the work of Bryk and Raudenbush (1992), we reestimated the second-order LGC, fixing the variance of the slope factor to zero. However, the resulting model provided a poor fit to the data, with $\chi^2(22) = 98.06$, $p < .001$, CFI = .95, and IFI = .95.

We next explored whether poor fit in this final model was due either to the inability of the latent trajectory model to adequately capture change in the deviance proneness factors over time or to constraints posed on the measurement structure (which were necessary to estimate the second-order LGC but also consistent with the general deviance model's hypothesis that the relations between the indicators of deviant behavior and the underlying deviance proneness factor were constant over time). To do so, we returned to the longitudinal measurement model to test whether these constraints contributed to significant decrements in model fit. We estimated the same longitudinal measurement model as described above, but we also constrained the factor loadings of like constructs to be equal over time (as we did for the second-order LGC). The resulting model provided a similarly poor fit to the data, with $\chi^2(19) = 62.07$, $p < .001$, CFI = .97, and IFI = .97. A nested χ^2 test indicated that the addition of these constraints to the longitudinal measurement model produced significant decrements in model fit, with $\chi^2(3) = 33.93$ and $p < .001$. These results indicate that, although a general deviance factor is a tenable model within any given assessment period, the manifestations of this construct were variable over young adulthood. Given such changes in the nature of the construct over time, a model of growth in this construct over time is untenable. For this reason, we concluded that the general deviance hypothesis is not consistent with these data.

Conclusion

The current findings confirm a long-standing but largely untested assumption in developmental research on antisocial behavior; namely, that there are significant individual differences in intraindividual patterns of crime desistance during the transition from adolescence to adulthood. Although a gradual, linear decline in antisocial behavior typified the process of desistance for men in the Dunedin sample, these men differed significantly from one another both in the extent of antisocial behavior that they showed in late adolescence and in the rate at which their antisocial behavior declined as they entered adulthood. Moreover, alcohol and marijuana abuse each accounted for significant interindividual variability in

antisocial behavior over time through two mechanisms. Our analyses of the launch model showed that men with greater substance abuse at the end of adolescence showed greater antisocial behavior across young adulthood, although their trajectories showed greater decline than those of their peers. In essence, these men started young adulthood with a very high level of antisocial involvement; thus, they had further to fall as they desisted. Supporting the snares hypothesis, we also found that men who abused substances during young adulthood showed greater antisocial behavior than would be expected based on their estimated individual trajectories of antisocial behavior over time. In other words, periods in which men reported greater symptoms of substance abuse corresponded to elevated antisocial behavior with respect to that individual's pattern of antisocial behavior over time. Further, this conclusion was most strongly supported in our younger adult assessments. As such, substance abuse appears to exert both proximal and distal effects on desistance in antisocial behavior over young adulthood.

We offer this conclusion in the context of limitations in the current study. First, longitudinal studies of desistance suffer from the lack of information beyond the study window, leaving open to question whether those showing decelerated antisocial behavior will continue on a path toward cessation or later return to further antisocial behavior (Laub & Sampson, 2001). Second, we sampled eight behaviors from among those that index antisocial behavior during young adulthood. The extent to which substance abuse varies as an ensnaring factor across other types of antisocial behaviors is a question left for future study. Third, we focused on the ensnaring role of substance abuse among men. However, differences in the timing and, potentially, the predictors of crime desistance suggest that gender-specific hypotheses may need to be tested to more fully understand the normative process of desistance in women as well as men (Moffitt et al., 2001). Fourth, we studied only one cohort in one part of the world, and the findings require replication, although we have good reason to be optimistic because previous findings from the Dunedin study have been replicated in and generalized to other samples and developmental settings (e.g., Moffitt, Caspi, Silva, & Stouthamer-Loeber, 1995). Fifth, although the present study identified the snaring effects of both alcohol abuse and marijuana abuse, further research is needed to explore the mechanisms that mediate these effects.

As evidenced by the present findings, LGC modeling offers a powerful alternative to traditional methods that study change over time and that examine hypotheses about intraindividual development. Using these techniques, the current study offers significant insights into the developmental associations that may emerge over time between substance abuse and antisocial behavior. These hypotheses suggest a direction of causality

in which substance abuse serves to maintain engagement in antisocial behavior. Alternatively, the direction of effect may be reversed, reflecting self-selection in which the maintenance of antisocial behavior over time increases the likelihood of substance abuse. This possibility cannot be ruled out for our test of the launch hypothesis. However, although self-selection and the snares hypothesis may coexist (Moffitt, 1993), results from our time-varying covariate analyses offer evidence that self-selection does not account for the impact of snares as an impediment to crime desistance during young adulthood. Because predictions of antisocial behavior within time held above and beyond predictions based on the underlying trajectory of individual behavior, effects of substance abuse on antisocial behavior were residualized from the effect of continuity and developmentally normative change in antisocial behavior over time. Thus, previous antisocial behavior cannot account for these associations.

More broadly, LGC models offer a useful approach to the study of turning points. Their flexibility to incorporate different forms of influence on individually varying trajectories of behavior permits comparisons, as demonstrated here, across differing definitions of turning points. However, such flexibility also imposes a burden for theory development, which must rise to the challenge of specifying which of these points of inflection and forms of influence within the LGC best capture that proposed to underlie our turning points. Through continuing to push this exchange between developmental researchers and quantitative methodologists, we better approach a productive confluence of theory and methods.

Acknowledgment

We thank the Dunedin study members, Dunedin Unit Director Richie Poulton, unit research staff, and study founder Phil Silva. Research assistance was provided by HonaLee Harrington. The Dunedin Multidisciplinary Health and Development Research Unit is supported by the New Zealand Health Research Council. This research received support from Grants US-NIDA DA15398, US-NIDA DA13148; US-NIMH Grants MH45070 and MH49414; the William T. Grant Foundation; and Air New Zealand.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (text revision). Washington, DC: Author.

Bachman, J. G., Wadsworth, K. N., O'Malley, P. M., Schulenberg, J., & Johnston, L. D. (1997). Marriage, divorce and parenthood during the transition to young adulthood: Impacts on drug use and abuse. In J. Schulenberg, J. L.

Maggs, & K. Hurrelmann (Eds.), *Health risks and developmental transitions during adolescence* (pp. 246–282). New York: Cambridge University Press.

Bauer, D. J. (2003). Estimating multilevel linear models as structural equation models. *Journal of Educational and Behavioral Statistics, 28,* 134–167.

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

Blumstein, A., Cohen, J., & Farrington, D. P. (1988). Criminal career research: Its value for criminology. *Criminology, 26,* 1–35.

Bollen, K. A. (1989). A new incremental fit index for general structural equation models. *Sociological Methods and Research, 17,* 303–316.

Bollen, K. A., & Curran, P. J. (2005). *Latent trajectory models: A structural equation approach.* New York: Wiley.

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

Bushway, S., Piquero, A., Broidy, L., Cauffman, E., & Mazerolle, P. (2001). An empirical framework for studying desistance as a process. *Criminology, 39,* 491–515.

Carrig, M. M., Wirth, R. J., & Curran, P. J. (2004). A SAS macro for estimating and visualizing individual growth curves. *Structural Equation Modeling, 11,* 132–149.

Curran, P. J. (2003). Have multilevel models been structural equation models all along? *Multivariate Behavioral Research, 38,* 529–569.

Curran, P. J., Bauer, D. J., & Willoughby, M. T. (2004). Testing main effects and interactions in latent curve analysis. *Psychological Methods, 9,* 220–237.

Curran, P. J., & Bollen, K. A. (2001). The best of both worlds: Combining autoregressive and latent curve models. In L. M. Collins, & A. G. Sayer (Eds.), *New methods for the analysis of change* (pp. 105–136). Washington, DC: American Psychological Association.

Curran, P. J., & Hussong, A. M. (2002). Structural equation modeling of repeated measures data. In D. Moskowitz & S. Hershberger (Eds.), *Modeling intra-individual variability with repeated measures data: Methods and applications* (pp. 59–86). Mahwah, NJ: Erlbaum.

Curran, P. J., Muthén, B. O., & Harford, T. C. (1998). The influence of changes in marital status on developmental trajectories of alcohol use in young adults. *Journal of Studies on Alcohol, 59,* 647–658.

Donovan, J. E., & Jessor, R. (1985). Structure of problem behavior in adolescence and young adulthood. *Journal of Consulting and Clinical Psychology, 53,* 890–904.

Duncan, T. E., Duncan, S. C., Strycker, L. A., Li, F., & Alpert, A. (1999). *An introduction to latent variable growth curve modeling: Concepts, issues, and applications.* Mahwah, NJ: Erlbaum.

Elliott, D. S., Huizinga, D., & Menard, S. (1989). *Multiple problem youth: Delinquency, substance use, and mental health problems.* New York: Springer-Verlag.

Farrington, D. P. (1986). Age and crime. In M. Tonry and N. Morris (Eds.), *Crime and justice: An annual review of research* (Vol. 7, pp. 189–250). Chicago: University of Chicago Press.

Gottfredson, M., & Hirschi, T. (1990). *A general theory of crime.* Stanford: Stanford University Press.

Gottfredson, M., & Hirschi, T. (1995). Control theory and the life-course perspective. *Studies on Crime and Crime Prevention, 4,* 131–142.

Hanna, E. Z., & Grant, B. F. (1997). Gender differences in *DSM-IV* alcohol use disorders and major depression as distributed in the general population: Clinical implications. *Comprehensive Psychiatry, 38,* 202–212.

Horney, J., Osgood, D. W., & Marshall, I. H. (1995). Criminal careers in the short-term: Intra-individual variability in crime and its relation to local life circumstances. *American Sociological Review, 60,* 655–673.

Hussong, A. M., Curran, P. J., Moffit, T. E., Caspi, A., & Carrig, M. (2005). Substance abuse hinders desistance in young adults' antisocial behavior. *Development and Psychopathology, 16,* 1029–1046.

Kinderman, T. A., & Skinner, E. A. (1992). Modeling environmental development: Individual and contextual trajectories. In J. B. Asendorpf & J. Valsiner (Eds.), *Stability and change in development: A study of methodological reasoning* (pp. 155–190). Newbury Park, CA: Sage.

Jessor, R., & Jessor, S. L. (1977). *Problem behavior and psychosocial development: A longitudinal study of youth.* New York: Academic Press.

Laub, J. H., Nagin, D. S., & Sampson, R .J. (1998). Trajectories of change in criminal offending: Good marriages and the desistance process. *American Sociological Review, 63,* 225–238.

Laub, J. H., & Sampson, R. J. (2001). Understanding desistance from crime. In M. Tonry (Ed.), *Crime and justice: An annual review,* 28 (pp. 1–69), Chicago: University of Chicago Press.

Leonard, K. E., & Rothbard, J. C. (2000). Alcohol and the marriage effect. *Journal of Studies on Alcohol, Supplemental, 13,* 139–146.

MacCallum, R. C. (1990). The need for alternative measures of fit in covariance structure modeling. *Multivariate Behavioral Research, 25,* 157–162.

McArdle, J. J. (1988). Dynamic but structural equation modeling of repeated measures data. In J. R. Nesselroade & R. B. Cattell (Eds.), *Handbook of multivariate experimental psychology* (2nd ed.), (pp. 561–614), Plenum Press: New York.

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

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

Moffitt, T. E. (1993). Adolescence-limited and life-course persistent antisocial behavior: A developmental taxonomy. *Psychological Review, 100,* 674–701.

Moffitt, T. E., Caspi, A., Rutter, M., & Silva, P. A. (2001). *Sex differences in antisocial behaviour: Conduct disorder, delinquency, and violence in the Dunedin longitudinal study.* Cambridge, UK: Cambridge University Press.

Moffitt, T. E., Caspi, A., Silva, P. A., & Stouthamer-Loeber, M. (1995). Individual differences in personality and intelligence are linked to crime: Cross-context evidence from nations, neighborhoods, genders, races, and age-cohorts. In J. Hagan (Ed.), *Current perspectives on aging and the life cycle* (Vol. 4, pp. 1–34). Greenwich, CT: JAI Press.

Moffitt, T. E., Silva, P. A., Lynam, D. R., & Henry, B. (1994). Self-reported delinquency at age 18: New Zealand's Dunedin Multidisciplinary Health and Development Study. In J. Junger-Tas & G. J. Terlouw (Eds.), *The International Self-report Delinquency Project* (pp. 354–369). Amsterdam: Kugler.

Munson, J. A., McMahon, R. J., & Spieker, S. J. (2001). Structure and variability in the developmental trajectory of children's externalizing problems: Impact of infant attachment, maternal depressive symptomatology, and child sex. *Development and Psychopathology, 13,* 277–296.

Muthén, L. K., & Muthén, B. O. (1998). Mplus: The comprehensive modeling program for applied researchers. User's Guide. Los Angeles: Muthén & Muthén.

Osgood, D. W., Johnston, L. D., O'Malley, P. M., & Bachman, J. G. (1988). The generality of deviance in late adolescence and early adulthood. *American Sociological Review, 53,* 81–93.

Osgood, D. W., & Rowe, D. C. (1994). Bridging criminal careers theory and policy through latent variable models of individual offending. *Criminology, 32,* 517–554.

Piquero, A., Blumstein, A., Brame, R., Haapanen, R., Mulvey, E. P., & Nagin, D. S. (2001). Assessing the impact of exposure time and incapacitation on longitudinal trajectories of criminal offending. *Journal of Adolescent Research, 16,* 54–74.

Piquero, A., Brame, R., Mazerolle, P., & Haapanen, R. (2001). *Crime in emerging adulthood.* Unpublished manuscript.

Quinton, D., Pickles, A., Maughan, B., & Rutter, M. (1993). Partners, peers, and pathways: Assortative pairing and continuities in conduct disorder. *Development and Psychopathology, 5,* 763–783.

Robins, L. N. (1978). Sturdy predictors of adult antisocial behaviour: Replications from longitudinal studies. *Psychological Medicine, 8,* 611–622.

Robins, L. N., Helzer, J. E., Cottler, L., & Goldring, E. (1989). *Diagnostic Interview Schedule, Version III-R.* St. Louis, MO: Washington University.

Robins, L. N., & Reiger, D. A. (Eds.) (1991). *Psychiatric disorders in America: The epidemiologic catchment area study.* New York: The Free Press.

Rutter, M. (1987). Psychosocial resilience and protective mechanisms. *American Journal of Orthopsychiatry, 57,* 316–331.

Rutter, M. (1996). Transitions and turning points in developmental psychopathology: As applied to the age span between childhood and mid-adulthood. *International Journal of Behavioral Development, 19,* 603–626.

Sayer, A. G., & Cumsille, P. (2001). Second-order latent growth models. In L. Collins & A. Sayer (Eds.), *New methods for the analysis of change* (pp. 179–200). Washington, DC: APA Books.

Shadish, W. R., Cook, T. D., & Campbell, D. T. (2001). *Experimental and quasi-experimental designs for generalized causal inference.* Boston: Houghton Mifflin.

Sher, K. J., & Gotham, H. J. (1999). Pathological alcohol involvement: A developmental disorder of young adulthood. *Development and Psychopathology, 11,* 933–956.

Silva, P. A., & Stanton, W. R. (1996). *From child to adult: The Dunedin Multidisciplinary Health and Development Study.* Auckland, New Zealand: Oxford University Press.

Vaillant, G. E. (1995). *The natural history of alcoholism revisited.* Cambridge: Harvard University Press.

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