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# Modeling Strategies in Developmental Psychopathology Research: Prediction of Individual Change

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Developmental psychopathologists often seek to explain change over time in psychiatric syndromes and behavioral constructs. Because the rate and form of change may be unique to particular children, complex interactions among person-level characteristics, environmental characteristics, genetic/biological characteristics, and time are often hypothesized and investigated (e.g., Petersen et al., 2012). However, before we can assess change over time in such constructs and before we can investigate how change differs across children, we must consider how to conceptualize the psychiatric constructs themselves, and we must consider what assumptions are required for quantifying change. In order to address these issues, we first briefly discuss preliminary statistical and conceptual issues involving the categorical versus continuous representation of psychopathological constructs at a given time point. Second, we discuss some preconditions for quantifying change in such constructs across development. The third and fourth sections of this chapter focus on methods for describing and predicting longitudinal change in psychopathological constructs; these methods allow recovery of interactions between

person characteristics and time. We conclude with extension topics relevant to the longitudinal modeling of psychopathology and some design and data considerations for such studies.

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## Conceptualizing Psychiatric Syndromes as Categorical or Continuous

Symptoms such as anhedonia, weight change, and depressed mood covary or co-occur in the population at large. At certain severities, frequencies, and durations, the joint presence of these symptoms, along with several others, is conventionally considered to define an (unobserved) depression syndrome in the Diagnostic and Statistical Manual of Mental Disorders V (American Psychiatric Association, 2013). More generally, a psychiatric syndrome may be conceptualized as a dimensional or a categorical underlying construct. Dimensional models of psychopathology posit that associations among such depression symptoms occur because they mutually depend on the same underlying dimensional syndrome (i.e., a depressogenic liability distribution). Categorical models of psychopathology posit that there are homogeneous groups with unique symptom profiles and furthermore that observed associations among such depression symptoms arise due to the mixing together of groups with different mean profiles. For instance, one group might have a mean profile with high anhedonia and insomnia and moderate levels of

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other symptoms, whereas another group might have a mean profile with high depressed mood and concentration problems but moderate levels of other symptoms.

There have been attempts to discriminate statistically between categorical and continuous representations of psychiatric constructs (for reviews, see Helzer, van den Brink, & Guth, 2006; Kraemer, Shrout, & Rubio-Stipec, 2007; Krueger, Markon, Patrick, & Iacono, 2005; Widiger & Samuel, 2005). Recent approaches (e.g., Brown & Barlow, 2005; Conway, Hammen, & Brennan, 2012; Gillespie, Neale, Legrand, et al., 2011; Hallquist & Pilkonis, 2012; Lubke, Muthén, & Moilanen, et al., 2007; Muthén, 2006; Trull & Durrett, 2005; Walton, Ormel, & Krueger, 2011; Witkiewitz et al., 2013) involving analyses of symptom-level data have often involved comparing the fit of alternative statistical models that explain associations among symptoms using either latent dimensions—factor analysis models—or categories—mixture models such as latent class or latent profile models. Representations that combine both categories and continua have also been considered and have received attention in DSM-V (Regier, Kuhl, & Kupfer, 2013). Although there is no guarantee that the better fitting model corresponds to the true nature of psychiatric syndromes in the population (Bollen, 1989; Lubke et al., 2007), this assumption often seems to be employed. The ability to accurately discriminate between these categorical and continuous representations of psychopathology has been shown to depend on, for instance, sample size and the separation among classes, if classes exist (Lubke, 2012; Lubke & Neale, 2006, 2008). Historically, taxometric methods have also been used for discriminating classes from continua for psychiatric constructs (see Haslam, Holland, & Kuppens, 2012; Waller & Meehl, 1998), though these approaches have recently been shown to have key limitations compared to mixture models for this purpose (see Lubke & Tueller, 2010).

Ongoing interest in representing psychiatric constructs categorically often stems from the fact that ultimately categorical decisions will need to

be made regarding who will get treatment (cases) and who will not (Costello & Angold, 2006; Zachar, 2000). However, syndromes may still be treated as dimensional in statistical models even if ultimately categorical treatment decisions will be made. In fact, dimensional models of psychopathology can have advantages in terms of *statistical power* (i.e., the chance of detecting an effect when there is one) and in terms of prediction accuracy (e.g., Bergman, von Eye, & Magnusson, 2006; MacCallum, Zhang, Preacher, & Rucker, 2002; Markon, Chmielewski, & Miller, 2011). On the flip side, syndromes may still be treated as categorical in statistical models even if ultimately theory considers them as continuous constructs. One rationale for doing so is that assumptions imposed by dimensional models of psychopathology (e.g., that the underlying liability distribution for depression is normal) may be violated, and categorical representations of psychopathology can avoid restrictive distributional assumptions. However, preliminary empirical examinations of such latent syndrome liability distributions (van den Oord, Pickles, & Waldman, 2003) have not evidenced profound nonnormality to date (see also Schmitt, Mehta, Aggen, Kubarych, & Neale, 2006; Sterba, Baldasaro, & Bauer, 2012).

In sum, there may be, but does not need to be, an exact match between how the psychopathological construct is conceptualized theoretically (as a discrete or continuous syndrome) and how the psychopathological construct is treated in statistical models. In statistical models, it may be treated as categorical—a binary depression diagnosis variable or a nominal depression class membership variable—versus continuous—a continuous score on a depression factor or a sum of depression items, or a combination.

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### **Are We Measuring the Same Syndrome Construct Over Time?**

There were relatively few explicit developmental modifications of DSM-IV Axis I psychiatric syndromes for particular age groups (see

Costello & Angold, 2006, for review). This was a topic of discussion in the revisions for DSM-V (e.g., Pine et al., 2011; Rutter, 2011) resulting in several more modifications for DSM-V (see Regier et al. (2013) for a review.) Historically, there has been an assumption that psychiatric syndromes manifest similarly across developmental time, though they may differ in rate (e.g., tendency for higher levels of a disruptive behavior latent construct in toddlers, higher levels of an anxiety latent construct in middle childhood, and higher levels of a depression latent construct in adolescence). In fact, in order to assess *quantitative* longitudinal change in a behavior or syndrome (a topic considered in detail shortly), we must be able to make this assumption that we are measuring the same thing over time—i.e., that our construct displays *measurement invariance*. Specifically, in the context of psychiatric syndromes, this means each symptom should relate to the underlying latent syndrome in the same way, regardless of age.

The popular theoretical concept of *developmental pathways* of psychopathological behavior (Loeber, Keenan, & Zhang, 1997; Pickles & Hill, 2006) is not inconsistent with the existence of measurement invariance of psychiatric constructs. For instance, in one common example of such pathways, some children with oppositional defiant behavior in middle childhood desist by adolescence. However, among the children with persistent oppositionality, some develop conduct disorder problems in adolescence. This phenomenon is also called successive comorbidity (Angold, Costello, & Erkanli, 1999). So long as oppositional defiant symptoms consistently represent that syndrome over time and so long as conduct disorder symptoms consistently represent that syndrome over time, measurement invariance could still hold. An example of a theory that suggests violation of measurement invariance is that of Patterson (1993) who suggests that there is one underlying liability for antisocial behavior that he likens to a chimera; it manifests qualitatively differently over time depending on the cognitive level and developmental milestones of a given developmental

period. Whereas biting could be an indicator of the antisocial behavior construct in toddlerhood, it would not be an equally valid indicator in adolescence. Another theory that suggests violation of measurement invariance posits developmental differentiation of psychopathology (Knapp & Jensen, 2006; Lilienfeld, Waldman, & Israel, 1994) in which psychiatric syndromes are thought to be undifferentiated in early childhood. With advances in cognitive and emotional capacity, distinct syndromes like those described in the DSM are thought to be eventually capable of manifesting.

It is possible to statistically evaluate whether measurement invariance holds, presuming the availability of multivariate, longitudinal, symptom-level data. The particular statistical method for doing so will depend on whether psychopathology is being represented dimensionally (i.e., using syndrome factors) or categorically (using discrete classes with differing symptom patterns). Using the dimensional representation, measurement invariance can be evaluated using a longitudinal factor analysis framework (e.g., Tisak & Meredith, 1990). A factor analysis model is specified at every time point, and increasingly restrictive constraints are tested regarding the stability of the relationship between symptom indicators and syndrome factors across time points. Instead of using the categorical representation, measurement invariance can be evaluated using a latent transition model framework (Collins & Wugalter, 1992). In this framework, a latent class model is specified at every time point, and classes at times  $t-1$  and  $t$  are related; increasingly restrictive constraints are tested regarding the stability of symptom endorsement probabilities within-class across-time (see Collins & Lanza, 2010 for an example).

One possible manifestation of measurement *noninvariance* in the form of developmental differentiation would be if the number of factors or number of classes representing a construct increased over time. In one illustrative analysis that used a dimensional representation of Axis I DSM-IV syndromes, the factor structure representing these syndromes in preschoolers (Sterba,

Egger & Angold, 2007; see also Strickland et al., 2011) remained largely similar in a separate sample across middle childhood to adolescence, with little evidence of developmental differentiation except with respect to generalized anxiety and depression in later adolescence (Sterba et al., 2010). If measurement invariance is partially supported (e.g., most but not all items retain the same relationship to their respective construct over time), longitudinal change in the construct can still be quantified so long as (a) some items display measurement invariance (called anchor items) and (b) a longitudinal model is chosen that explicitly allows for noninvariant symptom-to-syndrome relationships over time. Quantifying change with partially invariant constructs is discussed in Edwards and Wirth (2009), and costs of assuming full invariance when only partial invariance holds are described in Wirth (2008). New Bayesian methods for more flexibly imposing partial measurement invariance are described in Muthén and Asparouhov (2013). In the subsequent sections, we assume measurement invariance of psychological constructs and focus instead on alternative approaches for quantifying change.

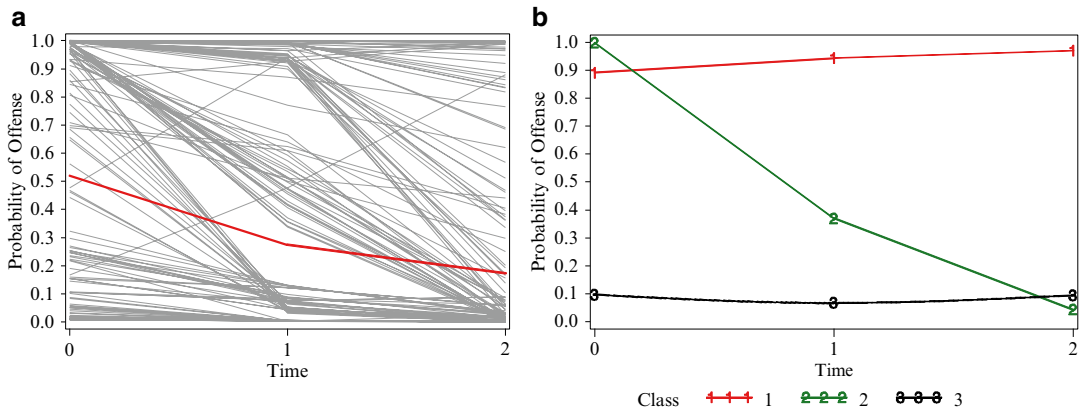
## Describing Growth in a Psychological Construct

A common objective of developmental psychopathology applications is describing and predicting growth in a target psychopathology construct over time (e.g., Curran & Willoughby, 2003; Dougherty, Klein, & Davila, 2004; Lenzenweger, Johnson, & Willett, 2004). Later we consider quantifying multivariate change in multiple constructs at once. For simplicity, suppose that we have an observed outcome repeatedly measured for  $N$  persons ( $i = 1 \dots N$ ) across  $t = 1 \dots T$  time points. Our observed repeated measure itself could be categorical or continuous. In the running example in this and the next section, our repeated measure is a binary physical aggression indicator recorded at  $T = 3$  time points spaced approximately one year apart.

This measure was collected from  $N = 428$  young adults who were recruited in 2002 at age 17–18<sup>1</sup> as they were transitioning out of Midwestern state-run or foster care facilities (Courtney & Cusick, 2007). This repeated measure will exhibit a particular mean trend over time, and its scores will be correlated over time. We can also expect that there will be heterogeneity around the sample mean trend in individual patterns of change over time—these individual patterns are often called individual *trajectories* of change.

Statistically, we have alternatives for modeling this heterogeneity. As two examples, we could account for this heterogeneity by assuming that individual trajectories vary continuously around a population mean trajectory, and then we could estimate a mean trend and continuous variability around this trend. This approach is often called random coefficient growth modeling (RCGM), hierarchical linear modeling, or latent curve modeling (Bollen & Curran, 2006; Singer & Willett, 2003). Figure 6.1 Panel (a) depicts a decreasing marginal mean trajectory (bold solid line) from a RCGM for our running example, superimposed upon a continuous distribution of individual trajectories implied by the model (thin grey lines). An alternative is to account for individual heterogeneity in change over time by assuming that it can be described by a finite number of prototype trajectories and that we can statistically select an optimal number of prototype trajectories. Children following the same prototype trajectory are considered members of their own latent *trajectory class*. Specifically, *within* a class, individuals are assumed to follow the same trend apart from random noise, although the functional form of the trend can differ *between* classes. This

<sup>1</sup>Exact ages for participants in this *Crime during the Transition to Adulthood* dataset, at [www.icpsr.umich.edu](http://www.icpsr.umich.edu), were not available to the public. A physically aggressive conduct offense was considered to have occurred if an adolescent over the past 12 months participated in a group fight, shot or stabbed someone, pulled a knife or gun, badly injured someone, or threatened someone with a weapon. Other representations of this aggression construct would be possible.



**Fig. 6.1** Unconditional RCGM versus LCGM aggression trajectories for the empirical example. (a) RCGM mean trajectory (*bold solid line*) & 150 model-implied

individual trajectories (*thin gray lines*) and (b) LCGM with 3 class trajectories. *Notes.* RCGM random coefficient growth model, LCGM latent class growth model

approach is often called latent class growth modeling (LCGM) or semiparametric group-based trajectory modeling (Muthén, 2001; Nagin, 1999); a related model not considered in detail here is called a growth mixture model (e.g., Muthén & Shedden, 1999). Figure 6.1 Panel (b) depicts the results of fitting a LCGM to the running example dataset. The best-fitting<sup>2</sup> 3-class solution is shown. These classes are seen to differ qualitatively in functional form (e.g., a high-chronic, low-stable, vs. decreasing shape). They also differ in probability of class membership (i.e., class proportions: 0.11 vs. 0.37 vs. 0.52, respectively).

Hundreds of applications of RCGMs and LCGMs (and closely related models) in the developmental psychopathology field have been published in the last decade alone (for reviews, see Nagin & Odgers, 2010; Sterba et al., 2012). Many of these applications have been in areas of substance abuse, delinquent behaviors, and internalizing behavior. Although there has been

some discussion of which model is best to apply under certain conditions (e.g., Maughan, 2005; Nagin & Tremblay, 2005b; Raudenbush, 2001, 2005; Sampson & Laub, 2005), this has remained unresolved because even when both models are fit to the same data, it is difficult to statistically tell if extracted LCGM trajectory classes truly exist or whether they are approximating an underlying continuous distribution of individual differences in change (Bauer & Curran, 2003a, 2003b).

Instead, there has been increasing interest in synthesizing LCGM and RCGM results across and within studies (e.g., Connell, Dishion, & Deater-Deckard, 2006; Hirsh-Pasek & Burchinal, 2006; Reinecke, 2006; Romens, Abramson, & Alloy, 2009). One obstacle to this synthesis has been the perception that a RCGM implies only one trajectory (the mean trend) and thus is not comparable to LCGM results that extract multiple class trajectories. Even efforts to synthesize LCGM results across studies have encountered obstacles. Many researchers expected that if classes literally correspond to population subgroups, the number of best-fitting class trajectories in LCGM should be replicable across studies using the same outcome

<sup>2</sup>The best-fitting number of classes was determined using Akaike's information criterion and the Lo-Mendell-Rubin adjusted likelihood ratio test.

(e.g., antisocial behavior). Such replicability has not been found (e.g., Fontaine, Carbonneau, Vitaro, Barker, & Tremblay, 2009; Horn, 2000; Nandi, Beard, & Galea, 2009; Skardhamar, 2010; van Dulmen, Goncy, Vest, & Flannery, 2009). For instance, in Fontaine et al.'s (2009) review of 21 applications of LCGM to girls' antisocial behavior, 5 % of studies had >5 classes, 29 % had 5 classes, 28 % had 4, 28 % had 3, and 10 % had 2. The proportions and shapes of these classes also differed widely [e.g., chronic (4 %), escalators (12 %), desistors (35 %), late onsetters (17 %), nonoffenders (32 %) vs. high rising (35 %), low (65 %) vs. high decreaseers (4 %), low decreaseers (15 %), near zero (81 %)]. Statistically, however, these findings are not surprising; the best-fitting number of LCGM trajectory classes extracted depends to some extent on  $N$  and  $T$ , just as the amount of continuous variability detectable in RCGM (e.g., continuous variation in just intercepts or also in linear and quadratic slopes of time) is known to depend on  $N$  and  $T$  (Fitzmaurice, Laird, & Ware, 2011; Hedeker & Gibbons, 2006). Other factors, such as measurement/distributional properties of the outcome and sampling characteristics, also affect the amount of heterogeneity that can be accounted for with either trajectory classes or continua (Bauer & Curran, 2003a; Eggleston, Laub, & Sampson, 2004; Jackson & Sher, 2008). Even if we could equate across-study characteristics when comparing LCGM applications within a given topic area (e.g., antisocial behavior), however, we still face the inability to integrate descriptive results across studies when LCGM is fit in one study and RCGM is fit in another study.

We can circumvent the latter impasse by shifting from focusing exclusively on description of individual change over time to focusing on the more concrete and arguably more clinically relevant objective of explaining and predicting individual patterns of change over time (Butler & Louis, 1992; Cudeck & Henly, 2003; Raudenbush, 2005; Sterba & Bauer, 2013). We will later see that considering prediction of change over time yields opportunities for comparing and synthesizing LCGM and RCGM results within and across developmental psychopathology studies.

## Predicting Growth in a Psychological Construct

Both RCGM and LCGM allow prediction of growth trajectories, using either time invariant covariates (TICs, e.g., gender, race, presence of birth trauma, presence of a particular gene) that are measured once or time-varying covariates (TVCs, e.g., whether an adolescent became homeless at time  $t$ , joined a gang at time  $t$ , or was pregnant at time  $t$ ) that are measured at multiple repeated time points. The effect (i.e., slope) of time may differ across values of a TIC, such as if rate of change in the antisocial behavior outcome is more positive for boys than girls. The effect of a TVC could also differ across time (e.g., if peer victimization at  $t = \text{age } 13$  had a larger effect on antisocial behavior than did peer victimization at  $t = \text{age } 18$ ). When the effect of a predictor differs across the levels of another predictor (here, for instance, time), this is statistically termed an interaction. Higher order interactions involve more than two variables. Nonlinear interactions imply that the effect of a predictor depends nonlinearly on the levels of another variable (see Aiken & West, 1991 for examples). It is also possible for TICs to interact with each other or to interact with particular TVCs, but our illustration here focuses on interactions involving time.

Recovery of potentially complex interactions involving person-level variables, environmental/contextual variables, and biological/neurological variables over time is central to many research traditions in the developmental psychopathology field, including the person-oriented research paradigm (Bergman & Magnusson, 1997; Cairns, Bergman, & Kagan, 1998; Muthén & Muthén, 2000; Sterba & Bauer, 2010a, 2010b; Von Eye & Bergman, 2003) and the holistic-interactional research paradigm (e.g., Gottlieb & Halpern, 2002; Magnusson, 1985). The latter paradigm, for instance, calls for investigating "how person factors and environmental factors—independently and jointly in interaction—operate and influence the course of development from childhood to adolescence" (Magnusson, 1985, p. 119). Put simply,

incorporating interaction relationships allows for conclusions to be made about change over time in a psychological construct with a greater degree of individual specificity. One could conclude that children with a particular constellation of characteristics may have differently shaped trajectories (with different rates of change over time in the outcome) than children with another constellation of characteristics.

Methods like LCGMs which classify children into classes or clusters are thought to have a distinct advantage for recovering complex potentially nonlinear interactions, compared to regression-based methods which do not extract classes, such as RCGM (e.g., Bergman, 2001; Bergman & Trost, 2006; Connell et al., 2006; Laursen & Hoff, 2006; Moffitt, 2006, 2008; Muthén, 2001, 2004; Nagin & Tremblay, 2005b; Segawa, Ngwe, Li, Flay, & Coinvestigators, 2005). The anticipated advantages of classification-based methods such as LCGMs may be based on the perspective that models like RCGMs can only accommodate linear predictive relationships (Hill, White, Chung, Hawkins, & Catalano, 2000; Shaw & Liang, 2012; Torppa, Poikkeus, Laakso, Eklund, & Lyytinen, 2006)—despite the fact that procedures exist for incorporating nonlinear and/or interactive predictor relationships in models such as RCGMs (Aiken & West, 1991; Curran, Bauer, & Willoughby, 2004). Anticipated advantages are also attributed to classification methods' greater flexibility in accounting for predictor relations (e.g., Laursen & Hoff, 2006; Pastor, Barron, Miller, & Davis, 2007).

However, Sterba and Bauer (2013) showed that, rather than one model being inherently superior at recovering such relationships, LCGMs and RCGMs accommodate interactions in different ways, and if specified appropriately both models can approximately equally well recover the same interactions—even higher-order nonlinear interactions. For instance, to accommodate interactions between TICs and time, RCGMs require explicitly including product terms (e.g.,  $TIC \times time$ ,  $TIC \times time^2$ ,  $TIC^2 \times time$ ) as predictors of the outcome. In contrast, LCGMs accommodate interactions between TICs and time by

including the TIC as a main effect predictor of class membership. The class trajectories, which differ in functional form of time, are then weighted by the probability of class membership—which is now conditional on the TIC. This specification intrinsically accommodates interactions between TIC and time. Thus, for recovering complex interactions involving TICs and time, these models require different things. RCGMs require entering higher-order product terms as covariates, whereas LCGMs require more classes, higher-order functional forms of time within class, and class-varying predictor effects (Sterba & Bauer, 2013). Yet for other kinds of interactions, both models require the same procedures. For instance, both models can account for an effect of a TVC that differs over time, by either including an explicit product term  $TVC \times time$  as a covariate or by specifying different slopes of the TVC at each time point.

We now use our running example on physical aggression to illustrate how our RCGM and our 3-class LCGM each account for similar patterns of change for adolescents with particular TIC and TVC characteristics. In other words, despite the fact that marginally the RCGM implies *one mean trajectory* and the LCGM implies *3 class-specific mean trajectories*, both models will be able to recover approximately the same predicted trajectories of change conditional on chosen person-level characteristics. For our example, TICs of interest are: presence of an alcohol or substance abuse diagnosis at time 1 ( $alc_i$ ), male gender ( $male_i$ ), level of social support ( $sup_i$ , a standardized scale score from Sherbourne and Stewart's [1991] inventory), and presence of a prior arrest record ( $arr_i$ ). TVCs of interest are whether an adolescent was in school at time  $t$  ( $sch_{it}$ ) and whether the adolescent was selling drugs at time  $t$  ( $sell_{it}$ ). An  $i$  subscript for a predictor denotes that it can have a unique value for every person, and an  $it$  subscript denotes that it can have a unique value for every person at every time point.

Though key results are shortly presented in graphical format, for interested readers, we briefly present the formulas for predicted trajectories—expected values of the outcome at each time point

given chosen values of the covariates. For the logic behind calculating predicted trajectories to convey conditional relationships over time, see Bauer and Shanahan (2007), Curran et al. (2004), Nagin and Tremblay (2005a), or Sterba and Bauer (2013). Importantly, although predicted trajectories are not often presented in LCGM applications, Nagin and Tremblay (2005a) recommend their use because “even if the groups [i.e., latent trajectory classes] are thought of as real entities, it

is not possible to assign individuals definitively to a specific trajectory ex ante based on number of risk factors. It is possible to construct only an expected trajectory” (p. 885).

Since our outcome is binary, our focus is on the predicted probability of physically aggressing at time  $t$  (i.e.,  $y_{it}=1$ ) given covariates, which we refer to as  $\hat{p}_{it}$ . For RCGM, we can calculate  $\hat{p}_{it}$  for person  $i$  at time  $t$  from the following equation for the log-odds:

$$\log(\hat{p}_{it} / (1 - \hat{p}_{it})) = \gamma_{00} + \gamma_{01}\text{alc}_i + \gamma_{02}\text{male}_i + \gamma_{03}\text{sup}_i + \gamma_{04}\text{arr}_i + (\gamma_{10} + \gamma_{11}\text{alc}_i + \gamma_{12}\text{male}_i + \gamma_{13}\text{sup}_i + \gamma_{14}\text{arr}_i)\text{time}_{it} + \gamma_{2r}\text{sell}_{it} + \gamma_{30}\text{sch}_{it} \quad (6.1)$$

$\gamma$ 's are estimated model coefficients. Note that the fact that time is multiplied by all quantities inside the parentheses implies interactions of time with each  $\text{alc}_i$ ,  $\text{male}_i$ ,  $\text{sup}_i$ , and  $\text{arr}_i$ . Finally, note that  $\text{sell}_{it}$  was allowed to interact semipara-

metrically with time because it has a different effect per time point ( $\gamma_{2r}$  for  $t=1-3$ ). For LCGM, we can calculate  $\hat{p}_{it}$  for person  $i$  at time  $t$  from the following equation for the log-odds:

$$\log(\hat{p}_{it} / (1 - \hat{p}_{it})) = \sum_{k=1}^K \pi_i^{(k)} \left( \beta_{00}^{(k)} + \beta_{10}^{(k)}\text{time}_{it} + \beta_{2r}^{(k)}\text{sell}_{it} + \beta_{30}^{(k)}\text{sch}_{it} \right) \quad (6.2)$$

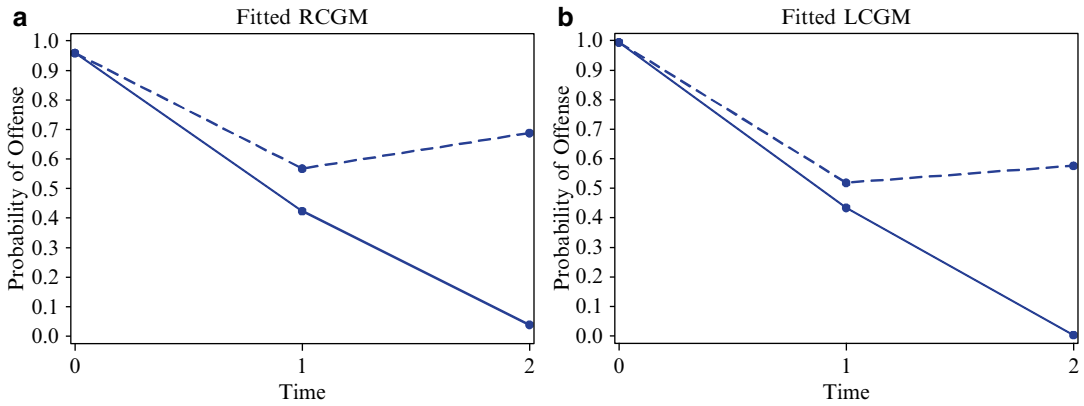
$$\pi_i^{(k)} = \frac{\exp\left(\delta_0^{(k)} + \delta_1^{(k)}\text{alc}_i + \delta_2^{(k)}\text{male}_i + \delta_3^{(k)}\text{sup}_i + \delta_4^{(k)}\text{arr}_i\right)}{\sum_{k=1}^K \exp\left(\delta_0^{(k)} + \delta_1^{(k)}\text{alc}_i + \delta_2^{(k)}\text{male}_i + \delta_3^{(k)}\text{sup}_i + \delta_4^{(k)}\text{arr}_i\right)} \quad (6.3)$$

$K$  is the number of classes (in our example, 3). A  $k$  superscript for a model coefficient implies that coefficient varies across latent classes  $k=1 \dots K$ . In Eq. (6.2),  $\beta$  s are estimated coefficients for time and for TVCs in the within-class trajectory. Note that  $\text{sell}_{it}$  is again allowed to interact with time as in Eq. (6.1) via a different effect per time point ( $\beta_{2r}^{(k)}$  for  $t=1 \dots 3$ ).  $\pi_i^{(k)}$  is person  $i$ 's probability of membership in class  $k$ . In Eq. (6.3), person  $i$ 's probability of class membership is shown to be predicted by the TICs using a multinomial logistic specification.  $\delta$  's are multinomial logistic coefficients and are fixed to 0 in the last class for identification.

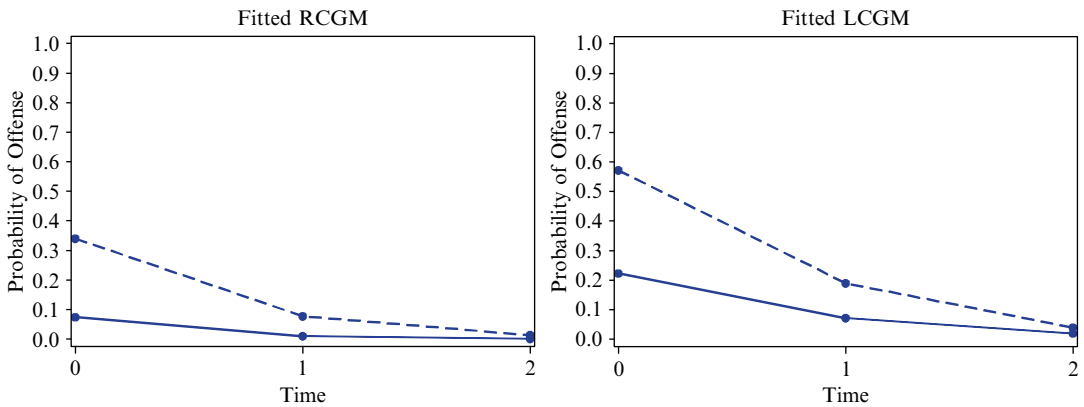
For illustrative purposes, we chose to plot predicted trajectories of physical aggression

propensity from each fitted model (RCGM and LCGM) at four chosen combinations of covariate values. Figure 6.2 depicts predicted trajectories for males with no baseline alcohol diagnosis, low social support, and a prior arrest record who quit school at time 2; these males either did (dashed line) or did not (solid line) begin to deal drugs. We can see that both the RCGM and LCGM predict that males with such multiple risk factors will likely start with high aggression at age 17–18 but rapidly decrease over time in their probability of physical aggression even if they quit school without a college degree. However, starting to deal drugs at approximately age 18–19 (time 2) stabilizes the probability of continued clinically meaningful aggression. Correspondingly, there





**Fig. 6.2** Predicted aggression trajectories for males with no baseline alcohol diagnosis, low social support, and a prior arrest record who drop out of school at time 2. At time 2 these males start dealing drugs (*dotted line*) versus do not start dealing drugs (*solid line*)



**Fig. 6.3** Predicted aggression trajectories for participants who have a baseline alcohol diagnosis but stay in school, do not deal drugs, have no arrest record, have high social support, and are female (*dotted line*) versus male (*solid line*)

was statistically significant evidence of a drug dealing by time interaction in both models. Figure 6.3 depicts predicted trajectories for both fitted models at a different combination of covariates: adolescents who have a baseline alcohol diagnosis, stay in school, do not deal drugs, have no arrest record, have high social support, and are either female (dashed line) or male (solid line). These adolescents have multiple contextual protective factors such as strong social support, though they do still have the risk factor of a prior substance abuse disorder. Nonetheless, particularly for females with these characteristics, we see

a relatively low probability of physical aggression over time; for males we see a moderate and decreasing propensity.

In sum, when we only talk about *describing* change over time with categorical versus continuous variation growth models (Fig. 6.1 Panels a vs. b), it is difficult to reconcile results across models. Nevertheless, when we move on to talk about *predicting* and explaining individual change over time using covariates, similar predictive patterns can emerge from both kinds of models given equivalently flexible specification of both. Still, flexible specifications of either

model can potentially run into practical problems recovering interactions of TICs or TVCs and time, particularly in small samples. For RCGMs, many product terms could induce estimation problems due to multicollinearity; for LCGMs, a sufficient number of classes to allow full variation of the predictor effect across time may not be estimable. Additionally, each kind of model presents unique conceptual challenges involving interpretation. Because TICs predict the entire trajectory as a whole in the LCGM (Eq. 6.3), we lack information about whether a TIC's effect entails a main effect or interaction with time. On the other hand, although the RCGM conveys whether particular main or interaction effects of predictors are statistically significant, the researcher is tasked with conceptually reintegrating this information to obtain a holistic understanding of predictive relations (Magnusson, 1998). For instance, from the running example LCGM, we learn that gender significantly differentiated class membership between each class 1 versus 3 and 2 versus 3, whereas from the RCGM we learn that there was a significant main effect of gender on intercepts, but not an interaction of gender with time.

Other interactions could have been investigated in our running example; for instance, if we posited that the amount by which predicted trajectories change across levels of social support differs by gender, RCGM would require inclusion of a three-way product term  $\text{male}_i \times \text{sup}_i \times \text{time}_i$  predictor, whereas LCGM would require inclusion of a two-way product term  $\text{male}_i \times \text{sup}_i$  predicting class membership, with its effect allowed to vary across class. This empirical dataset was limited in the kinds of nonlinear interactions with time that could be investigated due to the relatively small number of time points ( $T=3$ ); to see examples of recovery of higher-order nonlinear predictive relationships recovered with both RCGM and LCGM, see Sterba and Bauer (2013). Finally, note that predicted trajectories can be calculated, plotted, and compared using estimates from already-published RCGM and LCGM applications (regardless of the number of classes) so long as similar predictor sets were used. Doing so would facilitate refining of

theories about longitudinal predictor-outcome relationships, in the context of methodological pluralism.

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## Modeling Psychopathology Across Developmental Time: Extension Topics

The earlier sections “Describing Growth in a Psychological Construct” and “Predicting Growth in a Psychological Construct” of this chapter focused on methods for describing and predicting change in univariate models for one behavioral or psychiatric construct over time. Many extensions are possible, a few of which are highlighted here. Addressing questions about whether the course of one behavior or syndrome (e.g., depression) concurrently or sequentially affects the course of another behavior or syndrome (e.g., separation anxiety) requires multivariate longitudinal models (e.g., Farrell, Sullivan, Esposito, Meyer, & Valois, 2005). *Multivariate* extensions of LCGM models that relate class membership on multiple behaviors are reviewed in Nagin and Tremblay (2001) and Nagin (2005). Multivariate extensions of RCGMs that relate aspects of change on multiple behaviors are reviewed in MacCallum, Kim, Malarkey, and Kiecolt-Glaser (1997) and Duncan, Duncan, and Stryker (2006). If repeated measures on behavior A were collected before repeated measures on behavior B, these models can capture sequential relations among the behaviors' patterns of change. If repeated measures on behavior A were collected simultaneously with repeated measures on behavior B, these models capture parallel relations among each behavior's pattern(s) of change. Using such models, it may be of interest to examine whether the effects of TICs (say, treatment) on the slopes of one syndrome are *mediated* by the intercept (or slope) of the other syndrome (e.g., von Soest & Hagtvet, 2011). Also, in the case of *multiple-informant data* (e.g., parent, child, teacher report), it would be possible to specify a parallel process RCGM or LCGM, interrelating change in maternal report (process A), child report (process B), and teacher report (process C), for example (e.g., Kober,

Takacs, Urban, & Csepe, 2012; O'Brien & Fitzmaurice, 2005). Other options for modeling change in multiple-informant data include fitting one change trajectory to a superordinate latent construct that is itself defined by repeated measures from multiple informants (Hancock, Kuo, & Lawrence, 2001; Petersen et al., 2012).

Additionally, although prior sections have focused on the description and prediction of change, another common goal is to use aspects of change themselves to predict a *distal outcome*, such as whether at a follow-up assessment a hospitalization, suicide attempt, psychiatric diagnosis, college graduation, employment, or incarceration had occurred (e.g., Rudolph, Troop-Gordon, Hessel, & Schmidt, 2011). LCGMs and RCGMs can be extended to include distal outcomes which are often predicted, in the former case, by class membership and, in the latter case, by the continuously distributed aspects of change, i.e., intercepts and/or slopes of time (see, e.g., Bollen & Curran, 2006; Muthén, 2004). For instance, if two latent trajectory classes of markedly different initial levels and functional forms had equivalent rates of a psychiatric diagnosis distal outcome, this would be an example of *equifinality* (Cicchetti & Rogosch, 1996).

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## Design and Data Considerations for Longitudinal Modeling of Psychopathology

We have thus far focused on alternative model specifications that may be of use in answering particular research questions in developmental psychopathology. New design and data collection features can expand these modeling possibilities. For instance, developmental psychopathology research is enriched by increasingly multimodal data collection methodologies. Neuroimaging data and/or DNA sequencing data collected on existing longitudinal samples provides new predictors of psychopathology trajectories and new avenues for investigating gene-environment interactions (for methodological reviews, see Dodge & Rutter, 2011; Lindquist, 2008). Developmental psychopathologists also have increasing possibilities for individual-specific

number, spacing, and timing of data collection occasions using technology developed for intensive longitudinal designs, also called daily diary studies (see Walls & Schafer, 2008 for review; see also Mehta & West, 2000; Sterba, 2013).

Additionally, it is now more feasible for developmental psychopathologists to conduct secondary data analyses of large-scale, and often publicly available, *complex probability samples* involving clustering, stratification, and known but unequal probabilities of selection (e.g., the National Comorbidity Survey). The use of such probability samples has long been recommended by developmental epidemiologists (e.g., Costello & Angold, 2006), and recent statistical developments allow for their complex design features to be accommodated in popular statistical models (Muthén & Satorra, 1995; Sterba, 2009; Wu & Kwok, 2012). New statistical developments in the area of integrative data analysis (IDA), involving pooling more than one sample in a single analysis (Curran, 2009), can help to alleviate persistent problems involving underpowered studies in the field. See Bauer and Hussong (2009) for an IDA application in the area of internalizing behavior. Finally, recent advances in statistical estimation involving nonnormal and categorical data in latent variable modeling frameworks (Bandalos, 2013; Wirth & Edwards, 2007) provide new possibilities for the analysis of symptom-level data using more complex models than were feasible even 10 years ago.

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## Summary

The increasing availability of repeated measures data and rapidly advancing statistical modeling techniques suitable for addressing longitudinal research questions present exciting opportunities for developmental psychopathologists. We began by identifying background conceptual and statistical issues involving the representation of *individual differences in psychopathological constructs* as continuous or discrete (using multiple symptom indicators at a single time point). This topic has received increased attention in DSM-V with respect to representing not only individual syndromes but also relations among

them (as higher-order dimensions and/or categories; Regier et al. 2013). Then, in the second section we discussed preconditions necessary for studying quantitative change in such constructs. In the third section we discussed alternative models (namely, RCGMs and LCGMs) for describing and predicting change; these models posit that *individual differences in change* are continuous or discrete (using repeated measures of a single construct). It was illustrated in the fourth section that, even when LCGMs and RCGMs give fundamentally different results regarding the description of change, they can provide convergent results regarding the prediction of change—which is often of ultimate interest to developmental psychopathologists. As such, the fourth section described new opportunities for investigating substantive convergence of published findings on prediction of individual change across studies using very different statistical modeling strategies. Finally, we concluded with modeling extension topics as well as several data collection and design considerations particularly relevant to developmental psychopathologists. Developmental psychopathologists are encouraged to seek models suited to emerging research questions and designs—while at the same time remaining familiar with the assumptions, limitations, and interconnections among new and existing models.

## References

- Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Newbury Park, CA: Sage.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorder IV text revision*. Washington, DC: American Psychiatric Association.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Association.
- Angold, A., Costello, J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry*, *40*, 57–87.
- Bandalos, D. (2013). Performance of the ML, MLMV, WLSMV, and WLS estimators under model misspecification, nonnormality, and coarse categorization. *Structural Equation Modeling* (in press).
- Bauer, D. J., & Curran, P. J. (2003a). Distributional assumptions of growth mixture models: Implications for overextraction of latent trajectory classes. *Psychological Methods*, *8*, 338–363.
- Bauer, D. J., & Curran, P. J. (2003b). Over-extraction of latent trajectory classes: Much ado about nothing? Reply to Rindskopf (2003), Muthén (2003), and Cudeck and Henly (2003). *Psychological Methods*, *8*, 384–393.
- Bauer, D. J., & Hussong, A. M. (2009). Psychometric approaches for developing commensurate measures across independent studies: Traditional and new models. *Psychological Methods*, *14*, 101–125.
- Bauer, D. J., & Shanahan, M. J. (2007). Modeling complex interactions: Person-centered and variable-centered approaches. In T. D. Little, J. A. Bovaird, & N. A. Card (Eds.), *Modeling contextual effects in longitudinal studies* (pp. 255–284). Mahwah, NJ: Lawrence Erlbaum.
- Bergman, L. R. (2001). A person approach in research on adolescence: Some methodological challenges. *Journal of Research on Adolescence*, *16*, 28–53.
- Bergman, L. R., & Magnusson, D. (1997). A person-oriented approach in research on developmental psychopathology. *Developmental Psychopathology*, *9*, 291–319.
- Bergman, L. R., & Trost, K. (2006). The person-oriented versus variable-oriented approach: Are they complementary, opposites, or exploring different worlds? *Merrill-Palmer Quarterly*, *52*, 601–632.
- Bergman, L., von Eye, A., & Magnusson, D. (2006). Person-oriented research strategies in developmental psychopathology. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology* (2nd ed., Vol. 1, pp. 850–888). Hoboken, NJ: Wiley.
- Bollen, K. A. (1989). *Structural equations with latent variables*. New York: Wiley.
- Bollen, K. A., & Curran, P. J. (2006). *Latent curve models: A structural equation approach*. Hoboken, NJ: Wiley.
- Brown, T., & Barlow, D. (2005). Dimensional versus categorical classification of mental disorders in the fifth edition of the Diagnostic and statistical manual of mental disorders and beyond: Comment on the special section. *Journal of Abnormal Psychology*, *114*, 551–556.
- Butler, S. M., & Louis, T. A. (1992). Random effects models with non-parametric priors. *Statistics in Medicine*, *11*, 1981–2000.
- Cairns, R. B., Bergman, L. R., & Kagan, J. (1998). *Methods and models for studying the individual*. London: Sage.
- Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, *8*, 597–600.
- Collins, L., & Lanza, S. (2010). *Latent class and latent transition analysis*. Hoboken, NJ: Wiley.
- Collins, L., & Wugalter, S. (1992). Latent class models for stage-sequential dynamic latent variables. *Multivariate Behavioral Research*, *27*, 131–157.

- Connell, A. M., Dishion, T. J., & Deater-Deckard, K. (2006). Variable- and person-centered approaches to the analysis of early adolescent substance use: Linking peer, family, and intervention effects with developmental trajectories. *Merrill-Palmer Quarterly*, *52*, 421–448.
- Conway, C., Hammen, C., & Brennan, P. (2012). A comparison of latent class, latent trait, and factor mixture models of DSM-IV borderline personality disorder criteria in a community setting: Implications for DSM-V. *Journal of Personality Disorders*, *26*, 793–803.
- Costello, E., & Angold, A. (2006). Developmental epidemiology. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology* (2nd ed., Vol. 1, pp. 41–75). Hoboken, NJ: Wiley.
- Courtney, M., & Cusick, G. (2007). Crime during the transition to adulthood: How youth fare as they leave out-of-home care in Illinois, Iowa and Wisconsin. [Computer file].
- Cudeck, R., & Henly, S. (2003). A realistic perspective on pattern representation in growth data: Comment on Bauer and Curran (2003). *Psychological Methods*, *8*, 378–383.
- Curran, P. J. (2009). The seemingly quixotic pursuit of a cumulative psychological science: Introduction to the special issue. *Psychological Methods*, *14*, 77–80.
- Curran, P., Bauer, D., & Willoughby, M. (2004). Testing and probing main effects and interactions in latent curve analysis. *Psychological Methods*, *9*, 220–237.
- Curran, P., & Willoughby, M. (2003). Implications of latent trajectory models for the study of developmental psychopathology. *Development and Psychopathology*, *15*, 581–612.
- Dodge, K., & Rutter, M. (2011). *Gene-environmental interactions in developmental psychopathology*. New York, NY: Guilford Press.
- Dougherty, L., Klein, D., & Davila, J. (2004). A growth curve analysis of the course of dysthymic disorder: The effects of chronic stress and moderation by adverse parent-child relationships and family history. *Journal of Consulting and Clinical Psychology*, *72*, 1012–1021.
- Duncan, T., Duncan, S., & Stryker, L. (2006). *An introduction to latent variable growth curve modeling* (2nd ed.). Mahwah, NJ: Erlbaum.
- Edwards, M. C., & Wirth, R. J. (2009). Measurement and the study of change. *Research in Human Development*, *6*, 74–96.
- Eggleston, E. P., Laub, J. H., & Sampson, R. J. (2004). Methodological sensitivities to latent class analysis of long-term criminal trajectories. *Journal of Quantitative Criminology*, *20*, 1–26.
- Farrell, A., Sullivan, T., Esposito, L., Meyer, A., & Valois, R. (2005). A latent growth curve analysis of the structure of aggression, drug use, and delinquent behaviors and their interrelations over time in urban and rural adolescents. *Journal of Research on Adolescence*, *15*, 179–204.
- Fitzmaurice, G., Laird, N., & Ware, J. (2011). *Applied longitudinal analysis* (2nd ed.). Hoboken, NJ: Wiley.
- Fontaine, N., Carbonneau, R., Vitaro, F., Barker, E. D., & Tremblay, R. E. (2009). Research review: A critical review of studies on the developmental trajectories of antisocial behavior in females. *Journal of Child Psychology and Psychiatry*, *50*, 363–385.
- Gillespie, N., Neale, M., Legrand, L., Iacono, W., & McGue, M. (2011). Are the symptoms of cannabis use disorder best accounted for by dimensional, categorical, or factor mixture models? A comparison of male and female young adults. *Psychology of Addictive Behaviors*, *26*, 68–77.
- Gottlieb, G., & Halpern, C. T. (2002). A relational view of causality in normal and abnormal development. *Development and Psychopathology*, *14*, 421–435.
- Hallquist, M., & Pilkonis, P. (2012). Refining the phenotype of borderline personality disorder: Diagnostic criteria and beyond. *Personality disorders: Theory, research, and treatment*, *3*, 228–246.
- Hancock, G., Kuo, W.-L., & Lawrence, F. (2001). An illustration of second-order latent growth curve models. *Structural Equation Modeling*, *8*, 470–489.
- Haslam, N., Holland, E., & Kuppens, P. (2012). Categories versus dimensions in personality and psychopathology: A quantitative review of taxometric research. *Psychological Medicine*, *42*, 903–920.
- Hedeker, D., & Gibbons, R. D. (2006). *Longitudinal data analysis*. New York: Wiley.
- Hill, K., White, H., Chung, I.-J., Hawkins, J., & Catalano, R. (2000). Early adult outcomes of adolescent binge drinking: Person- and variable-centered analyses of binge drinking trajectories. *Alcoholism, Clinical and Experimental Research*, *24*, 892–901.
- Helzer, J., van den Brink, W., & Guth, S. (2006). Should there be both categorical and dimensional criteria for the substance use disorders in DSM-V? *Addiction*, *101*, 17–22.
- Hirsh-Pasek, K., & Burchinal, M. (2006). Mother and caregiver sensitivity over time: Predicting language and academic outcomes with variable- and person-centered approaches. *Merrill Palmer Quarterly*, *52*, 449–485.
- Horn, J. L. (2000). Comments on integrating person-centered and variable-centered research on problems associated with the use of alcohol. *Alcoholism, Clinical and Experimental Research*, *24*, 924–930.
- Jackson, K. M., & Sher, K. J. (2008). Comparison of longitudinal phenotypes based on alternate heavy drinking cut scores: A systematic comparison of trajectory approaches III. *Psychology of Addictive Behaviors*, *22*, 198–209.
- Knapp, P., & Jensen, P. S. (2006). Recommendations for DSM-V. In P. S. Jensen, P. Knapp, & D. Mrazek (Eds.), *Toward a new diagnostic system for child psychopathology: Moving beyond the DSM* (pp. 162–182). New York: Guilford Press.
- Kobor, A., Takacs, A., Urban, R., & Csepe, V. (2012). The latent classes of subclinical ADHD symptoms:

- Convergences of multiple informant reports. *Research in Developmental Disabilities*, 33, 1677–1689.
- Kraemer, H. C., Shrout, P. E., & Rubio-Stipec, M. (2007). Developing the diagnostic and statistical manual V: What will “statistical” mean in DSM-V? *Social Psychiatry and Psychiatric Epidemiology*, 42, 259–267.
- Krueger, R., Markon, K., Patrick, C., & Iacono, W. (2005). Externalizing psychopathology in adulthood: A dimensional-spectrum conceptualization and its implications for DSM-V. *Journal of Abnormal Psychology*, 114, 537–550.
- Laursen, B., & Hoff, E. (2006). Person-centered and variable-centered approaches to longitudinal data. *Merrill-Palmer Quarterly*, 52, 377–389.
- Lenzenweger, M., Johnson, M., & Willett, J. (2004). Individual growth curve analysis illuminates stability and change in personality disorder features. *Archives of General Psychiatry*, 61, 1015–1024.
- Lilienfeld, S., Waldman, I., & Israel, A. (1994). A critical examination of the use of the term and concept of comorbidity in psychopathology research. *Clinical Psychology: Science and Practice*, 1, 71–83.
- Lindquist, M. (2008). The statistical analysis of fMRI data. *Statistical Science*, 23, 439–464.
- Loeber, R., Keenan, K., & Zhang, Q. (1997). Boys’ experimentation and persistence in developmental pathways toward serious delinquency. *Journal of Child and Family Studies*, 6, 321–357.
- Lubke, G. (2012). Old issues in a new jacket: Power and validation in the context of mixture modeling. *Measurement*, 10, 212–216.
- Lubke, G., Muthén, B., Moilanen, I., McGovgh, J., Loo, S., Swanson, J. et al. (2007). Subtypes versus severity differences in attention-deficit/hyperactivity disorder in the Northern Finnish Birth Cohort. *American Academy of Child and Adolescent Psychiatry*, 46, 1584–1593.
- Lubke, G., & Neale, M. (2006). Distinguishing between latent classes and continuous factors: Resolution by maximum likelihood? *Multivariate Behavioral Research*, 41, 499–532.
- Lubke, G., & Neale, M. (2008). Distinguishing between latent classes and continuous factors with categorical outcomes: Class invariance of parameters of factor mixture models. *Multivariate Behavioral Research*, 43, 592–620.
- Lubke, G., & Tueller, S. (2010). Latent class detection and class assignment: A comparison of the MAXEIG taxometric procedure and factor mixture modeling approaches. *Structural Equation Modeling*, 17, 605–628.
- MacCallum, R. C., Kim, C., Malarkey, W. B., & Kiecolt-Glaser, J. K. (1997). Studying multivariate change using multilevel models and latent curve models. *Multivariate Behavioral Research*, 32, 215–253.
- MacCallum, R., Zhang, S., Preacher, K., & Rucker, D. (2002). On the practice of dichotomization of quantitative variables. *Psychological Methods*, 7, 19–40.
- Magnusson, D. (1985). Implications of an interactional paradigm for research on human development. *International Journal of Behavioral Development*, 8, 115–137.
- Magnusson, D. (1998). The logic and implications of a person-oriented approach. In R. Cairns, L. Bergman & J. Kagan (Eds.), *Methods and models for studying the individual*. London, Sage.
- Markon, K., Chmielewski, M., & Miller, C. (2011). The reliability and validity of discrete and continuous measures of psychopathology: A quantitative review. *Psychological Bulletin*, 137, 856–879.
- Maughan, B. (2005). Developmental trajectory modeling: A view from developmental psychopathology. *Annals of the American Academy of Political and Social Science*, 602, 118–130.
- Mehta, P. D., & West, S. G. (2000). Putting the individual back into individual growth curves. *Psychological Methods*, 5, 23–43.
- Moffitt, T. (2006). Life-course persistent versus adolescent-limited antisocial behavior. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology* (Vol. 3). New York: Wiley.
- Moffitt, T. E. (2008). A review of research on the taxonomy of life-course persistent versus adolescence-limited antisocial behavior. In F. T. Cullen, J. P. Wright, & K. R. Blevins (Eds.), *Taking stock—The status of criminological theory: Advances in criminological theory* (Vol. 15). New Brunswick, NJ: Transaction Publishers.
- Muthén, B. (2001). Latent variable mixture modeling. In G. A. Marcoulides & R. E. Schumacker (Eds.), *New developments and techniques in structural equation modeling* (pp. 1–33). Mahwah, NJ: Lawrence Erlbaum.
- Muthén, B. (2004). Latent variable analysis: Growth mixture modeling and related techniques for longitudinal data. In D. Kaplan (Ed.), *Handbook of quantitative methodology for the social sciences* (pp. 345–368). Newbury Park, CA: Sage.
- Muthén, B. O. (2006). Should substance use disorders be considered as categorical or dimensional? *Addiction*, 101, 6–16.
- Muthén, B., & Asparouhov, T. (2013). *BSEM measurement invariance analysis*. Retrieved from <http://www.statmodel.com>
- Muthén, B., & Muthén, L. (2000). Integrating person-centered and variable-centered analyses: Growth mixture modeling with latent trajectory classes. *Alcoholism, Clinical and Experimental Research*, 24, 882–891.
- Muthén, B. O., & Satorra, A. (1995). Complex sample data in structural equation modeling. *Sociological Methodology*, 25, 267–316.
- Muthén, B., & Shedden, K. (1999). Finite mixture modeling with mixture outcomes using the EM algorithm. *Biometrics*, 55, 463–469.
- Nagin, D. S. (1999). Analyzing developmental trajectories: A semi-parametric, group-based approach. *Psychological Methods*, 4, 139–157.
- Nagin, D. S. (2005). *Group-based modeling of development*. Cambridge: Harvard University Press.

- Nagin, D. S., & Odgers, C. (2010). Group-based trajectory modeling in clinical research. *Annual Reviews of Clinical Psychology, 6*, 109–138.
- Nagin, D. S., & Tremblay, R. E. (2001). Analyzing developmental trajectories of distinct but related behaviors: A group-based method. *Psychological Methods, 6*, 18–34.
- Nagin, D. S., & Tremblay, R. E. (2005a). Developmental trajectory groups: Fact or a useful statistical fiction? *Criminology, 43*, 873–904.
- Nagin, D. S., & Tremblay, R. E. (2005b). From seduction to passion: A response to Sampson and Laub. *Criminology, 43*, 915–918.
- Nandi, A., Beard, J., & Galea, S. (2009). Epidemiologic heterogeneity of common mood and anxiety disorders over the lifecourse in the general population: A systematic review. *BMC Psychiatry, 9*, 31.
- O'Brien, L., & Fitzmaurice, G. (2005). Regression models for the analysis of longitudinal Gaussian data from multiple sources. *Statistics in Medicine, 24*, 1725–1744.
- Pastor, D., Barron, K., Miller, B., & Davis, S. (2007). A latent profile analysis of college students' achievement goal orientation. *Contemporary Educational Psychology, 32*, 8–47.
- Patterson, G. (1993). Orderly change in a stable world: The antisocial trait as a chimera. *Journal of Consulting and Clinical Psychology, 61*, 911–919.
- Petersen, I., Bates, J., Goodnight, J., Dodge, K., Lansford, J., Pettit, G., et al. (2012). Interaction between serotonin transporter polymorphism (5-HTTLPR) and stressful life events in adolescents' trajectories of anxious/depressed symptoms. *Developmental Psychology, 48*, 1463–1475.
- Pickles, A., & Hill, J. (2006). Developmental pathways. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology* (2nd ed., Vol. 1, pp. 211–243). Hoboken, NJ: Wiley.
- Pine, D., Costello, J., Dahl, R., James, R., Leckman, J., Leibenluft, E., et al. (2011). Increasing the developmental focus in DSM-V: Broad issues and specific potential applications in anxiety. In D. Rogier, W. Narrow, E. Kuhl, & D. Kumpfer (Eds.), *The conceptual evolution of DSM-5* (pp. 305–321). Washington, DC: American Psychiatric Publishing.
- Raudenbush, S. W. (2001). Comparing-personal trajectories and drawing causal inferences from longitudinal data. *Annual Review of Psychology, 52*, 501–525.
- Raudenbush, S. W. (2005). How do we study “what happens next?”. *Annals of the American Academy of Political and Social Science, 602*, 131–144.
- Regier, D., Kuhl, E., & Kupfer, D. (2013). The DSM-5: Classification and criteria changes. *World Psychiatry, 12*, 92–98.
- Reinecke, J. (2006). Longitudinal analysis of adolescents' deviant and delinquent behavior: Applications of latent class growth curves and growth mixture models. *Methodology, 2*, 100–112.
- Romens, S., Abramson, L., & Alloy, L. B. (2009). High and low cognitive risk for depression: Stability from late adolescence to early adulthood. *Cognitive Therapy and Research, 33*, 480–498.
- Rudolph, K., Troop-Gordon, W., Hessel, E., & Schmidt, J. (2011). A latent growth curve analysis of early and increasing peer victimization as predictors of mental health across elementary school. *Journal of Clinical Child & Adolescent Psychology, 40*, 111–122.
- Rutter, M. (2011). Research review: Child psychiatric diagnosis and classification: Concepts, findings, challenges and potential. *Journal of Child Psychology and Psychiatry, 52*, 647–660.
- Sampson, R. J., & Laub, J. H. (2005). Seductions of method: Rejoinder to Nagin and Tremblay's “Developmental Trajectory Groups: Fact or Fiction?”. *Criminology, 43*, 905–913.
- Schmitt, J., Mehta, P., Aggen, S., Kubarych, T., & Neale, M. (2006). Semi-nonparametric methods for detecting latent non-normality: A fusion of latent trait and ordered latent class modeling. *Multivariate Behavioral Research, 47*, 427–443.
- Segawa, E., Ngwe, J. E., Li, Y., Flay, B., & Aban Aya Coinvestigators. (2005). Evaluation of the effects of the Aban Aya Youth Project in reducing violence among African American adolescent males using latent class growth mixture modeling techniques. *Evaluation Review, 29*, 128–148.
- Shaw, B., & Liang, J. (2012). Growth models with multi-level regression. In J. Newsom, R. Jones, & S. Hofer (Eds.), *Longitudinal data analysis: A practical guide for researchers in aging, health, and social sciences* (pp. 217–242). New York: Routledge.
- Sherbourne, C., & Stewart, A. (1991). The MOS social support survey. *Social Science & Medicine, 32*(6), 705–714.
- Singer, J., & Willett, J. (2003). *Applied longitudinal data analysis: Modeling change and event occurrence*. New York: Oxford University Press.
- Skardhamar, T. (2010). Distinguishing facts and artifacts in group-based modeling. *Criminology, 48*, 295–320.
- Sterba, S. K. (2009). Alternative model-based and design-based frameworks for inference from samples to populations: From polarization to integration. *Multivariate Behavioral Research, 44*, 711–740.
- Sterba, S. K. (2013). Fitting nonlinear latent growth models with individually-varying time points. *Structural Equation Modeling* (in press).
- Sterba, S. K., Baldasaro, R. E., & Bauer, D. J. (2012). Factors affecting the adequacy and preferability of semiparametric groups-based approximations of continuous growth trajectories. *Multivariate Behavioral Research, 40*, 590–634.
- Sterba, S. K., & Bauer, D. J. (2010a). Statistically evaluating person-oriented principles revisited: Reply to Molenaar (2010), von Eye (2010), Ialongo (2010) and Mun, Bates and Vaschillo (2010). *Development and Psychopathology, 22*, 287–294.
- Sterba, S. K., & Bauer, D. J. (2010b). Matching method with theory in person-oriented developmental psychopathology research. *Development and Psychopathology, 22*, 239–254.

- Sterba, S. K., & Bauer, D. J. (2013). Predictions of individual change recovered with latent class or random coefficient growth models. *Structural Equation Modeling* (in press).
- Sterba, S. K., Copeland, W., Egger, H., Costello, J., Erkanli, A., & Angold, A. (2010). Longitudinal dimensionality of adolescent psychopathology: Testing the differentiation hypothesis. *Journal of Child Psychology and Psychiatry*, *51*, 871–884.
- Sterba, S. K., Egger, H. L., & Angold, A. (2007). Diagnostic specificity and non-specificity in the dimensions of preschool psychopathology. *Journal of Child Psychology and Psychiatry*, *48*, 1005–1013.
- Strickland, J., Keller, J., Lavigne, J., Gouze, K., Hopkins, J., & LeBailly, S. (2011). The structure of psychopathology in a community sample of preschoolers. *Journal of Abnormal Child Psychology*, *39*, 601–610.
- Tisak, J., & Meredith, W. (1990). Longitudinal factor analysis. In A. von Eye (Ed.), *Statistical methods in longitudinal research* (Vol. 1, pp. 125–149). Boston, MA: Academic.
- Torppa, M., Poikkeus, A., Laakso, M., Eklund, K., & Lyytinen, H. (2006). Predicting delayed letter knowledge development and its relation to grade 1 reading achievement among children with and without familial risk for dyslexia. *Developmental Psychology*, *42*, 1128–1142.
- Trull, T., & Durrett, C. (2005). Categorical and dimensional models of personality disorder. *Annual Review of Clinical Psychology*, *1*, 355–380.
- van den Oord, E., Pickles, A., & Waldman, I. D. (2003). Normal variation and abnormality: An empirical study of the liability distributions underlying depression and delinquency. *Journal of Child Psychology and Psychiatry*, *44*, 180–192.
- van Dulmen, M., Goncy, E., Vest, A., Flannery, D. (2009). Group-based trajectory modeling of externalizing behavior problems from childhood through adulthood: Exploring discrepancies in the empirical findings. In J. Savage (Ed.), *The development of persistent criminality*. Oxford Scholarship Online Monographs
- von Eye, A., & Bergman, L. R. (2003). Research strategies in developmental psychopathology: Dimensional identity and the person-oriented approach. *Development and Psychopathology*, *15*, 553–580.
- von Soest, T., & Hagtvet, K. (2011). Mediation analysis in a latent growth curve modeling framework. *Structural Equation Modeling*, *18*, 289–314.
- Waller, N., & Meehl, P. (1998). *Multivariate taxometric procedures: Distinguishing types from continua*. Thousand Oaks, CA: Sage.
- Walls, T., & Schafer, J. (2008). *Models for intensive longitudinal data*. New York: Oxford University Press.
- Walton, K., Ormel, J., & Krueger, R. (2011). The dimensional nature of externalizing behaviors in adolescence: Evidence from a direct comparison of categorical, dimensional, and hybrid models. *Journal of Abnormal Child Psychology*, *39*, 553–561.
- Widiger, T., & Samuel, D. (2005). Diagnostic categories or dimensions? A question for the diagnostic and statistical manual of mental disorders—Fifth edition. *Journal of Abnormal Psychology*, *114*, 494–504.
- Wirth, R. J. (2008). *The effects of measurement non-invariance on parameter estimation in latent growth models*. Unpublished dissertation, University of North Carolina at Chapel Hill.
- Wirth, R. J., & Edwards, M. E. (2007). Item factor analysis: Current approaches and future directions. *Psychological Methods*, *12*, 58–79.
- Witkiewitz, K., King, K., McMahon, R., Wu, J., Luk, J., Bierman, K., et al. (2013). Evidence for a multidimensional latent structural model of externalizing disorders. *Journal of Abnormal Child Psychology*, *41*, 223–237.
- Wu, J., & Kwok, O. (2012). Using SEM to analyze complex survey data: A comparison between design-based single-level and model-based multilevel approaches. *Structural Equation Modeling*, *19*, 16–35.
- Zachar, P. (2000). Psychiatric disorders are not natural kinds. *Philosophy, Psychiatry, Psychology*, *7*, 167–182.