8 Structural Equation Modelling for Latent Curve Models

8.1 Introduction

Latent curve models (LCMs) are popular longitudinal methods in the analysis of individual differences in the patterns of change, which usually involves a random intercept and a random slope (they are grouped as a latent growth factor) with each pair forming a different trajectory over time. Techniques of LCMs have been developed via the incorporation of many features of structural equation models (SEMs), see Meredith and Tisak (1990), and Bollen and Curran (2006). For instance, the random intercept and slope are respectively considered as latent variables representing initial status and rate of change of the outcome variable. In this way, SEM techniques provide powerful tools for developing more useful LCMs for analyzing complex dynamic changes. In this chapter, we introduce the basic LCM and some of its extensions, for example LCM involving second-order latent variables (Jöreskog, 1970) with ordered categorical variables. Again, Bayesian methods that utilize data augmentation and MCMC methods are introduced to analyze the LCMs. Two real longitudinal studies are presented to illustrate the Bayesian methodologies. One is about the health-related quality of life of stroke survivors, and the other is related to a longitudinal study of cocaine use.

This chapter is organized as follows. Backgrounds of the aforementioned real studies are descried in Section 8.2. Some LCMs are described in Section 8.3, using the longitudinal study of cocaine use as an illustrative example. The Bayesian approach is presented in Section 8.4. Section 8.5 provides the Bayesian analyses of the two real studies. Finally, Section 8.6 introduces some other extensions of the basic LCM.

8.2 Background of the Real Studies

8.2.1 A Longitudinal Study of Quality of Life of Stroke Survivors

Stroke is a major health issue in the older population as it not only affects physical impairment but also leads to disability for activities of daily living (ADL), social non-participation (handicap), and depression. Such changes have a substantial influence on health-related quality of life (HRQOL) of stroke survivors. Analyses about the dynamic changes of HRQOL of stroke survivors and the associated factors of these changes have received much attention in the field. Ahlsiö *et al.* (1984) found that even though HRQOL was associated with greater disability, it failed to improve over time even when activities of daily living function increased. Kwok *et al.* (2006) found that the environment and social interaction domains of HRQOL decreased during the first year of stroke, and depression had a more generalized adverse effect on HRQOL than basic functional disabilities. Moreover, it was observed that ADL including instrumental ADL remained stable, while occupation and orientation domains of handicap, and depression deteriorated. In order to examine the relative importance of the abovementioned inter-related factors in determining HRQOL in the first year of stroke, we will apply a dynamic latent curve model to study how the changes in HRQOL relate to the changes in ADL, handicap, and depression. The results would help steer the development of appropriate interventions to promote HRQOL of stroke survivors.

Data are obtained from patients with acute stroke within 2 days of admission, and follow-ups at three, six, and twelve months post-stroke. Outcome measures obtained from questionnaires including the modified Barthel Index (MBI) score (Shah, Vanclay and Cooper, 1989), Geriatric Depression Scale (GDS) score (Chiu *et al.*, 1994), World Health Organization Quality of Life measure (abbreviated Hong Kong version) (WHO-

QOL BREF (HK)) scores, and the London Handicap Scale (LHS; Harwood *et al.*, 1994). Patients' ADL were assessed through the MBI, which is a good measurement instrument in terms of reliability and validity; and has been already applied to most studies of stroke as the measure of functional status. MBI has 10 items on activities of daily living. The total score of 20 indicates full independence in ADL; a higher score represents a higher level of independence. Post-stroke depression was measured using the GDS that has 15 items. Score of 8 or more indicates depression. The HRQOL data set comprises 24 items covering 4 domains: physical health (PHY), psychological health (PSY), social interaction (SO), and environment (EN). The stroke survivors are required to respond to the items on a 5-point Likert scale in which the categories range from "Not at all" to "An extreme amount" (scores ranging from 1-5). The domain scores are converted by normogram to 100% score. Finally, handicap is defined as the disadvantage brought on by impairment and disabilities experienced by an individual, taking into account the influence of physical and psychological effects of a disease, the physical and social environment, and the effects of health service provisions. The most applied generic measure of handicap in stroke survivors is the LHS. It consists of six questions measuring the levels of handicap (scores ranging from 1-6) in six dimensions including mobility, independence, occupation, social integration, orientation, and economic self-sufficiency. Higher score indicates greater handicap. The stroke survivors are requested to mark the response that best describes their situations. The total score of all six domains was used to indicate overall handicap level. Recognizing that MBI, LHS, and GDS could change over time, LCMs are used to assess how the changes of these variables influence the growth of each of the four HRQOL domains at three time points.

8.2.2 A Longitudinal Study about Cocaine Use

Cocaine use is a major social and health problem in many countries. An interesting topic is to study the dynamic influences of various latent variables on the dynamic change in cocaine use. In the literature on cocaine use, many studies have shown that psychiatric problems have substantial impact on cocaine use (Brown *et al.*, 1998; Hser *et al.*, 2006). Moreover, the treatment of a cocaine patient and family support are other important influence on cocaine use. For achieving better understanding of the aforementioned issues, the UCLA Center for Advancing Longitudinal Drug Abuse Research collected various measures from patients admitted in 1988-89 to the West Los Angeles Veterans Affairs Medical Center and met the DSM III-R criteria for cocaine dependence (Kasarabada *et al.*, 1999). These patients were assessed at baseline, one year after treatment, two years after treatment, and 12 years ($t = 1,\ 2,\ 3,\ 4$) after treatment in 2002. Measures at each time point include days of cocaine use per month (CC), times per month in formal treatment (TR), Beck Inventory (BI), depression (DEP), anxiety (AN), and family support (FS), where CC, TR, BI, DEP, and AN are continuous, while FS is ordered categorical with a 3-point scale. Since some patients were confirmed to be deceased, some declined to be interviewed, and some were out of country, there is a considerable amount of missing data. In order to efficiently analyze the data, it is necessary to take into account the following features in developing LCMs and associated statistical methods. (i) We first consider an LCM based on cocaine use measured at $t = 1,\ 2,\ 3,\ 4$ with a latent variable η_1 on initial status (intercept) of cocaine use, and a latent variable η_2 on rate of change (slope) of cocaine use. Another LCM is considered on the basis of TR measured at $t = 1,\ 2,\ 3,\ 4$ with latent variables ξ_{11} and ξ_{21} on the initial status of TR and the rate of change of TR, respectively. The effects of $\{\xi_{11}, \xi_{21}\}$ on $\{\eta_1, \eta_2\}$ can be assessed

through a simple structural equation. Similarly, we consider one more LCM based on FS measured at $t = 1,\ 2,\ 3,\ 4$, and obtain the initial status of FS (ξ_{12}) and rate of change of FS (ξ_{22}) via growth curve modeling. The effects of $\{\xi_{12}, \xi_{22}\}$ together with $\{\xi_{11}, \xi_{21}\}$ on $\{\eta_1, \eta_2\}$ are assessed by incorporating them in a structural equation. Based on the same rationale, we can first obtain latent variables (intercept, slope) based on the measures of DEP at each time point, and then assess the effects of these latent variables on $\{\eta_1, \eta_2\}$. However, with the inclusion of BI and AN, our interest is not only on DEP, but also on a latent variable 'psychiatric problem (PP)' that should be assessed by three observed variables BI, DEP, and AN. Hence, at each time point we first use a measurement equation to group BI, DEP, and AN into the latent variable 'PP', then construct an LCM on the basis of PP at $t = 1,\ 2,\ 3,\ 4$ for obtaining the initial status of PP (ξ_{13}) and rate of change of PP (ξ_{23}). Here, we call a latent variable obtained from several observed variables a "first-order latent variable" (for example, $\eta_1,\ \eta_2,\ \xi_{11},\ \xi_{21},\ \xi_{12},\ \xi_{22}$, PP); and call a latent variable obtained from several latent variables (for example, ξ_{13}, ξ_{23}) a "second-order latent variable" (see Jöreskog, 1970). Hence, it is desirable to establish a comprehensive LCM that accommodates the effects of both first- and second-order latent variables on the growth factors of the outcome variables. (ii) As pointed out by Li, Duncan and Acock (2000), it is important to include the interaction of latent variables in assessing the joint effect of dynamic latent variables. Hence, the LCM accommodates a nonlinear structural equation which subsumes polynomials (including interaction and higher order terms) of the first-order and second-order latent variables. (iii) The LCM accommodates mixed continuous and ordered categorical data, which are very common in practice. (iv) The LCM accommodates missing data, which are very common in longitudinal studies.

8.3 Latent Curve Models

8.3.1 Basic Latent Curve Models

The basic latent curve model (LCM) (see Meredith and Tisak, 1990; Bollen and Curran, 2006) can be viewed as the following common factor analysis model:

$$\mathbf{y} = \boldsymbol{\Lambda}\boldsymbol{\eta} + \boldsymbol{\epsilon}_y, \tag{8.1}$$

where $\mathbf{y}$ is a $T \times 1$ vector of repeated measures, $\boldsymbol{\Lambda}$ is a $T \times m$ parameter matrix of sequential fixed values of the growth curve records, $\boldsymbol{\eta}$ is an $m \times 1$ outcome latent growth factor, which contains scores on the m factors for a given individual, and $\boldsymbol{\epsilon}_y$ is a $T \times 1$ vector of residual errors. The pattern of $\boldsymbol{\Lambda}$ can be interpreted as representing a particular aspect of change in y across the T occasions. It is assumed that $\boldsymbol{\epsilon}_y$ is distributed as $N[\mathbf{0}, \boldsymbol{\Psi}_y]$, where $\boldsymbol{\Psi}_y$ is a diagonal matrix with diagonal elements ψ_{yt}, for $t = 1, \cdots, T$. When $m = 2$, Equation (8.1) is expressed in the following matrix form:

$$\begin{pmatrix} y_1 \\ y_2 \\ \vdots \\ y_T \end{pmatrix} = \begin{pmatrix} 1 & t_1 \\ 1 & t_2 \\ \vdots & \vdots \\ 1 & t_T \end{pmatrix} \begin{pmatrix} \eta_1 \\ \eta_2 \end{pmatrix} + \begin{pmatrix} \epsilon_{y1} \\ \epsilon_{y2} \\ \vdots \\ \epsilon_{yT} \end{pmatrix}. \tag{8.2}$$

The first column in $\boldsymbol{\Lambda}$ can be used to define an intercept factor, which represents an initial status of change in y. The second column in $\boldsymbol{\Lambda}$ represents the known times of measurement (t_1 through t_T) and constraints (the values of t should reflect the spacing between measurement occasions), and the latent growth factor $\boldsymbol{\eta}$ contains the initial status η_1 (intercept) and rate of change η_2 (slope). The two-factor linear latent curve model is specified in (8.2) so that the intercept factor (fixed at a constant value of 1) serves as the starting point (initial status) for any change (growth) across time and the slope

factor captures the rate of change of the trajectory over time. The scaling of the slope can be specified by using either fixed value restrictions (e.g. $0, 1, \cdots, T-1$) representing a straight line growth, or unspecified value restrictions (where $t_1 = 0$ and $t_2 = 1$ are fixed for model identification, and the remaining $t_3, \cdots, t_T$ are freely estimated) allowing estimation of an optimal pattern of change over measurement occasions (Meredith and Tisak, 1990).

The equation for the latent growth factor $\boldsymbol{\eta}$ can be simply written as:

$$\boldsymbol{\eta} = \boldsymbol{\beta} + \boldsymbol{\Pi}\boldsymbol{\eta} + \boldsymbol{\delta}, \tag{8.3}$$

where $\boldsymbol{\beta}$ is an $m \times 1$ vector of the population average of the latent individual growth factors, $\boldsymbol{\Pi}$ is an $m \times m$ matrix of coefficients expressing the structural relations between the $\boldsymbol{\eta}$ variables, and $\boldsymbol{\delta}$ is an $m \times 1$ vector of random residuals.

To illustrate the model framework, we use the example in the longitudinal study of cocaine use across four measurement occasions. Let $\mathbf{y} = (y_1, y_2, y_3, y_4)^T$, and $\boldsymbol{\Pi} = \mathbf{0}$, then Equations (8.2) and (8.3) can be expressed in the following form to represent the model in Figure 8.1:

$$\begin{pmatrix} y_1 \\ y_2 \\ y_3 \\ y_4 \end{pmatrix} = \begin{pmatrix} 1 & 0 \\ 1 & 1 \\ 1 & 2 \\ 1 & 12 \end{pmatrix} \begin{pmatrix} \eta_1 \\ \eta_2 \end{pmatrix} + \begin{pmatrix} \epsilon_{y1} \\ \epsilon_{y2} \\ \epsilon_{y3} \\ \epsilon_{y4} \end{pmatrix}, \tag{8.4}$$

$$\begin{pmatrix} \eta_1 \\ \eta_2 \end{pmatrix} = \begin{pmatrix} \beta_1 \\ \beta_2 \end{pmatrix} + \begin{pmatrix} \delta_1 \\ \delta_2 \end{pmatrix}. \tag{8.5}$$

The four measurement occasions are 1989 (baseline), 1990 (one year after treatment), 1991 (two years after treatment), and 2002 (12 years after treatment). Thus, the fixed

times of measurement $t_1,\ t_2,\ t_3$, and t_4 are set to be 0, 1, 2, and 12 to reflect the spacing between measurement occasions.

Figure 8.1 here

8.3.2 Latent Curve Models with Explanatory Latent Variables

The previous model can be extended to include the following explanatory latent growth factors:

$$\begin{pmatrix} \mathbf{x}_1 \\ \mathbf{x}_2 \\ \vdots \\ \mathbf{x}_r \end{pmatrix} = \begin{pmatrix} \mathbf{\Lambda} & \mathbf{0} & \cdots & \mathbf{0} \\ \mathbf{0} & \mathbf{\Lambda} & \cdots & \mathbf{0} \\ \vdots & \vdots & \ddots & \vdots \\ \mathbf{0} & \mathbf{0} & \cdots & \mathbf{\Lambda} \end{pmatrix} \begin{pmatrix} \boldsymbol{\xi}_1 \\ \boldsymbol{\xi}_2 \\ \vdots \\ \boldsymbol{\xi}_r \end{pmatrix} + \begin{pmatrix} \boldsymbol{\epsilon}_{x1} \\ \boldsymbol{\epsilon}_{x2} \\ \vdots \\ \boldsymbol{\epsilon}_{xr} \end{pmatrix}, \tag{8.6}$$

where $\mathbf{0}$ is a $T \times m$ matrix with elements 0. For $k = 1, \cdots, r$, $\boldsymbol{\xi}_k$ is the kth latent growth factor that includes growth parameters (e.g., intercept, slope), $\mathbf{x}_k$ and $\boldsymbol{\epsilon}_{xk}$ are similarly defined as $\mathbf{y}$ and $\boldsymbol{\epsilon}_y$, and $\mathbf{\Lambda}$ is similarly defined as in Equation (8.1). It is assumed that $\boldsymbol{\epsilon}_{xk}$ is distributed as $N[\mathbf{0}, \mathbf{\Psi}_{xk}]$, where $\mathbf{\Psi}_{xk}$ is a diagonal matrix with diagonal elements $\psi_{x1k}, \cdots, \psi_{xTk}$. The latent growth factors $\boldsymbol{\xi}_k$ $(k = 1, \cdots, r)$ are the first-order explanatory latent variables.

In the cocaine use data, the key outcome variable is 'cocaine use (CC)', and η_1 and η_2 are the latent growth parameters where η_1 (intercept) specifies the initial status of individual growth whereas η_2 (slope) specifies the rate of change over the four measurement time points. Let $\mathbf{x}_1 = (x_{11}, x_{21}, x_{31}, x_{41})^T$ be a vector of treatment measured at four time

occasions, and $\boldsymbol{\xi}_1 = (\xi_{11}, \xi_{21})^T$ such that

$$\begin{pmatrix} x_{11} \\ x_{21} \\ x_{31} \\ x_{41} \end{pmatrix} = \begin{pmatrix} 1 & 0 \\ 1 & 1 \\ 1 & 2 \\ 1 & 12 \end{pmatrix} \begin{pmatrix} \xi_{11} \\ \xi_{21} \end{pmatrix} + \begin{pmatrix} \epsilon_{x11} \\ \epsilon_{x21} \\ \epsilon_{x31} \\ \epsilon_{x41} \end{pmatrix}. \tag{8.7}$$

The growth factor in relation to treatment, $\boldsymbol{\xi}_1$, can be regarded as an explanatory latent growth factor with intercept (ξ_{11}) and slope (ξ_{21}). The LCM with this explanatory latent growth factor can be expressed by Equations (8.4) and (8.7), and the following equation:

$$\begin{pmatrix} \eta_1 \\ \eta_2 \end{pmatrix} = \begin{pmatrix} \beta_1 \\ \beta_2 \end{pmatrix} + \begin{pmatrix} \gamma_{11} & \gamma_{12} \\ \gamma_{21} & \gamma_{22} \end{pmatrix} \begin{pmatrix} \xi_{11} \\ \xi_{21} \end{pmatrix} + \begin{pmatrix} \delta_1 \\ \delta_2 \end{pmatrix}. \tag{8.8}$$

The path diagram of this LCM is depicted in Figure 8.2.

Figure 8.2 here

8.3.3 Latent Curve Models with Longitudinal Latent Variables

To incorporate longitudinal latent variables, we further consider the following measurement model (Song, Lee and Hser, 2009):

$$\mathbf{u}_t = \boldsymbol{\Lambda}_{ut}\boldsymbol{\omega}_{ut} + \boldsymbol{\epsilon}_{ut}, \quad t = 1, \cdots, T, \tag{8.9}$$

where $\mathbf{u}_t$ is a $p \times 1$ vector of measurements, $\boldsymbol{\Lambda}_{ut}$ is a $p \times q$ loading matrix, $\boldsymbol{\omega}_{ut}$ is a $q \times 1$ vector of latent variables, and $\boldsymbol{\epsilon}_{ut}$ is a $p \times 1$ vector of unique variances in $\mathbf{u}_t$. It is assumed that $\boldsymbol{\epsilon}_{ut}$ follows the distribution $N[\mathbf{0}, \boldsymbol{\Psi}_{ut}]$, where $\boldsymbol{\Psi}_{ut}$ is a diagonal matrix with diagonal

elements $\psi_{ut1}, \cdots, \psi_{utp}$. Equation (8.9) can be expressed in the following form:

$$\begin{pmatrix} u_{t1} \\ u_{t2} \\ \vdots \\ u_{tp} \end{pmatrix} = \begin{pmatrix} \lambda_{ut,11} & \lambda_{ut,12} & \cdots & \lambda_{ut,1q} \\ \lambda_{ut,21} & \lambda_{ut,22} & \cdots & \lambda_{ut,2q} \\ \vdots & \vdots & \ddots & \vdots \\ \lambda_{ut,p1} & \lambda_{ut,p2} & \cdots & \lambda_{ut,pq} \end{pmatrix} \begin{pmatrix} \omega_{ut1} \\ \omega_{ut2} \\ \vdots \\ \omega_{utq} \end{pmatrix} + \begin{pmatrix} \epsilon_{ut1} \\ \epsilon_{ut2} \\ \vdots \\ \epsilon_{utp} \end{pmatrix}, \tag{8.10}$$

where some $\lambda_{ut,ij}$ are fixed at 0 or 1 for model identification. As the latent variables ω_{utk}, $k = 1, \cdots, q$ defined in Equation (8.9) or (8.10) are related to observed variables measured at multiple time points $t_1, \cdots, t_T$, they are regarded as longitudinal latent variables. Let $\boldsymbol{\omega}_k = (\omega_{u1k}, \cdots, \omega_{uTk})^T$, and $\boldsymbol{\epsilon}_{\omega k} = (\epsilon_{\omega 1k}, \cdots, \epsilon_{\omega Tk})^T$, we can further use the following LCM to investigate the patterns of change in these longitudinal latent variables:

$$\boldsymbol{\omega}_k = \boldsymbol{\Lambda}\boldsymbol{\xi}_{r+k} + \boldsymbol{\epsilon}_{\omega k}, \quad k = 1, \cdots, q, \tag{8.11}$$

where $\boldsymbol{\xi}_{r+k}$ is an $m \times 1$ latent growth factor that includes growth parameters, r is specified in Equation (8.6), and $\boldsymbol{\Lambda}$ is similarly defined as in Equation (8.1). We assume that $\boldsymbol{\epsilon}_{\omega k}$ follows the distribution $N[\mathbf{0}, \boldsymbol{\Psi}_{\omega k}]$, where $\boldsymbol{\Psi}_{\omega k}$ is a diagonal matrix with diagonal elements $\psi_{\omega tk},\ t = 1, \cdots, T,\ k = 1, \cdots, q$. If $m = 2$, then $\boldsymbol{\xi}_{r+k} = (\xi_{1,r+k}, \xi_{2,r+k})^T$, and Equation (8.11) has the form:

$$\begin{pmatrix} \omega_{u1k} \\ \omega_{u2k} \\ \vdots \\ \omega_{uTk} \end{pmatrix} = \begin{pmatrix} 1 & t_1 \\ 1 & t_2 \\ \vdots & \vdots \\ 1 & t_T \end{pmatrix} \begin{pmatrix} \xi_{1,r+k} \\ \xi_{2,r+k} \end{pmatrix} + \begin{pmatrix} \epsilon_{\omega 1k} \\ \epsilon_{\omega 2k} \\ \vdots \\ \epsilon_{\omega Tk} \end{pmatrix}, \quad k = 1, \cdots, q, \tag{8.12}$$

where $\xi_{1,r+k}$ and $\xi_{2,r+k}$ indicate the intercept and slope in latent growth factor $\boldsymbol{\xi}_{r+k}$.

In the cocaine use example, 'psychiatric problem' is a longitudinal latent variable, which is formed by three observed variables, Beck Inventory (BI), Depression (DEP),

and Anxiety (AN), at multiple time points. The related measurement equation, which is a special case of Equation (8.10), is given by the following equation:

$$\begin{pmatrix} u_{t1} \\ u_{t2} \\ u_{t3} \end{pmatrix} = \begin{pmatrix} 1 \\ \lambda_{ut,21} \\ \lambda_{ut,31} \end{pmatrix} \omega_{ut1} + \begin{pmatrix} \epsilon_{ut1} \\ \epsilon_{ut2} \\ \epsilon_{ut3} \end{pmatrix}, \quad t = 1, \cdots, 4, \tag{8.13}$$

where 1's in $\mathbf{\Lambda}_{ut}$ are fixed to identify the factor analysis model. The LCM (see Equation (8.12)) that describes the pattern of change of the longitudinal latent variable ω_{ut1} is defined as:

$$\begin{pmatrix} \omega_{u11} \\ \omega_{u21} \\ \omega_{u31} \\ \omega_{u41} \end{pmatrix} = \begin{pmatrix} 1 & 0 \\ 1 & 1 \\ 1 & 2 \\ 1 & 12 \end{pmatrix} \begin{pmatrix} \xi_{13} \\ \xi_{23} \end{pmatrix} + \begin{pmatrix} \epsilon_{\omega 11} \\ \epsilon_{\omega 21} \\ \epsilon_{\omega 31} \\ \epsilon_{\omega 41} \end{pmatrix}, \tag{8.14}$$

where ξ_{13} and ξ_{23} can be regarded as the second-order latent variables. The path diagram in relation to Equations (8.13) and (8.14) is depicted in Figure 8.3.

Figure 8.3 here

In general, let $\boldsymbol{\xi} = (\boldsymbol{\xi}_1^T, \cdots, \boldsymbol{\xi}_r^T, \boldsymbol{\xi}_{r+1}^T, \cdots, \boldsymbol{\xi}_{r+q}^T)^T$ be the vector of explanatory latent growth factors that are either first- or second-order latent variables. To assess the linear and nonlinear effects of the explanatory latent growth factors in $\boldsymbol{\xi}$ on the latent growth factor $\boldsymbol{\eta}$, we further propose the following nonlinear structural equation:

$$\boldsymbol{\eta} = \boldsymbol{\beta} + \mathbf{\Pi}\boldsymbol{\eta} + \mathbf{\Gamma}\mathbf{F}(\boldsymbol{\xi}) + \boldsymbol{\delta}, \tag{8.15}$$

where $\mathbf{\Gamma}$ is a matrix of unknown regression coefficients, $\mathbf{F}(\boldsymbol{\xi}) = (f_1(\boldsymbol{\xi}), \cdots, f_h(\boldsymbol{\xi}))^T$ is a vector-valued function containing nonzero differentiable functions $f_1, \cdots, f_h$, and $h \geq r+q$, Here, $\boldsymbol{\xi}$ is distributed as $N[\mathbf{0}, \mathbf{\Phi}]$, $\boldsymbol{\delta}$ is a vector of residuals with distribution $N[\mathbf{0}, \mathbf{\Psi}_\delta]$,

in which $\mathbf{\Psi}_\delta$ is a diagonal matrix with diagonal elements $\psi_{\delta k},\ k = 1, \cdots, m$, and $\boldsymbol{\xi}$ and $\boldsymbol{\delta}$ are independent. Note that $\mathbf{\Phi}$ contains variances and covariances of explanatory latent growth factors which describe the relationships between the aspects of change represented by various growth curve parameters for different response variables. Let $\mathbf{\Lambda}_\delta = (\mathbf{\Pi}, \mathbf{\Gamma})$, $\boldsymbol{\zeta} = (\boldsymbol{\eta}^T, \boldsymbol{\xi}^T)^T$, and $\mathbf{G}(\boldsymbol{\zeta}) = (\boldsymbol{\eta}^T, \mathbf{F}(\boldsymbol{\xi})^T)^T$, then Equation (8.15) can be rewritten as

$$\boldsymbol{\eta} = \boldsymbol{\beta} + \mathbf{\Lambda}_\delta \mathbf{G}(\boldsymbol{\zeta}) + \boldsymbol{\delta}. \tag{8.16}$$

In the analysis of cocaine use data, we are interested in the longitudinal effects of treatment (TR), family support (FS), and psychiatric problem (PP) on cocaine use (CC), where 'psychiatric problem (PP)' is a latent variable. Specifically, we aim to study how the dynamic changes of the latent growth factors $\boldsymbol{\xi}_1$ (TR), $\boldsymbol{\xi}_2$ (FS), and $\boldsymbol{\xi}_3$ (PP) influence the latent growth factor $\boldsymbol{\eta}$ (CC). Here, $m = 2$, $r = 2$, $q = 1$, and each $\boldsymbol{\xi}_k = (\xi_{1k}, \xi_{2k})^T$ $(k = 1,\ 2,\ 3)$ includes the intercept and slope that represent the initial status and rate of changes in TR, FS, and PP, respectively. An interesting model is described as follows and depicted in Figure 8.4:

$$\eta_1 = \beta_1 + \gamma_{11}\xi_{11} + \gamma_{12}\xi_{12} + \gamma_{13}\xi_{13} + \gamma_{14}\xi_{11}\xi_{12} + \gamma_{15}\xi_{11}\xi_{13} + \gamma_{16}\xi_{12}\xi_{13} + \delta_1, \tag{8.17}$$

$$\eta_2 = \beta_2 + \gamma_{21}\xi_{21} + \gamma_{22}\xi_{22} + \gamma_{23}\xi_{23} + \gamma_{24}\xi_{21}\xi_{22} + \gamma_{25}\xi_{21}\xi_{23} + \gamma_{26}\xi_{22}\xi_{23} + \delta_2, \tag{8.18}$$

where the regression coefficients $\gamma_{11},\ \gamma_{12}$, and γ_{13} respectively represent the effect of initial status in treatment, family support, and psychiatric problem on the initial status in cocaine use, whereas $\gamma_{21},\ \gamma_{22}$, and γ_{23} respectively represent the effect of rate of change in treatment, family support, and psychiatric problem on the rate of change in cocaine use. The other coefficients represent various interactive effects of explanatory growth factors on the outcome growth factor. For example, Equation (8.18) can be used to examine the impact of joint changes in TR, FS, and PP influences the change in variable CC.

Hence, the significance of parameters γ_{24}, γ_{25}, and γ_{26} essentially answers the question: Does the simultaneous change (interaction effect) in explanatory latent growth factors in treatment, psychiatric problem, or family support influence the rate of change in the outcome growth factor in cocaine use?

Figure 8.4 here

So far we have focused our attention on continuous repeated measures. However, in the cocaine use study, some of the observed variables y, x, and u are ordered categorical variables. These ordered categorical variables will be treated similarly as before. Let z be the underlying continuous unobservable variable that corresponds to an ordered categorical variable x. Then, for $k = 1, \cdots, r$, $t = 1, \cdots, T$:

$$x_{tk} = j, \quad \text{if} \quad \alpha_{tk,j} \leq z_{tk} < \alpha_{tk,j+1}, \quad j = 0, \cdots, h_k, \tag{8.19}$$

where $\{-\infty = \alpha_{tk,0} < \alpha_{tk,1} < \cdots < \alpha_{tk,h_k} < \alpha_{tk,h_k+1} = \infty\}$ is the set of threshold parameters that defines the $h_k + 1$ categories. For identification, we follow the suggestion given in Bollen and Curran (2006) to set the first threshold $\alpha_{tk,1}$ to 0 and ψ_{xtk} to 1 for $k = 1, \cdots, r$, $t = 1, 2, \cdots, T$. The extension of more complex situations that all y, x, and u include ordered categorical components is straightforward.

In Section 8.5, we will apply a comprehensive LCM defined by Equations (8.1), (8.6), (8.9), (8.11), and (8.15) to the two sets of longitudinal data that are respectively related to the HRQOL of stroke survivors and the cocaine use study. Equation (8.19) will be incorporated if we encounter ordered categorical variables. Moreover, data that are missing at random can be treated using the method as described in Chapter 5.

8.4 Bayesian Analysis

To utilize the Bayesian approach to analyze the above LCM, let $\boldsymbol{\theta}$ be the vector that contains the unknown free parameters of the model, and $\boldsymbol{\alpha}$ be the vector that contains the unknown thresholds. In a Bayesian approach, $(\boldsymbol{\theta}, \boldsymbol{\alpha})$ is considered as random with a prior distribution $p(\boldsymbol{\theta}, \boldsymbol{\alpha})$. Let $\mathbf{D} = \{\mathbf{Y}, \mathbf{X}, \mathbf{U}\}$, where $\mathbf{Y}$, $\mathbf{X}$, and $\mathbf{U}$ include all three kinds of observed variables defined in Equations (8.2), (8.6), and (8.10), and $\mathbf{Z}$ includes all underlying continuous variables that correspond to ordered categorical variables in $\mathbf{D}$. The Bayesian inference is based on the posterior distribution of $(\boldsymbol{\theta}, \boldsymbol{\alpha})$ given the observed data set $\mathbf{D}$ and $p(\boldsymbol{\theta}, \boldsymbol{\alpha})$.

In the context of the introduced LCM, it is rather difficult to compute the posterior mean mainly because of the existence of a lot of first- and second-order latent variables, unobservable continuous variables, and the nonlinearity of the structural equation. To solve this difficulty, we adopt the commonly used strategy in Bayesian analysis that involves two key steps. The first step is to augment the observed data with all the latent variables to form a complete data set. Let $\boldsymbol{\Xi}$ include all latent growth factors ($\boldsymbol{\eta}$ and $\boldsymbol{\xi}$), $\boldsymbol{\Omega}$ include all longitudinal latent variables. The complete data set is $\{\mathbf{Z}, \boldsymbol{\Xi}, \boldsymbol{\Omega}, \mathbf{D}\}$. Treating $\mathbf{Z}$, $\boldsymbol{\Xi}$, and $\boldsymbol{\Omega}$ as random unknown quantities, the joint posterior density is $p(\boldsymbol{\theta}, \boldsymbol{\alpha}, \mathbf{Z}, \boldsymbol{\Xi}, \boldsymbol{\Omega}|\mathbf{D})$. The second step is to draw a sufficiently large sample of observations from the joint posterior distribution $[\boldsymbol{\theta}, \boldsymbol{\alpha}, \mathbf{Z}, \boldsymbol{\Xi}, \boldsymbol{\Omega}|\mathbf{D}]$, say $\{(\boldsymbol{\theta}^{(j)}, \boldsymbol{\alpha}^{(j)}, \mathbf{Z}^{(j)}, \boldsymbol{\Xi}^{(j)}, \boldsymbol{\Omega}^{(j)}),\ j = 1, \cdots, J\}$. The Bayesian estimate of $(\boldsymbol{\theta}, \boldsymbol{\alpha})$ can be obtained through the sample mean, and the standard error estimates can be obtained through the sample covariance matrix. Moreover, estimates of the various latent variables can be obtained from $\boldsymbol{\Xi}^{(j)}$ and $\boldsymbol{\Omega}^{(j)}$ for $j = 1, \cdots, J$, respectively. The task for simulating observations from the joint posterior distributions can be carried out with the well-known

Gibbs sampler (Geman and Geman, 1984), which iteratively simulates observations from $p(\boldsymbol{\theta}|\boldsymbol{\alpha}, \mathbf{Z}, \boldsymbol{\Xi}, \boldsymbol{\Omega}, \mathbf{D})$, $p(\boldsymbol{\alpha}, \mathbf{Z}|\boldsymbol{\theta}, \boldsymbol{\Xi}, \boldsymbol{\Omega}, \mathbf{D})$, $p(\boldsymbol{\Xi}|\boldsymbol{\theta}, \boldsymbol{\alpha}, \mathbf{Z}, \boldsymbol{\Omega}, \mathbf{D})$, and $p(\boldsymbol{\Omega}|\boldsymbol{\theta}, \boldsymbol{\alpha}, \mathbf{Z}, \boldsymbol{\Xi}, \mathbf{D})$.

Remind that the selection of the appropriate prior distribution for $(\boldsymbol{\theta}, \boldsymbol{\alpha})$ is an important issue in the Bayesian approach. As before, it is natural to assume that $p(\boldsymbol{\theta}, \boldsymbol{\alpha}) = p(\boldsymbol{\theta})p(\boldsymbol{\alpha})$. To deal with the situation with little or no information about threshold parameters, the following noninformative prior distribution is used for the prior distribution $p(\boldsymbol{\alpha})$:

$$p(\alpha_{tk,2}, \cdots, \alpha_{tk,h_k}) \propto C, \quad k = 1, \cdots, r, \quad t = 1, \cdots, T, \tag{8.20}$$

where C is a constant. Inspired by the most Bayesian analyses of latent variable models (see previous chapters, and Lee, 2007), we use conjugate prior distributions for $p(\boldsymbol{\theta})$. More specifically, the following conjugate prior distributions are considered:

$$\begin{aligned}
&\psi_{yt}^{-1} \stackrel{D}{=} Gamma[a_{0yt}, b_{0yt}], \quad t = 1, \cdots, T, \\
&\psi_{xtk}^{-1} \stackrel{D}{=} Gamma[a_{0xtk}, b_{0xtk}], \quad k = 1, \cdots, r, \quad t = 1, \cdots, T, \\
&\psi_{\omega tk}^{-1} \stackrel{D}{=} Gamma[a_{0\omega tk}, b_{0\omega tk}], \quad k = 1, \cdots, q, \quad t = 1, \cdots, T, \\
&\psi_{utk}^{-1} \stackrel{D}{=} Gamma[a_{0utk}, b_{0utk}], \quad k = 1, \cdots, p, \quad t = 1, \cdots, T, \\
&[\boldsymbol{\Lambda}_{utk}|\psi_{utk}] \stackrel{D}{=} N[\boldsymbol{\Lambda}_{0utk}, \psi_{utk}\boldsymbol{\Sigma}_{0utk}], \quad k = 1, \cdots, p, \quad t = 1, \cdots, T, \\
&\psi_{\delta k}^{-1} \stackrel{D}{=} Gamma[a_{0\delta k}, b_{0\delta k}], \quad [\boldsymbol{\Lambda}_{\delta k}|\psi_{\delta k}] \stackrel{D}{=} N[\boldsymbol{\Lambda}_{0\delta k}, \psi_{\delta k}\boldsymbol{\Sigma}_{0\delta k}], \quad k = 1, \cdots, m, \\
&\boldsymbol{\beta} \stackrel{D}{=} N[\boldsymbol{\beta}_0, \boldsymbol{\Sigma}_0], \quad \text{and} \quad \boldsymbol{\Phi}^{-1} \stackrel{D}{=} W_{m(r+q)}[\mathbf{R}_0, \rho_0],
\end{aligned} \tag{8.21}$$

where ψ_{yt}, ψ_{xtk}, and $\psi_{\omega tk}$ are the tth diagonal element of $\boldsymbol{\Psi}_y$, $\boldsymbol{\Psi}_{xk}$, and $\boldsymbol{\Psi}_{\omega k}$, and ψ_{utk} and $\psi_{\delta k}$ are the kth diagonal element of $\boldsymbol{\Psi}_{ut}$ and $\boldsymbol{\Psi}_{\delta}$, respectively; $\boldsymbol{\Lambda}_{utk}^T$ and $\boldsymbol{\Lambda}_{\delta k}^T$ are the kth row of $\boldsymbol{\Lambda}_{ut}$ and $\boldsymbol{\Lambda}_{\delta}$, respectively; and a_{0yt}, b_{0yt}, a_{0xtk}, b_{0xtk}, $a_{0\omega tk}$, $b_{0\omega tk}$, a_{0utk}, b_{0utk}, $a_{0\delta k}$, $b_{0\delta k}$, $\boldsymbol{\beta}_0$, $\boldsymbol{\Sigma}_0$, $\boldsymbol{\Lambda}_{0utk}$, $\boldsymbol{\Sigma}_{0utk}$, $\boldsymbol{\Lambda}_{0\delta k}$, $\boldsymbol{\Sigma}_{0\delta k}$, $\mathbf{R}_0$, and ρ_0 are the hyperparameters in the prior distributions. Under the above conjugate prior distributions, the full conditional

distributions required for implementing the Gibbs sampler are presented in Appendix 8.1.

Similarly as before, the convergence of the MCMC algorithm can be determined by examining the plots of a number (usually three) of different sequences of simulated observations with different initial values. At convergence, these sequences of observations should mix well. Suppose that the algorithm reaches convergence after the Kth iteration, a sufficient number of simulated observations, say J, after the Kth iteration are then collected to conduct statistical inference. Finally, Bayesian model comparison statistics, such as Bayes factor, DIC, or L_ν-measure, can be calculated using the simulated observations through the methods as described in previous chapters.

8.5 Applications to Two Longitudinal Studies

8.5.1 Longitudinal Study of Cocaine Use

The data set in this example is obtained from a longitudinal study about cocaine use conducted by the UCLA Center for Advancing Longitudinal Drug Abuse Research. Various measures were collected from 321 patients who were admitted in 1989 to the West Los Angeles Veterans Affairs Medical Center and who met the DSM III-R criteria for cocaine dependence (Kasarabada *et al.*, 1999). These patients were assessed at baseline, one year after treatment, two years after treatment, and 12 years after treatment in 2002 ($t_1 = 0$, $t_2 = 1$, $t_3 = 2$, $t_4 = 12$). Among these patients located at the 12-year follow-up (96.9%), some were confirmed to be deceased (8.7%), some declined to be interviewed, and some were either out of country or too ill to be interviewed after one year, two years, or at the 12-year after treatment. Consequently, there is a large amount of missing data in this data set. For brevity, the missing data are treated as missing at random (MAR). In this analysis, seven variables are involved. The first variable 'Days of cocaine use per month (CC)' associates with the cocaine use of the patients. The second variable

'Times per month in formal treatment (TR)' associates with frequencies that the patients participated in treatment. The third variable 'Family support (FS)' associates with the family support that the patients had, which is an ordered categorical variable with a 3-point scale. The following three variables 'Beck Inventory (BI)', 'Depression (DEP)', and 'Anxiety (AN)' associate with the mental health or psychiatric problem of the patients; the latter two are measured based on the Hopkins Symptom Check List (HSCL-58) (Hser *et al.*, 2006).

In this analysis, the outcome latent growth factor ($\boldsymbol{\eta}$) represents the pattern of change in CC. The first explanatory latent growth factor $\boldsymbol{\xi}_1$ represents the pattern of change in TR. The second explanatory latent growth factor $\boldsymbol{\xi}_2$ represents the pattern of change in FS. The variables BI, DEP, and AN with repeated measurements (at $t = 1,\ 2,\ 3,\ 4$) formed a longitudinal latent variable $\boldsymbol{\omega}_{ut}$, which can be interpreted as 'psychiatric problem (PP)'. Thus, the third explanatory latent growth factors $\boldsymbol{\xi}_3$ represents the patterns of change in PP. To study the possible effects of treatment, family support, and psychiatric problem of the patients on cocaine use, we propose the multivariate LCM as described in Figure 8.5. The interaction effects between the initial status or between the rate of changes are assessed through the structural equations given in (8.17) and (8.18). Among the observed variables, only FS is an ordered categorical variable with a 3-point scale, so we simply denote $\alpha_{tk,j}$ as $\alpha_{t,j}$, and we fix $\alpha_{t,1} = 0.0$ and $\psi_{xt2} = 1.0$ to identify the model.

Figure 8.5 here

The Bayesian estimates of the unknown parameters were obtained using WinBUGS. In specifying the hyperparameters in the prior distributions, we use the following hierarchical prior inputs: $\boldsymbol{\beta}_0 \sim N[\mathbf{0}, 4\mathbf{I}]$, $\boldsymbol{\Lambda}_{0utk} \sim N[\mathbf{0}, 4\mathbf{I}]$, and $\boldsymbol{\Lambda}_{0\delta k} \sim N[\mathbf{0}, 4\mathbf{I}]$, where $\mathbf{I}$ is an identity matrix with an appropriate dimension. The values of other hyperparameters are given

by $a_{0yt} = a_{0xtk} = a_{0\omega tk} = a_{0utk} = a_{0\delta k} = 10$, $b_{0yt} = b_{0xtk} = b_{0\omega tk} = b_{0utk} = b_{0\delta k} = 4$, $\rho_0 = 6$, and $\mathbf{R}_0 = \mathbf{I}$. With three different starting values, the WinBUGS outputs showed that the sequences produced by Gibbs sampler algorithm mixed well in less than 4,000 iterations. We took a burn-in-phase of 4,000 iterations, and further collected $J = 30,000$ observations to produce the Bayesian estimates, which are reported in Table 8.1. The corresponding WinBUGS code is presented in Appendix 8.2. The estimated structural equations of (8.17) and (8.18) are given as follows. The values in parentheses provide the standard error estimates of the associated parameters in the following equations and interpretations.

$$\eta_1 = \underset{(0.254)}{0.566} - \underset{(0.291)}{0.064}\xi_{11} + \underset{(0.362)}{0.383}\xi_{12} + \underset{(0.353)}{0.159}\xi_{13} - \underset{(0.293)}{0.123}\xi_{11}\xi_{12} + \underset{(0.254)}{0.016}\xi_{11}\xi_{13} - \underset{(0.173)}{0.014}\xi_{12}\xi_{13},$$

$$\eta_2 = \underset{(0.014)}{-0.060} + \underset{(0.135)}{0.006}\xi_{21} - \underset{(0.065)}{0.034}\xi_{22} + \underset{(0.219)}{0.517}\xi_{23} + \underset{(0.410)}{0.619}\xi_{21}\xi_{22} + \underset{(0.494)}{0.951}\xi_{21}\xi_{23} + \underset{(0.465)}{0.704}\xi_{22}\xi_{23}.$$

From Table 8.1 and the above equations, we have the following conclusions: (i) All estimates of $\lambda_{ut,i1},\ t = 1, \cdots, 4,\ i = 2,\ 3$ are large, indicating the strong associations between observed variables and the corresponding latent variables. (ii) In the above estimated structural equations, $\beta_1 = 0.566$ (0.254), $\beta_2 = -0.060$ (0.014), and $\gamma_{23} = 0.517$ (0.219) are statistically significant based on a p-value of 0.05. The value of γ_{23} represents the positive effect of rate of change in 'psychiatric problem' on the rate of change in 'cocaine use'. This positive effect shows that as a patient's rate of change in 'psychiatric problem' increases over time, his/her rate of change in cocaine use also increases. The following interesting phenomenon is also found in predicting the longitudinal change in cocaine use (η_2). Although the coefficients $\gamma_{21} = 0.006$ (0.135), $\gamma_{22} = -0.034$ (0.065) are small and insignificant, the coefficients of interaction terms, $\gamma_{24} = 0.619$ (0.410), $\gamma_{25} = 0.951$ (0.494), and $\gamma_{26} = 0.704$ (0.465), are relatively large. These marginally significant

coefficients indicate interactions between the dynamic latent growth factors (ξ_{21} and ξ_{22}, ξ_{21} and ξ_{23}, or ξ_{22} and ξ_{23}), which suggest joint effects of longitudinal changes in treatment, family support, and psychiatric problem on longitudinal change in cocaine use behavior. This finding shows that the integrated approach (combining formal medical treatment, increasing family support, and reducing psychiatric problem) is more efficient than simple medical treatment in reducing cocaine use of patients. (iii) The significant variances in both the intercept and the slope, $\psi_{\delta 1} = 0.340$ (0.060) and $\psi_{\delta 2} = 0.038$ (0.003), indicate substantial individual differences in both initial level and growth of cocaine use. Similarly, the significant variances $\phi_{11} = 0.669$ (0.077), $\phi_{33} = 1.430$ (0.293), and $\phi_{55} = 2.064$ (0.190) indicate substantial individual differences in initial level of treatment, family support, and psychiatric problem. Although variances $\phi_{22} = 0.015$ (0.001), $\phi_{44} = 0.053$ (0.014), and $\phi_{66} = 0.007$ (0.000) are small, they are also significant due to their small standard errors, indicating substantial individual differences in the rate of change in treatment, family support, and psychiatric problem. (iv) Most of the covariances in $\mathbf{\Phi}$ are small except for ϕ_{13}, ϕ_{15}, and ϕ_{35}. The large positive covariance $\phi_{13} = 0.528$ (0.109) indicates a strong positive correlation between the initial levels of treatment and family support. Similarly, $\phi_{15} = 0.708$ (0.091) indicates a strong positive correlation between the initial levels of treatment and psychiatric problem, and $\phi_{35} = 1.596$ (0.209) indicates a very strong positive correlation between the initial levels of family support and psychiatric problem. Interpretation about other less important parameters are not presented here to save space.

Table 8.1 here

To cross-validate the results, different prior inputs are used to conduct the Bayesian analysis. We found that the obtained results were very close to those given in Table 8.1.

This finding is consistent with the fact that the Bayesian estimates are not very sensitive to the prior inputs.

To provide more confidence in results given above, and to demonstrate the comprehensive LCM and statistical methodology, we presented a simulation study with a similar design as the longitudinal study of the cocaine use. The model is defined by Equations (8.4), (8.7), (8.13), (8.14), (8.17), and (8.18) and is depicted in Figure 8.5 except that the linear time trajectories are designated as $t_1 = 1,\ t_2 = 2,\ t_3 = 3,\ t_4 = 4$. The sample size is 500 and the population values of the parameters are set as follows: $\lambda_{ut,21} = \lambda_{ut,31} = 0.8,\ t = 1, \cdots, 4;\ \gamma_{11} = \gamma_{13} = \gamma_{22} = 0.6, \gamma_{12} = \gamma_{21} = \gamma_{23} = -0.6,$ $\gamma_{14} = \gamma_{16} = \gamma_{25} = -0.4, \gamma_{15} = \gamma_{24} = \gamma_{26} = 0.4;\ \beta_1 = 0.5,\ \beta_2 = -0.5;\ \psi_{yt} = \psi_{xt1} = \psi_{\omega t} = \psi_{utj} = 0.5,\ t = 1, \cdots, 4,\ j = 1,\ 2,\ 3;\ \psi_{\delta 1} = \psi_{\delta 2} = 0.36;\ \mathbf{\Phi} = (\phi_{kj})_{6\times 6}$ with $\phi_{kk} = 1.0$ and $\phi_{kj} = 0.3, k \neq j$. We assume that $\mathbf{x}_2$ in Equation (8.6) includes ordered categorical measurements at each time point with a 5-point scale. The population values of thresholds are $\alpha_{t,1} = 0.0,\ \alpha_{t,2} = 1.0,\ \alpha_{t,3} = 2.0$, and $\alpha_{t,4} = 3.0$, for $t = 1, \cdots, 4$. Here, $\alpha_{t,1}$ and ψ_{xt2} $(t = 1,\ 2,\ 3,\ 4)$ are respectively fixed at 0 and 1 to identify the auxiliary threshold model. To accelerate the convergence of the MCMC algorithm in the simulation, $\alpha_{t,4}$ $(t = 1,\ 2,\ 3,\ 4)$ are also fixed at their true values. About 30% of the data points are created to be missing, and they are treated as missing at random in this simulation.

The Bayesian estimates of the unknown parameters based on 100 replications were conducted using R2WinBUGS (Sturtz, Ligges and Gelman, 2005). First, a few tests performed using WinBUGS showed that the Gibbs sampler algorithm converged in less than 4,000 iterations, then $J = 6,000$ observations were collected after a burn-in-phase of 4,000 iterations to produce the Bayesian estimates in each replication. The mean (Mean) and the root mean squares (Rms) of the Bayesian estimates are reported in Table 8.2.

We see from this table that the mean of Bayesian estimates of the unknown parameters are close to the true values, and the root mean squares of the Bayesian estimates based on 100 replications are small, indicating that the Bayesian estimates are accurate.

Table 8.2 here

8.5.2 Health-related Quality of Life for Stroke Survivors

In order to better understand the relationships among HRQOL and (MBI, LHS, GDS), we used the LCM to analyze the related longitudinal data. Ideally, a comprehensive LCM that involves all the twenty-four items associated with HRQOL, MBI, LHS, and GDS should be considered at each of all the time points. At each time point, physical health (PHY), psychological health (PSY), social interaction (SO), and environment (EN) are identified as latent variables through the measurement equation of an SEM based on the appropriate items in the HRQOL data set. Then the dynamic interrelationships of these latent variables and (MBI, LHS, GDS) are assessed using the comprehensive LCM by considering all the time points together. However, this comprehensive LCM would be very tedious. For brevity, it may be desirable to obtain scores of the abovementioned latent variables first, then the dynamic interrelationships of each of those latent variables with (MBI, LHS, GDS) were analyzed with four separate LCMs, respectively. There are at least two methods to obtain such scores. One is to use a factor analysis model to estimate the factor scores; while the other is simply to take the averages of the corresponding items. In this illustrative example, we use the later method for simplicity. Therefore, based on three time points, four LCMs for {PHY, MBI, LHS, GDS}, {PSY, MBI, LHS, GDS}, {SO, MBI, LHS, GDS}, and {EN, MBI, LHS, GDS} are established, respectively. The results of the analysis for the four HRQOL domains were shown in Table 8.3. As an example,

the LCM for physical health domain was shown in Figure 8.6, and the corresponding estimated structural equations, predicted by MBI, LHS, and GDS were as follows:

$$\eta_1 = -0.001 + 0.174\xi_1 - 0.475\xi_3 - 0.310\xi_5,$$

$$\eta_2 = -0.013 + 0.280\xi_2 - 0.312\xi_4 - 0.328\xi_6,$$

where η_1 is the initial level of physical health domain, and η_2 is the rate of change in physical health domain. Therefore, initial physical health was significantly associated with ADL, handicap, and depression, while the rate of change in physical health was significantly and inversely associated with rate of change in GDS only.

The initial levels of the other three HRQOL domains were associated with depression only, except that psychological domain was marginally significantly associated with ADL as well. Similar to physical health domain, the rates of change in these three HRQOL domains were associated with the rate of change in depression only.

For the stroke survivors, activities of daily living, handicap, and depression are all important predictors of HRQOL. The relative importance of these interrelated variables in determining HRQOL was the subject of this study. The main finding of our analysis was that change in mood is the most significant effect on the HRQOL of stroke survivors, while changes in basic ADL and handicap had insignificant effects; see Pan *et al.* (2008) for more detailed interpretation of the results.

Table 8.3 and Figure 8.6 here

8.6 Other Latent Curve Models

In previous sections, we have introduced a comprehensive LCM that accommodates the effects of dynamic changes and their interactions of explanatory variables on the

dynamic changes of outcome variables. We have introduced second-order latent variables in LCMs to explore dynamic changes of longitudinal latent variables. As there has been an increasing application of LCMs in various fields, we hope that modeling interactions among the first- and second-order latent variables (growth factors) will have wide applications in the analysis of longitudinal studies in various disciplines. Besides the comprehensive LCM described above, the following extensions of the basic LCM are also useful in the substantive research.

8.6.1 Nonlinear Latent Curve Models

The basic LCM can be generalized to incorporate nonlinear trajectories and effects of covariates. With this extension, a higher order polynomial is used to describe a nonlinear pattern of dynamic change in individual characteristics. A longitudinal study about depression in multiple sclerosis (MS) (Beal *et al.*, 2007) is used here to illustrate the nonlinear LCMs. This study focused on three specific research issues: (1) To reveal the patterns of change in depressive symptoms over time. (2) To identify substantial effects of covariates such as age (Age), type of MS (TMS), years since diagnosis of MS (YMS), and functional limitation (FL) on the trajectory of depression over time. (3) To examine the correlations between characteristics of change in functional limitations and depressive symptoms over the seven-year time period. The data were collected from 607 persons with MS over a seven-year period, with initial recruitment in 1999, as part of an ongoing longitudinal study of quality of life (Stuifbergen *et al.*, 2006) in chronic illness. A nonlinear trajectory in depression for the sample is suggested by an examination of randomly selected empirical growth plots. A three factor LCM is used to model the

trajectories of depression for the MS sample:

$$\begin{pmatrix} y_1 \\ y_2 \\ \vdots \\ y_T \end{pmatrix} = \begin{pmatrix} 1 & t_1 & t_1^2 \\ 1 & t_2 & t_2^2 \\ \vdots & \vdots & \vdots \\ 1 & t_T & t_T^2 \end{pmatrix} \begin{pmatrix} \eta_1 \\ \eta_2 \\ \eta_3 \end{pmatrix} + \begin{pmatrix} \epsilon_{y1} \\ \epsilon_{y2} \\ \vdots \\ \epsilon_{yT} \end{pmatrix}, \tag{8.22}$$

where η_1, η_2, and η_3 represent individual specific random intercept, slope, and quadratic slope, respectively. As depicted in Figure 8.7, the change pattern and the correlations between the characteristics of change in depressive symptoms are analyzed. Furthermore, predictors in change of depression are examined by regressing the intercept, slope, and quadratic slope on the covariates of interest, which leads to the following equations:

$$\eta_1 = \beta_1 + \gamma_{11}(\text{Age}) + \gamma_{12}(\text{TMS}) + \gamma_{13}(\text{FL}) + \delta_1, \tag{8.23}$$

$$\eta_2 = \beta_2 + \gamma_{21}(\text{Age}) + \gamma_{22}(\text{TMS}) + \gamma_{23}(\text{FL}) + \delta_2, \tag{8.24}$$

$$\eta_3 = \beta_3 + \gamma_{31}(\text{Age}) + \gamma_{32}(\text{TMS}) + \gamma_{33}(\text{FL}) + \delta_3, \tag{8.25}$$

Their findings associated with the aforementioned three specific issues were obtained through the nonlinear LCM (Beal *et al.*, 2007). First, there is no significant increase or decrease trend in depressive symptoms although it fluctuates over time for individuals. Second, younger age, longer time since diagnosis of MS, progressive forms of MS, and greater extent of functional limitations will result in greater depressive symptoms at Time 1. Third, functional limitation shows an association with depression at all time periods, but other covariates do not. In addition, gender does not predict the changes in depressive symptoms. These results indicate that screening for depression in all persons with MS is necessary and important.

8.6.2 Multilevel Latent Curve Models

In some circumstances, clinical trials involve a multilevel design, leading to hierarchically longitudinal data. For example, to evaluate the effects of a neighborhood walking program on quality of life (QOL) among older adults, Fisher and Li (2004) used a multilevel sampling scheme to collect a sample of neighborhoods from a large metropolitan city, from which residents of older adults were randomly recruited. This two-level design results in a nested data structure in which participants are clustered within neighborhoods. The substantive interest of this study focuses on whether a 6-month neighborhood walking program improves neighborhood-level QOL for senior residents. A two-level LCM of QOL, with individual- and neighborhood-level data structures, is shown in Figure 8.8. All the involved measures are assessed at baseline, 3 months, and 6 months of the study period. Compared to the control neighborhoods, results from the two-level LCM indicate that Physical, Mental, and Satisfaction with Life aspects of QOL are significantly improved over the course of the 6-month intervention, which concludes that it is feasible and beneficial to implement a neighborhood-based walking program of low to moderate intensity in order to promote QOL among senior residents at a community level.

8.6.3 Mixture Latent Curve Models

Heterogeneity is commonly encountered in longitudinal analysis of practical applications. For heterogeneous data, there exist some latent classes under which the interested characteristics may present completely different change patterns. Mixture LCMs can be used to characterize the heterogeneity and to reveal specific change pattern for each distinctive latent class. Compared with the basic LCM, the additional tasks in applying mixture LCMs are to identify the number of latent classes, to detect the membership of each individual observation, and to predict the probability of each individual falling

in a specific class. In order to formulate the probability of individual i belonging to latent class k, the following multinomial logistic regression model is introduced (see also Chapter 7, Section 7.3). For $k = 1, \cdots, K$,

$$p(C_i = k|x_{i1}, \cdots, x_{ip}) = \frac{\exp(a_{0k} + a_{1k}x_{i1} + \cdots + a_{pk}x_{ip})}{\sum_{j=1}^{K} \exp(a_{0j} + a_{1j}x_{i1} + \cdots + a_{pj}x_{ip})}, \tag{8.26}$$

where K is the number of latent classes, C_i is the class membership for individual i, $x_{i1}, \cdots, x_{ip}$ are covariates that may potentially influence the chance of individual i belonging to latent class k, and $a_{0k}, a_{1k}, \cdots, a_{pk}$ are corresponding regression coefficients that reflect the importance of potential covariates.

Hser *et al.* (2008) applied this model to examine long-term trajectories of drug use for primary heroin, cocaine, and meth users. The data include 629 primary heroin users, 694 cocaine users, and 474 meth users. The main outcome measure is the number of days using the primary drug per month. As shown in Figure 8.9, the analysis of mixture LCMs reveals five distinctive groups with different drug use trajectories over ten-year follow-up: Consistently High Use, Increasing Use, Decreasing Use, Moderate Use, and Low Use. In addition, primary drug type is significantly associated with different trajectory patterns. Heroin users are most likely in the Consistently High Use group, cocaine and meth users are most likely in the Moderate Use group. The study also reveals that users in the High Use group have earlier onsets of drug use and crime, longer incarceration durations, and less employed periods than those in other groups. Compared with other existing studies of drug addiction, the use of mixture LCMs in this analysis emphasizes the heterogeneity of drug use patterns and the importance of understanding and addressing the full spectrum of drug use patterns over time.

Another application of mixture LCMs is the analysis of depression in the persons with myocardial infarction (MI). Elliott *et al.* (2006) analyzed affect and event data from

subjects post-MI in order to understand how mood and reactivity to negative events over time relate to diagnostic level depression. In this study, 35 patients who had experienced an MI within the past year and were in treatment currently are investigated. The affect scores and event indicators (indicating presence of positive, negative, and neutral events) of the patients are collected for up to 35 consecutive days. The analysis of mixture LCMs suggests a two-class model for the MI patients: an optimist class with stable positive affect and declining perceived negative events; and a pessimist class with declining positive affect and continuing perceived negative events. Depressed subjects have a 92% chance of belonging to the pessimist class, compared with 62% among non-depressed subjects. This finding uncovers some hitherto unobserved structure in the positive affect and negative event data in this sample. The key advantage of using mixture LCMs in this study is that persons who are most at risk of developing major or minor depression can be identified, which will assist in developing more specific interventions or treatments.

Figures 8.7, 8.8 and 8.9 here

Appendix 8.1 Conditional Distributions

(i) *Conditional distribution* $p(\boldsymbol{\alpha}, \mathbf{Z}|\cdot)$

In this part and following parts, we only discuss the situation that $\mathbf{z}_k$ includes the underlying unobservable continuous variables corresponding to $\mathbf{x}_k$, and $\mathbf{Z} = (\mathbf{z}_1, \cdots, \mathbf{z}_r)$ and $\mathbf{X} = (\mathbf{x}_1, \cdots, \mathbf{x}_r)$. Other situations that $\mathbf{Z}$ corresponds to $\mathbf{Y}$ and $\mathbf{U}$ are similar. Let $\boldsymbol{\alpha}_{kt} = (\alpha_{tk,2}, \cdots, \alpha_{tk,h_k})$, $\boldsymbol{\Lambda}_t^T$ be the tth row of $\boldsymbol{\Lambda}$, z_{tk} is the tth element of $\mathbf{z}_k$, then

$$p(\boldsymbol{\alpha}, \mathbf{Z}|\cdot) = \prod_{k=1}^{r}\prod_{t=1}^{T} p(\boldsymbol{\alpha}_{tk}, z_{tk}|\cdot) \propto \prod_{k=1}^{r}\prod_{t=1}^{T} \phi\{\psi_{xtk}^{-1/2}(z_{tk} - \boldsymbol{\Lambda}_t^T\boldsymbol{\xi}_k)\} I_{[\alpha_{tk,x_{tk}},\ \alpha_{tk,x_{tk}+1})}(z_{tk}), \tag{8.A1}$$

where $\phi(\cdot)$ is the standard normal density function.

(ii) *Conditional distribution* $p(\boldsymbol{\Xi}|\cdot)$.

Note that once $\mathbf{Z}$ is given, $\boldsymbol{\Xi}$ is independent of $\boldsymbol{\alpha}$ and $\mathbf{X}$. Note that $\boldsymbol{\zeta} = (\boldsymbol{\eta}^T, \boldsymbol{\xi}^T)^T$, we have

$$\begin{aligned} p(\boldsymbol{\zeta}|\boldsymbol{\theta}, \boldsymbol{\omega}, \mathbf{y}, \mathbf{z}, \mathbf{u}) &\propto p(\boldsymbol{\omega}, \mathbf{y}, \mathbf{z}, \mathbf{u}|\boldsymbol{\theta}, \boldsymbol{\zeta}) p(\boldsymbol{\zeta}|\boldsymbol{\theta}) \\ &\propto p(\mathbf{y}|\boldsymbol{\theta}, \boldsymbol{\eta}) p(\boldsymbol{\eta}|\boldsymbol{\xi}) p(\boldsymbol{\xi}|\boldsymbol{\theta}) \Big[\prod_{k=1}^{r} p(\mathbf{z}_k|\boldsymbol{\theta}, \boldsymbol{\xi}_k)\Big] \Big[\prod_{k=1}^{q} p(\boldsymbol{\omega}_k|\boldsymbol{\theta}, \boldsymbol{\xi}_{r+k})\Big]. \end{aligned}$$

It follows that $p(\boldsymbol{\zeta}|\cdot)$ is proportional to

$$\begin{aligned} &\exp\Bigg[-\frac{1}{2}\Big\{(\mathbf{y} - \boldsymbol{\Lambda}\boldsymbol{\eta})^T\boldsymbol{\Psi}_y^{-1}(\mathbf{y} - \boldsymbol{\Lambda}\boldsymbol{\eta}) + [\boldsymbol{\eta} - \boldsymbol{\beta} - \boldsymbol{\Lambda}_\delta\mathbf{G}(\boldsymbol{\zeta})]^T\boldsymbol{\Psi}_\delta^{-1}[\boldsymbol{\eta} - \boldsymbol{\beta} - \boldsymbol{\Lambda}_\delta\mathbf{G}(\boldsymbol{\zeta})] \\ &+ \boldsymbol{\xi}^T\boldsymbol{\Phi}^{-1}\boldsymbol{\xi} + \sum_{k=1}^{r}(\mathbf{z}_k - \boldsymbol{\Lambda}\boldsymbol{\xi}_k)^T\boldsymbol{\Psi}_{xk}^{-1}(\mathbf{z}_k - \boldsymbol{\Lambda}\boldsymbol{\xi}_k) + \sum_{k=1}^{q}(\boldsymbol{\omega}_k - \boldsymbol{\Lambda}\boldsymbol{\xi}_{r+k})^T\boldsymbol{\Psi}_{\omega k}^{-1}(\boldsymbol{\omega}_k - \boldsymbol{\Lambda}\boldsymbol{\xi}_{r+k})\Big\}\Bigg]. \end{aligned} \tag{8.A2}$$

(iii) *Conditional distribution* $p(\boldsymbol{\Omega}|\cdot)$.

Note that given $\boldsymbol{\Xi}$, $\boldsymbol{\Omega}$ is independent of $\boldsymbol{\alpha}$, $\mathbf{Z}$, $\mathbf{Y}$, and $\mathbf{X}$, and $\{\boldsymbol{\omega}_k,\ k = 1, \cdots, q\}$ is the appropriate permutation of $\{\boldsymbol{\omega}_{ut},\ t = 1, \cdots, T\}$, thus we have

$$p(\boldsymbol{\omega}|\cdot) = p(\boldsymbol{\omega}|\boldsymbol{\theta}, \boldsymbol{\xi}, \mathbf{u}) \propto p(\mathbf{u}|\boldsymbol{\theta}, \boldsymbol{\omega}) p(\boldsymbol{\omega}|\boldsymbol{\theta}, \boldsymbol{\xi}) = \prod_{t=1}^{T} p(\mathbf{u}_t|\boldsymbol{\theta}, \boldsymbol{\omega}_{ut}) \prod_{k=1}^{q} p(\boldsymbol{\omega}_k|\boldsymbol{\theta}, \boldsymbol{\xi}_{r+k}).$$

It follows that $p(\boldsymbol{\omega}|\cdot)$ is proportional to

$$\exp\Big[-\frac{1}{2}\Big\{\sum_{t=1}^{T}(\mathbf{u}_t-\boldsymbol{\Lambda}_{ut}\boldsymbol{\omega}_{ut})^T\boldsymbol{\Psi}_{ut}^{-1}(\mathbf{u}_t-\boldsymbol{\Lambda}_{ut}\boldsymbol{\omega}_{ut})+\sum_{k=1}^{q}(\boldsymbol{\omega}_k-\boldsymbol{\Lambda}\boldsymbol{\xi}_{r+k})^T\boldsymbol{\Psi}_{\omega k}^{-1}(\boldsymbol{\omega}_k-\boldsymbol{\Lambda}\boldsymbol{\xi}_{r+k})\Big\}\Big], \tag{8.A3}$$

where $\boldsymbol{\omega}_{ut}=(\omega_{ut1},\cdots,\omega_{utq})^T$ for $t=1,\cdots,T$, and $\boldsymbol{\omega}_k=(\omega_{u1k},\cdots,\omega_{uTk})^T$ for $k=1,\cdots,q$.

(iv) *Conditional Distribution* $p(\boldsymbol{\theta}|\cdot)$.

Let $\mathbf{Z}_k=(\mathbf{z}_{k1},\cdots,\mathbf{z}_{kn})$, $\mathbf{U}_t=(\mathbf{u}_{t1},\cdots,\mathbf{u}_{tn})$, $\boldsymbol{\Omega}_1=(\boldsymbol{\eta}_1,\cdots,\boldsymbol{\eta}_n)$, $\tilde{\boldsymbol{\Omega}}_1=(\boldsymbol{\eta}_1-\boldsymbol{\beta},\cdots,\boldsymbol{\eta}_n-\boldsymbol{\beta})$, $\boldsymbol{\Omega}_{2k}=(\boldsymbol{\omega}_{k1},\cdots,\boldsymbol{\omega}_{kn})$ for $k=1,\cdots,q$, $\boldsymbol{\Omega}_{3t}=(\boldsymbol{\omega}_{ut1},\cdots,\boldsymbol{\omega}_{utn})$ for $t=1,\cdots,T$, $\boldsymbol{\Xi}_k=(\boldsymbol{\xi}_{k1},\cdots,\boldsymbol{\xi}_{kn})$ for $k=1,\cdots,r+q$, $\boldsymbol{\Omega}_2=(\boldsymbol{\Xi}_1^T,\cdots,\boldsymbol{\Xi}_{r+q}^T)^T$, and $\mathbf{G}=(\mathbf{G}(\boldsymbol{\zeta}_1),\cdots,\mathbf{G}(\boldsymbol{\zeta}_n))$, where n is the sample size. Let $\mathbf{Y}_t^T$, $\mathbf{Z}_{tk}^T$, $\boldsymbol{\Omega}_{2tk}^T$, and $\boldsymbol{\Lambda}_t^T$ be the tth row of $\mathbf{Y},\mathbf{Z}_k$, $\boldsymbol{\Omega}_{2k}$, and $\boldsymbol{\Lambda}$, respectively, and let $\mathbf{U}_{tk}^T$, $\boldsymbol{\Omega}_{1k}^T$, $\tilde{\boldsymbol{\Omega}}_{1k}^T$, and $\boldsymbol{\Lambda}_{utk}^T$ be the kth row of $\mathbf{U}_t$, $\boldsymbol{\Omega}_1$, $\tilde{\boldsymbol{\Omega}}_1$, and $\boldsymbol{\Lambda}_{ut}$, respectively. It can be shown (see Lee and Song, 2003) that

$$\begin{aligned}
&[\psi_{yt}^{-1}|\mathbf{Y}_t,\boldsymbol{\Omega}_1,\boldsymbol{\Lambda}_t]\overset{D}{=}Gamma[n/2+a_{0yt},b_{yt}],\quad t=1,\cdots,T,\\
&[\psi_{xtk}^{-1}|\mathbf{Z}_k,\boldsymbol{\Xi}_k,\boldsymbol{\Lambda}_t]\overset{D}{=}Gamma[n/2+a_{0xtk},b_{xtk}],\quad k=1,\cdots,r,\quad t=1,\cdots,T,\\
&[\psi_{\omega tk}^{-1}|\boldsymbol{\Omega}_{2k},\boldsymbol{\Xi}_{r+k},\boldsymbol{\Lambda}_t]\overset{D}{=}Gamma[n/2+a_{0\omega tk},b_{\omega tk}],\quad k=1,\cdots,q,\quad t=1,\cdots,T,\\
&[\boldsymbol{\Lambda}_{utk}|\mathbf{U}_t,\boldsymbol{\Omega}_{3t},\psi_{utk}]\overset{D}{=}N[\boldsymbol{\mu}_{utk},\psi_{utk}\boldsymbol{\Sigma}_{utk}],\quad k=1,\cdots,p,\quad t=1,\cdots,T,\\
&[\psi_{utk}^{-1}|\mathbf{U}_t,\boldsymbol{\Omega}_{3t}]\overset{D}{=}Gamma[n/2+a_{0utk},b_{utk}],\quad k=1,\cdots,p,\quad t=1,\cdots,T,\\
&[\boldsymbol{\Lambda}_{\delta k}|\tilde{\boldsymbol{\Omega}}_1,\boldsymbol{\Omega}_2,\psi_{\delta k}]\overset{D}{=}N[\boldsymbol{\mu}_{\delta k},\psi_{\delta k}\boldsymbol{\Sigma}_{\delta k}],\quad k=1,\cdots,m,\\
&[\psi_{\delta k}^{-1}|\tilde{\boldsymbol{\Omega}}_1,\boldsymbol{\Omega}_2]\overset{D}{=}Gamma[n/2+a_{0\delta k},b_{\delta k}],\quad k=1,\cdots,m,\\
&[\boldsymbol{\beta}|\boldsymbol{\Omega}_1,\boldsymbol{\Omega}_2,\boldsymbol{\Lambda}_\delta,\boldsymbol{\Psi}_\delta]\overset{D}{=}N[\boldsymbol{\mu}_\beta,\boldsymbol{\Sigma}_\beta],\quad [\boldsymbol{\Phi}|\boldsymbol{\Omega}_2]\overset{D}{=}IW_{m(r+q)}[(\boldsymbol{\Omega}_2\boldsymbol{\Omega}_2^T+\mathbf{R}_0^{-1}),n+\rho_0],
\end{aligned} \tag{8.A4}$$

where $b_{yt}=b_{0yt}+[\mathbf{Y}_t^T\mathbf{Y}_t-2\boldsymbol{\Lambda}_t^T\boldsymbol{\Omega}_1\mathbf{Y}_t+\boldsymbol{\Lambda}_t^T(\boldsymbol{\Omega}_1\boldsymbol{\Omega}_1^T)\boldsymbol{\Lambda}_t]/2$, $b_{xtk}=b_{0xtk}+[\mathbf{Z}_{tk}^T\mathbf{Z}_{tk}-2\boldsymbol{\Lambda}_t^T\boldsymbol{\Xi}_k\mathbf{Z}_{tk}+\boldsymbol{\Lambda}_t^T(\boldsymbol{\Xi}_k\boldsymbol{\Xi}_k^T)\boldsymbol{\Lambda}_t]/2$, $b_{\omega tk}=b_{0\omega tk}+[\boldsymbol{\Omega}_{2tk}^T\boldsymbol{\Omega}_{2tk}-2\boldsymbol{\Lambda}_t^T\boldsymbol{\Xi}_{r+k}\boldsymbol{\Omega}_{2tk}+\boldsymbol{\Lambda}_t^T(\boldsymbol{\Xi}_{r+k}\boldsymbol{\Xi}_{r+k}^T)\boldsymbol{\Lambda}_t]/2$, $\boldsymbol{\mu}_{utk}=\boldsymbol{\Sigma}_{utk}(\boldsymbol{\Sigma}_{0utk}^{-1}\boldsymbol{\Lambda}_{0utk}+\boldsymbol{\Omega}_{3t}\mathbf{U}_{tk})$, $\boldsymbol{\Sigma}_{utk}=(\boldsymbol{\Sigma}_{0utk}^{-1}+\boldsymbol{\Omega}_{3t}\boldsymbol{\Omega}_{3t}^T)^{-1}$, $b_{utk}=b_{0utk}+[\mathbf{U}_{tk}^T\mathbf{U}_{tk}-$

$\boldsymbol{\mu}_{utk}^T\boldsymbol{\Sigma}_{utk}^{-1}\boldsymbol{\mu}_{utk}+\boldsymbol{\Lambda}_{0utk}^T\boldsymbol{\Sigma}_{0utk}^{-1}\boldsymbol{\Lambda}_{0utk}]/2$, $\boldsymbol{\mu}_{\delta k}=\boldsymbol{\Sigma}_{\delta k}(\boldsymbol{\Sigma}_{0\delta k}^{-1}\boldsymbol{\Lambda}_{0\delta k}+\mathbf{G}\tilde{\boldsymbol{\Omega}}_{1k})$, $\boldsymbol{\Sigma}_{\delta k}=(\boldsymbol{\Sigma}_{0\delta k}^{-1}+\mathbf{G}\mathbf{G}^T)^{-1}$, $b_{\delta k}=b_{0\delta k}+[\tilde{\boldsymbol{\Omega}}_{1k}^T\tilde{\boldsymbol{\Omega}}_{1k}-\boldsymbol{\mu}_{\delta k}^T\boldsymbol{\Sigma}_{\delta k}^{-1}\boldsymbol{\mu}_{\delta k}+\boldsymbol{\Lambda}_{0\delta k}^T\boldsymbol{\Sigma}_{0\delta k}^{-1}\boldsymbol{\Lambda}_{0k}]/2$, $\boldsymbol{\mu}_\beta=\boldsymbol{\Sigma}_\beta[\boldsymbol{\Psi}_\delta^{-1}\sum_{i=1}^n(\boldsymbol{\eta}_i-\boldsymbol{\Lambda}_\delta\mathbf{G}(\boldsymbol{\eta},\boldsymbol{\xi}))+\boldsymbol{\Sigma}_0^{-1}\boldsymbol{\beta}_0]$, and $\boldsymbol{\Sigma}_\beta=(\boldsymbol{\Sigma}_0^{-1}+n\boldsymbol{\Psi}_\delta^{-1})^{-1}$.

The conditional distributions involved in (8.A3) and (8.A4) are standard, simulating observations from them is straightforward. As the conditional distributions in (8.A1) and (8.A2) are nonstandard, the Metropolis-Hastings (MH, Metropolis *et al.*, 1953; Hastings, 1970) algorithm will be used to simulate observations from them, see Cowles (1996), Lee and Song (2003).

Appendix 8.2 WinBUGS Code in the Analysis of Cocaine Use Data

```
model{
     for(i in 1:N){
     # structural equation
     xi[i,1:6]~dmnorm(u[1:6],phi[1:6,1:6])
     for(j in 1:2){ xxi[i,j]~dnorm(nu[i,j],psd[j]) }
     nu[i,1]<-vu[1]+gam[1]*xi[i,1]+gam[2]*xi[i,3]+gam[3]*xi[i,5]
     +gam[4]*xi[i,1]*xi[i,3]+gam[5]*xi[i,1]*xi[i,5]+gam[6]*xi[i,3]*xi[i,5]
     nu[i,2]<-vu[2]+gam[7]*xi[i,2]+gam[8]*xi[i,4]+gam[9]*xi[i,6]
     +gam[10]*xi[i,2]*xi[i,4]+gam[11]*xi[i,2]*xi[i,6]+gam[12]*xi[i,4]*xi[i,6]

     # measurement models
     for(j in 1:20){ y[i,j]~dnorm(mu[i,j],psi[j]) }
         mu[i,1]<- xxi[i,1]
         mu[i,2]<- xxi[i,1]+xxi[i,2]
         mu[i,3]<- xxi[i,1]+2*xxi[i,2]
         mu[i,4]<- xxi[i,1]+12*xxi[i,2]
         mu[i,5]<- xi[i,1]
         mu[i,6]<- xi[i,1]+xi[i,2]
         mu[i,7]<- xi[i,1]+2*xi[i,2]
         mu[i,8]<- xi[i,1]+12*xi[i,2]
         mu[i,9]<- xi1[i,1]
         mu[i,10]<- lam[1]*xi1[i,1]
         mu[i,11]<- lam[2]*xi1[i,1]
```

```
  mu[i,12]<- xi1[i,2]
  mu[i,13]<- lam[3]*xi1[i,2]
  mu[i,14]<- lam[4]*xi1[i,2]
  mu[i,15]<- xi1[i,3]
  mu[i,16]<- lam[5]*xi1[i,3]
  mu[i,17]<- lam[6]*xi1[i,3]
  mu[i,18]<- xi1[i,4]
  mu[i,19]<- lam[7]*xi1[i,4]
  mu[i,20]<- lam[8]*xi1[i,4]

  xi1[i,1]<- xi[i,3]
  xi1[i,2]<- xi[i,3]+xi[i,4]
  xi1[i,3]<- xi[i,3]+2*xi[i,4]
  xi1[i,4]<- xi[i,3]+12*xi[i,4]

for(j in 1:4){ z1[i,j]~dcat(p1[i,j,1:C1])
               p1[i,j,1]<-Q1[i,j,1]
               for(t in 2:C1-1){ p1[i,j,t]<- Q1[i,j,t]-Q1[i,j,t-1] }
               p1[i,j,C1]<- 1-Q1[i,j,C1-1]
for(t in 1:C1-1){ logit(Q1[i,j,t])<- alph1[j,t]-mu[i,20+2*j-1] } }  #end j

for(j in 1:4){ z2[i,j]~dcat(p2[i,j,1:C2])
               p2[i,j,1]<-Q2[i,j,1]
               for(t in 2:C2-1){ p2[i,j,t]<- Q2[i,j,t]-Q2[i,j,t-1] }
               p2[i,j,C2]<- 1-Q2[i,j,C2-1]
for(t in 1:C2-1){ logit(Q2[i,j,t])<- alph2[j,t]-mu[i,20+2*j] } }  #end j

mu[i,21]<- xi2[i,1]
mu[i,22]<- lam[9]*xi2[i,1]
mu[i,23]<- xi2[i,2]
mu[i,24]<- lam[10]*xi2[i,2]
mu[i,25]<- xi2[i,3]
mu[i,26]<- lam[11]*xi2[i,3]
mu[i,27]<- xi2[i,4]
mu[i,28]<- lam[12]*xi2[i,4]
```

```
   xi2[i,1]<- xi[i,5]
   xi2[i,2]<- xi[i,5]+xi[i,6]
   xi2[i,3]<- xi[i,5]+2*xi[i,6]
   xi2[i,4]<- xi[i,5]+12*xi[i,6] }

  # thresholds for latent variables

for(j in 1:4){ alph1[j,2]~ dnorm(0.0, 2.0)I(alph1[j,1],) }

for(j in 1:4){
          alph2[j,2]~ dnorm(0.0, 2.0)I(alph2[j,1],alph2[j,3])
          alph2[j,3]~ dnorm(0.0, 2.0)I(alph2[j,2],alph2[j,4])
          alph2[j,4]~ dnorm(0.0, 2.0)I(alph2[j,3],) }

        for(j in 1:4){ alph1[j, 1]<- 0   alph2[j, 1]<- 0 }

 # priors on loadings and coefficients
 vu[1]~dnorm(0.0,4.0)      vu[2]~dnorm(0.0,4.0)
 for(j in 1:12){ lam[j]~ dnorm(1.0, 4.0) }
 for(j in 1:12){ gam[j]~ dnorm(1.0, 4.0) }

# priors on precisions
 for(j in 1:20){ psi[j]~dgamma(10.0,4.0)
                  v[j]<-1/psi[j] }
 for(j in 1:2) { psd[j]~dgamma(10.0,4.0)
                  vd[j]<- 1/psd[j] }
 phi[1:6,1:6]~dwish(RR[1:6,1:6],6)
 phx[1:6,1:6]<- inverse(phi[1:6,1:6])

 # put all the parameters' results into bb

 for(j in 1:2){ bb[j]<- vu[j]  }
 for(j in 1:12){ bb[2+j]<- lam[j] }
 for(j in 1:12){ bb[14+j]<- gam[j] }
 for(j in 1:2){ bb[26+j]<- vd[j] }
 for(j in 1:20){ bb[28+j]<- v[j] }
```

```
     for(j in 1:6){ bb[48+j]<- phx[1,j] }
     for(j in 2:6){ bb[54+j-1]<- phx[2,j] }
     for(j in 3:6){ bb[59+j-2]<- phx[3,j] }
     for(j in 4:6){ bb[63+j-3]<- phx[4,j] }
     for(j in 5:6){ bb[66+j-4]<- phx[5,j] }
     for(j in 6:6){ bb[68+j-5]<- phx[6,j] }
     for(j in 1:4){ bb[69+j]<- alph1[j,2] }
     for(j in 1:4){ bb[73+j]<- alph2[j,2] }
     for(j in 1:4){ bb[77+j]<- alph2[j,3] }
     for(j in 1:4){ bb[81+j]<- alph2[j,4] }
}

# init 1 list(paste initial value 1)
# init 2 list(paste initial value 2)
# init 3 list(paste initial value 3)

#Data list(N=321, u=c(0,0,0,0,0,0),  C1=3, C2=5,
    RR=structure(.Data= c(1.0, 0.0, 0.0, 0.0, 0.0, 0.0,
                          0.0, 1.0, 0.0, 0.0, 0.0, 0.0,
                          0.0, 0.0, 1.0, 0.0, 0.0, 0.0,
                          0.0, 0.0, 0.0, 1.0, 0.0, 0.0,
                          0.0, 0.0, 0.0, 0.0, 1.0, 0.0,
                          0.0, 0.0, 0.0, 0.0, 0.0, 1.0), .Dim= c(6,6)),
y=structure(.Data=c(paste continuous variable), .Dim=c(321,20)),
z1=structure(.Data=c(paste the first ordinal variable), .Dim=c(321,4)),
z2=structure(.Data=c(paste the second ordinal variable), .Dim=c(321,4)))
} #end
```

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Table 8.1: Bayesian estimates and their standard error estimates of the parameters in the cocaine study example.

Par	Est	Std	Par	Est	Std	Par	Est	Std
$\lambda_{u1,21}$	2.083	0.053	ψ_{y1}	1.361	0.136	ϕ_{11}	0.669	0.077
$\lambda_{u1,31}$	1.828	0.051	ψ_{y2}	0.564	0.068	ϕ_{12}	-0.052	0.009
$\lambda_{u2,21}$	2.169	0.052	ψ_{y3}	0.564	0.065	ϕ_{13}	0.528	0.109
$\lambda_{u2,31}$	2.173	0.055	ψ_{y4}	0.293	0.070	ϕ_{14}	0.014	0.021
$\lambda_{u3,21}$	2.028	0.046	ψ_{x11}	0.591	0.065	ϕ_{15}	0.708	0.091
$\lambda_{u3,31}$	2.091	0.051	ψ_{x21}	0.746	0.073	ϕ_{16}	0.002	0.006
$\lambda_{u4,21}$	1.959	0.065	ψ_{x31}	0.614	0.063	ϕ_{22}	0.015	0.001
$\lambda_{u4,31}$	1.843	0.063	ψ_{x41}	0.249	0.052	ϕ_{23}	-0.031	0.012
γ_{11}	-0.064	0.291	ψ_{u11}	0.554	0.054	ϕ_{24}	-0.007	0.002
γ_{12}	0.383	0.362	ψ_{u12}	0.203	0.034	ϕ_{25}	-0.040	0.012
γ_{13}	0.159	0.353	ψ_{u13}	0.384	0.048	ϕ_{26}	0.002	0.000
γ_{14}	-0.123	0.293	ψ_{u21}	0.608	0.056	ϕ_{33}	1.430	0.293
γ_{15}	0.016	0.254	ψ_{u22}	0.220	0.037	ϕ_{34}	0.023	0.034
γ_{16}	-0.014	0.173	ψ_{u23}	0.437	0.056	ϕ_{35}	1.596	0.209
γ_{21}	0.006	0.135	ψ_{u31}	0.547	0.051	ϕ_{36}	-0.009	0.009
γ_{22}	-0.034	0.065	ψ_{u32}	0.159	0.024	ϕ_{44}	0.053	0.014
γ_{23}	0.517	0.219	ψ_{u33}	0.344	0.043	ϕ_{45}	0.045	0.039
γ_{24}	0.619	0.410	ψ_{u41}	0.604	0.060	ϕ_{46}	-0.000	0.001
γ_{25}	0.951	0.494	ψ_{u42}	0.181	0.028	ϕ_{55}	2.064	0.190
γ_{26}	0.704	0.465	ψ_{u43}	0.277	0.035	ϕ_{56}	-0.012	0.011
β_1	0.566	0.254	$\psi_{\omega 1}$	0.175	0.025	ϕ_{66}	0.007	0.000
β_2	-0.060	0.014	$\psi_{\omega 2}$	0.128	0.017	$\alpha_{1,2}$	2.703	0.196
$\psi_{\eta 1}$	0.340	0.060	$\psi_{\omega 3}$	0.126	0.016	$\alpha_{2,2}$	3.172	0.218
$\psi_{\eta 2}$	0.038	0.003	$\psi_{\omega 4}$	0.163	0.026	$\alpha_{3,2}$	3.425	0.236
						$\alpha_{4,2}$	2.502	0.269

Table 8.2: Bayesian estimates, standard error estimates, and root mean squares of the parameters in simulation study.

True value	Mean	Rms	True value	Mean	Rms	True value	Mean	Rms
$\lambda_{u1,21}$=0.8	0.810	0.038	ψ_{y1}=0.5	0.479	0.056	ϕ_{11}=1.0	0.986	0.087
$\lambda_{u1,31}$=0.8	0.815	0.044	ψ_{y2}=0.5	0.497	0.040	ϕ_{12}=0.3	0.310	0.058
$\lambda_{u2,21}$=0.8	0.805	0.023	ψ_{y3}=0.5	0.497	0.049	ϕ_{13}=0.3	0.367	0.116
$\lambda_{u2,31}$=0.8	0.805	0.025	ψ_{y4}=0.5	0.482	0.084	ϕ_{14}=0.3	0.332	0.078
$\lambda_{u3,21}$=0.8	0.800	0.013	ψ_{x11}=0.5	0.509	0.056	ϕ_{15}=0.3	0.289	0.064
$\lambda_{u3,31}$=0.8	0.800	0.017	ψ_{x21}=0.5	0.504	0.038	ϕ_{16}=0.3	0.303	0.063
$\lambda_{u4,21}$=0.8	0.799	0.012	ψ_{x31}=0.5	0.499	0.046	ϕ_{22}=1.0	1.006	0.067
$\lambda_{u4,31}$=0.8	0.800	0.013	ψ_{x41}=0.5	0.494	0.079	ϕ_{23}=0.3	0.378	0.117
γ_{11}= 0.6	0.621	0.070	ψ_{u11}=0.5	0.506	0.049	ϕ_{24}=0.3	0.320	0.074
γ_{12}=-0.6	-0.567	0.085	ψ_{u12}=0.5	0.502	0.045	ϕ_{25}=0.3	0.287	0.059
γ_{13}= 0.6	0.622	0.081	ψ_{u13}=0.5	0.500	0.046	ϕ_{26}=0.3	0.302	0.050
γ_{14}=-0.4	-0.353	0.089	ψ_{u21}=0.5	0.503	0.044	ϕ_{33}=1.0	1.182	0.277
γ_{15}= 0.4	0.454	0.108	ψ_{u22}=0.5	0.499	0.039	ϕ_{34}=0.3	0.509	0.238
γ_{16}=-0.4	-0.375	0.088	ψ_{u23}=0.5	0.501	0.040	ϕ_{35}=0.3	0.333	0.104
γ_{21}=-0.6	-0.573	0.054	ψ_{u31}=0.5	0.506	0.054	ϕ_{36}=0.3	0.403	0.133
γ_{22}= 0.6	0.584	0.054	ψ_{u32}=0.5	0.498	0.041	ϕ_{44}=1.0	1.132	0.210
γ_{23}=-0.6	-0.574	0.051	ψ_{u33}=0.5	0.495	0.042	ϕ_{45}=0.3	0.345	0.087
γ_{24}= 0.4	0.390	0.057	ψ_{u41}=0.5	0.491	0.048	ϕ_{46}=0.3	0.318	0.080
γ_{25}=-0.4	-0.392	0.055	ψ_{u42}=0.5	0.500	0.042	ϕ_{55}=1.0	0.939	0.132
γ_{26}= 0.4	0.387	0.062	ψ_{u43}=0.5	0.504	0.036	ϕ_{56}=0.3	0.311	0.056
β_1= 0.5	0.464	0.070	$\psi_{\omega 1}$=0.5	0.513	0.073	ϕ_{66}=1.0	1.010	0.077
β_2=-0.5	-0.466	0.053	$\psi_{\omega 2}$=0.5	0.503	0.062			
$\psi_{\eta 1}$=0.36	0.388	0.062	$\psi_{\omega 3}$=0.5	0.491	0.058			
$\psi_{\eta 2}$=0.36	0.359	0.043	$\psi_{\omega 4}$=0.5	0.501	0.089			
$\alpha_{1,2}$ = 1.0	1.200	0.218	$\alpha_{1,3}$ = 2.0	2.264	0.278			
$\alpha_{2,2}$ = 1.0	1.104	0.132	$\alpha_{2,3}$ = 2.0	2.211	0.173			
$\alpha_{3,2}$ = 1.0	1.049	0.100	$\alpha_{3,3}$ = 2.0	2.064	0.123			
$\alpha_{4,2}$ = 1.0	0.967	0.096	$\alpha_{4,3}$ = 2.0	1.962	0.109			

Table 8.3: Results of Latent Curve Model Analysis for PHY, PSY, SO, and EN domains of HRQOL.

	Initial Level of ADLs (ξ_1)	Initial Level of Handicap (ξ_3)	Initial Level of Depression (ξ_5)
Initial Level of PHY	0.174* (0.069) [0.039, 0.307]	-0.475* (0.097) [-0.669, -0.290]	-0.310* (0.068) [-0.439, -0.177]
Initial Level of PSY	0.167* (0.068) [0.034, 0.303]	-0.070 (0.095) [-0.193, 0.184]	-0.782* (0.068) [-0.910, -0.647]
Initial Level of SO	0.032 (0.096) [-0.166, 0.211]	0.088 (0.135) [-0.182, 0.350]	-0.511* (0.096) [-0.698, -0.330]
Initial Level of EN	0.075 (0.081) [-0.084, 0.232]	-0.191 (0.114) [-0.431, 0.025]	-0.540* (0.081) [-0.700, -0.385]
	Rate of Change of ADLs (ξ_2)	Rate of Change of Handicap (ξ_4)	Rate of Change of Depression (ξ_6)
Rate of Change of PHY	0.280 (0.167) [-0.031, 0.617]	-0.312 (0.163) [-0.623, 0.014]	-0.328* (0.152) [-0.645, -0.042]
Rate of Change of PSY	0.168 (0.166) [-0.139,0.510]	-0.179 (0.163) [-0.499, 0.139]	-0.445* (0.146) [-0.726, -0.154]
Rate of Change of SO	-0.083 (0.203) [-0.478, 0.315]	-0.001 (0.201) [-0.381, 0.404]	-0.417* (0.186) [-0.771, -0.045]
Rate of Change of EN	-0.128 (0.169) [-0.459, 0.202]	-0.282 (0.168) [-0.617, 0.042]	-0.539* (0.155) [-0.848, -0.242]

Note: Values are coefficient estimates, standard error estimates (given in parentheses) and 95% Highest Probability Density (HPD) Interval (given in [ ]). The estimates with asterisk are statistically significant as their associated HPD intervals do not include zero.

Abbreviations: QOL - WHO QOL (abbreviated Hong Kong version); PHY - physical health; PSY - psychological health; SO - social interaction; EN - environment; ADL - activities of daily living.

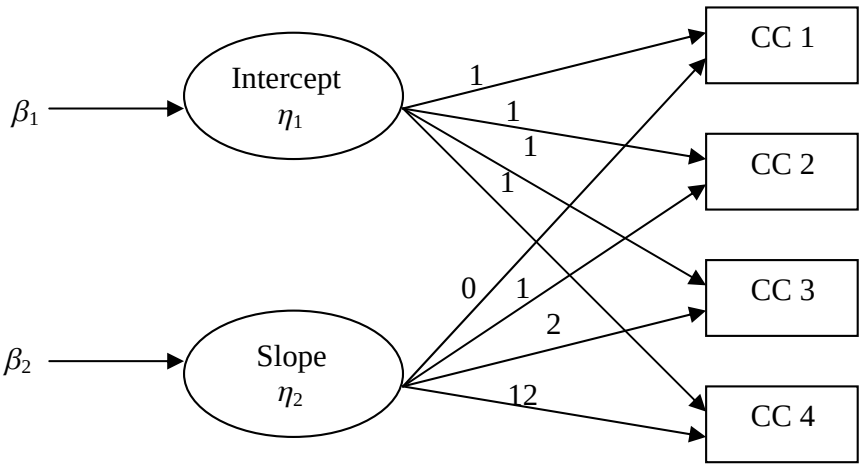

Figure 8.1: Path diagram of the model defined in (8.4) and (8.5). In this and the following figures, the error terms are not displayed for brevity.

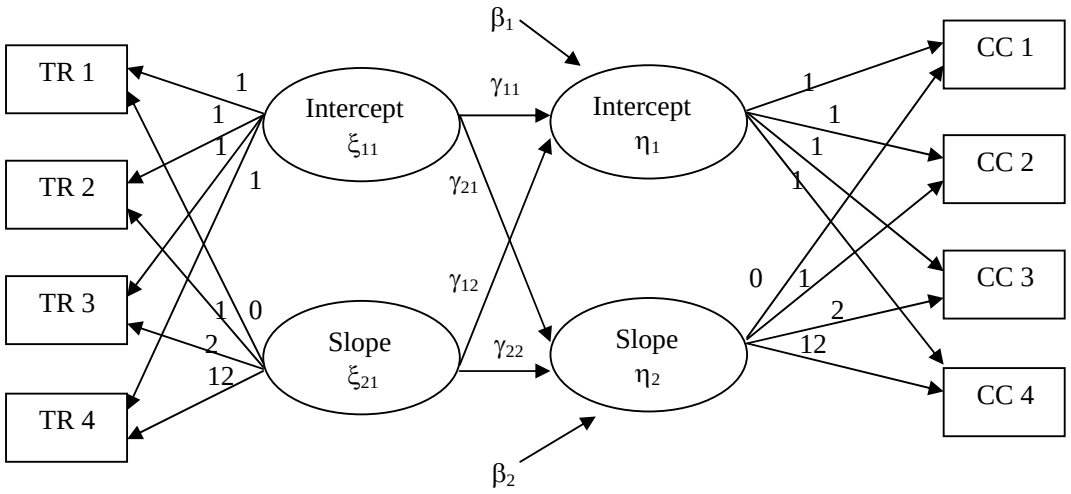

Figure 8.2: Path diagram of the model defined in (8.4), (8.7), and (8.8).

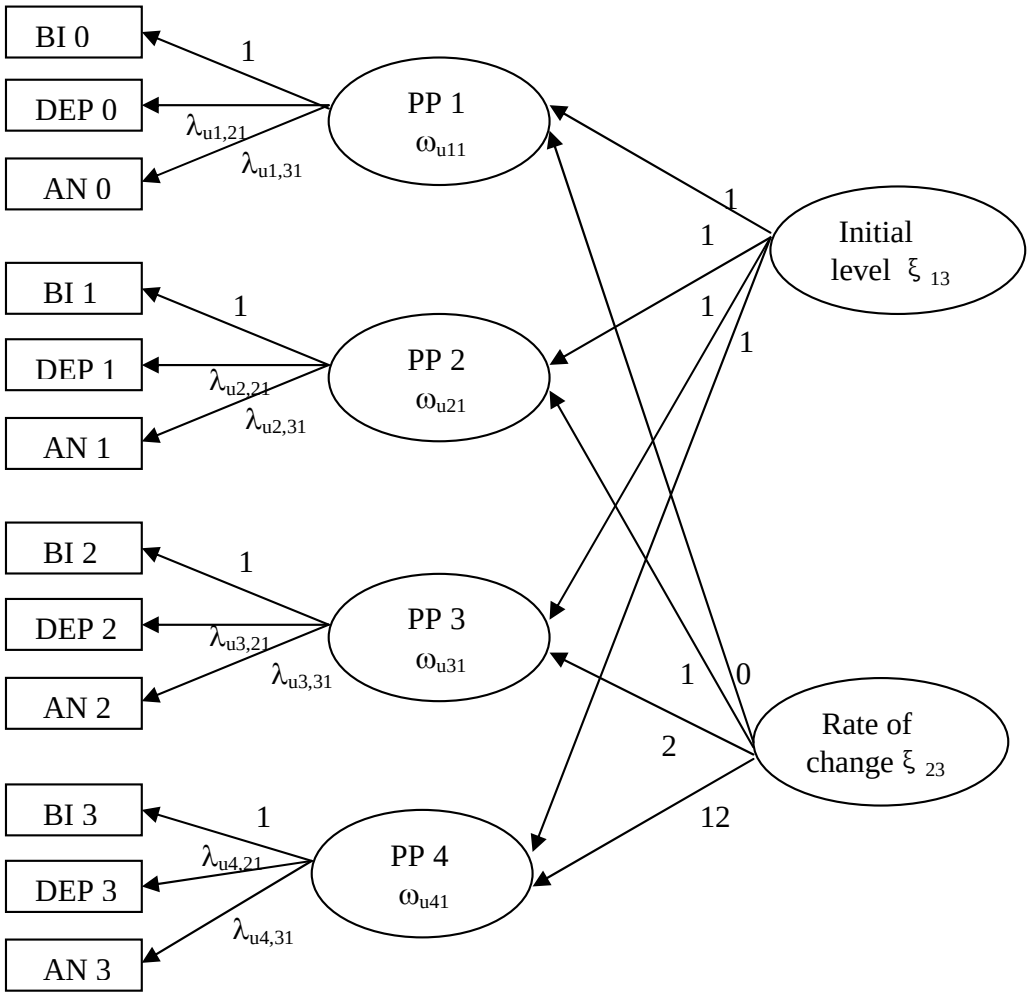

Figure 8.3: Path diagram of the model defined in (8.13) and (8.14).

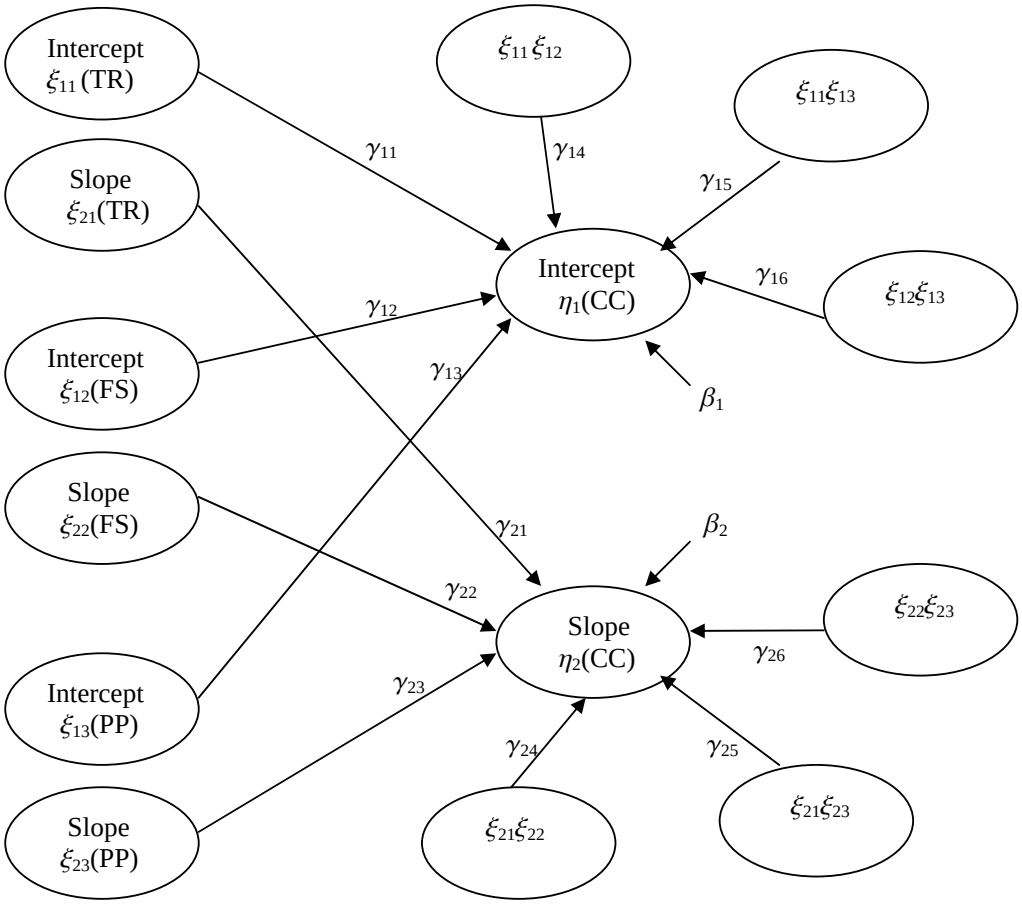

Figure 8.4: Path diagram of the model defined in (8.17) and (8.18).

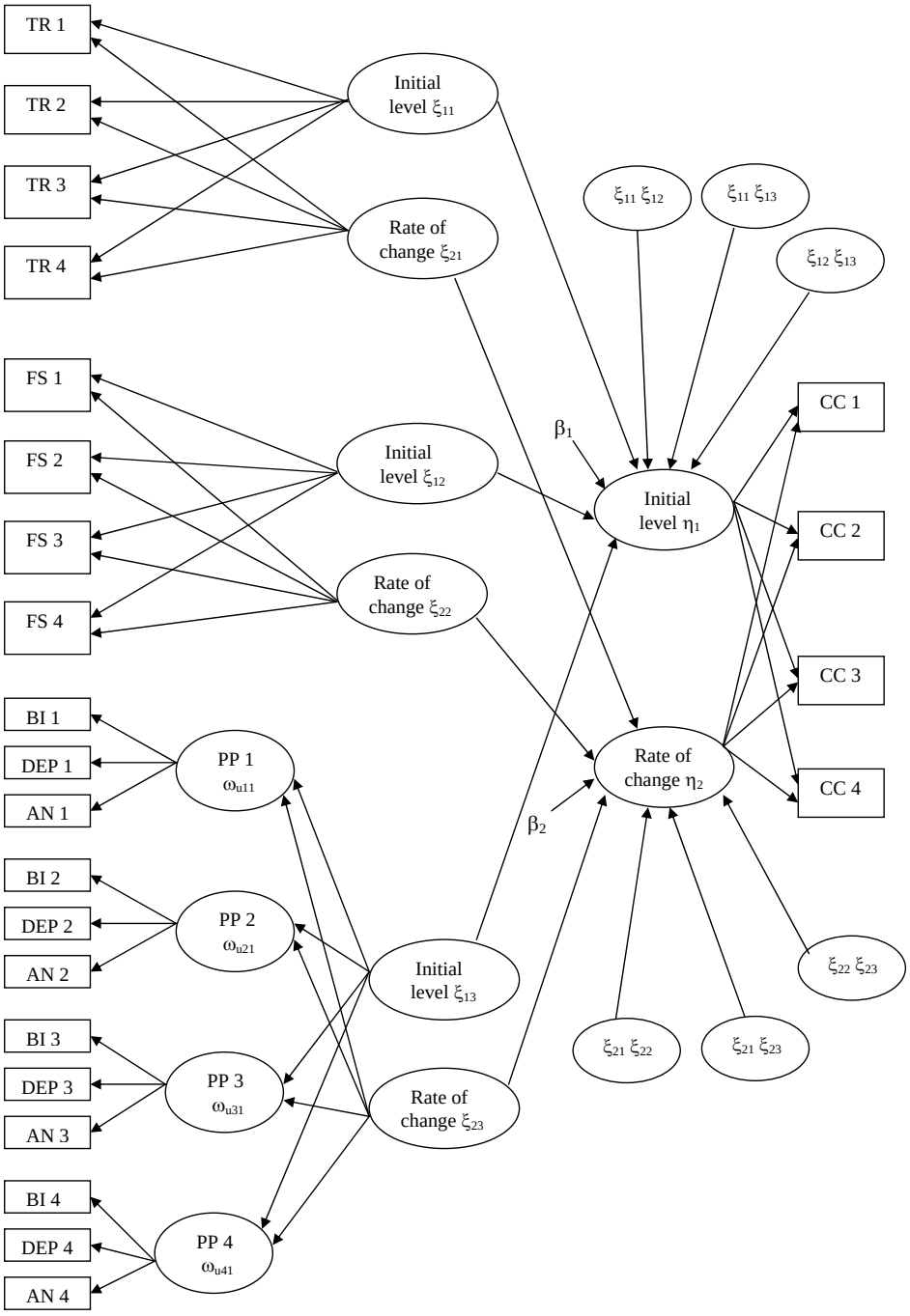

Figure 8.5: Path diagram of the LCM for the longitudinal study of cocaine use.

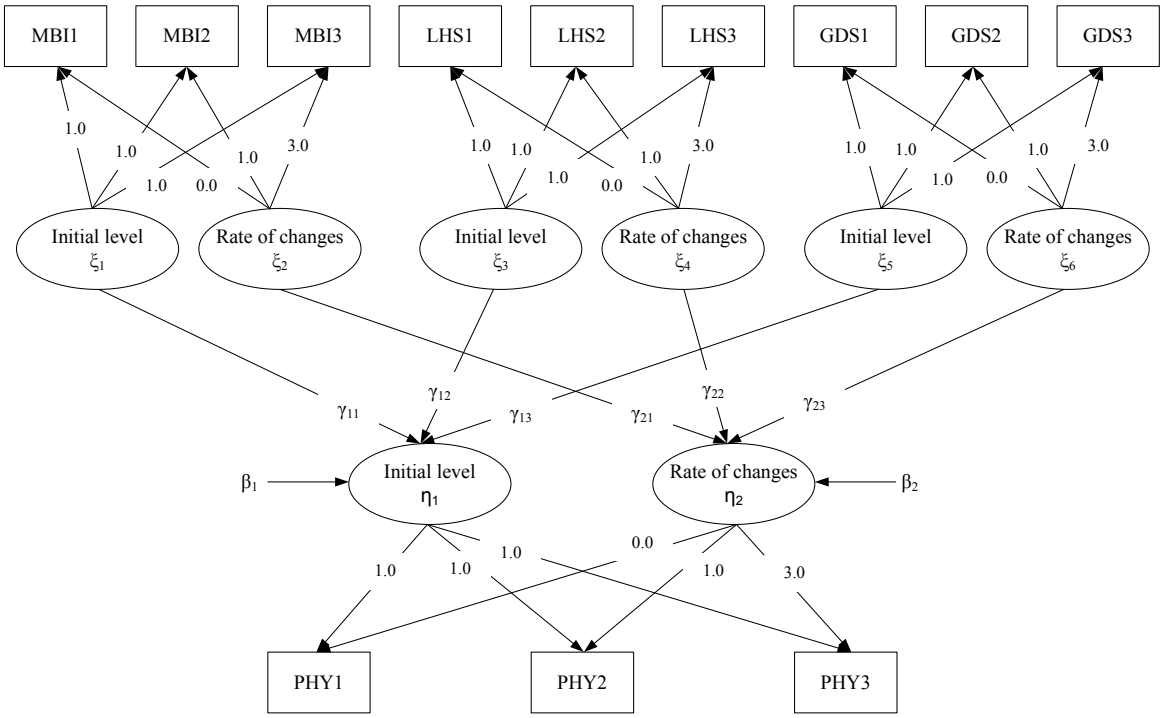

Figure 8.6: Path diagram of the LCM for HRQOL of stroke survivors.

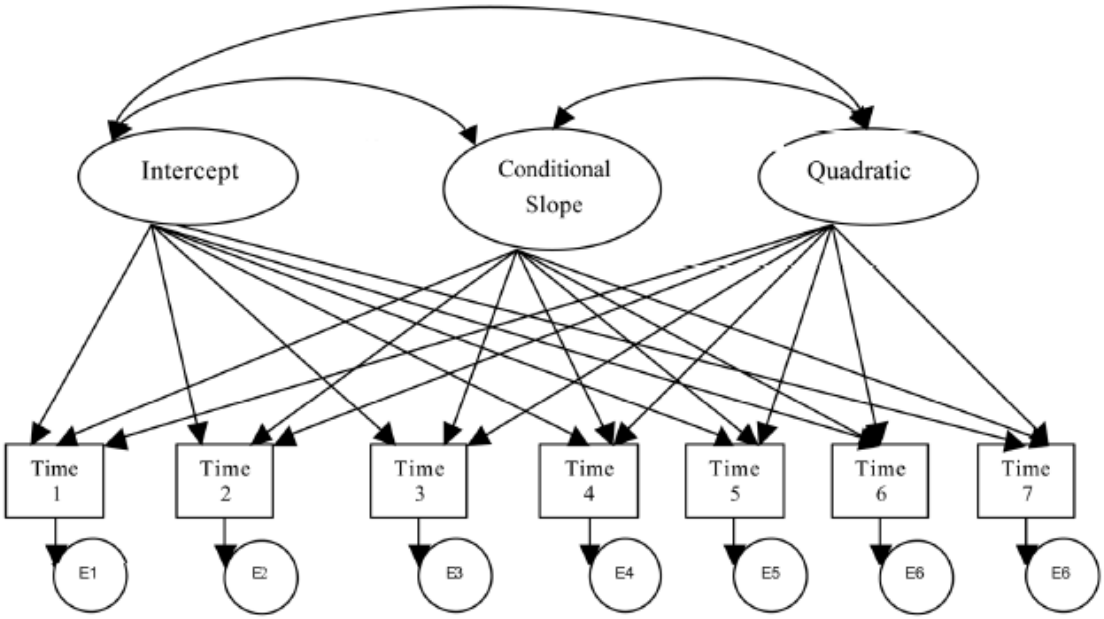

Figure 8.7: Quadratic LCM of depression across seven time points (taken from Beal *et al.*, 2007)

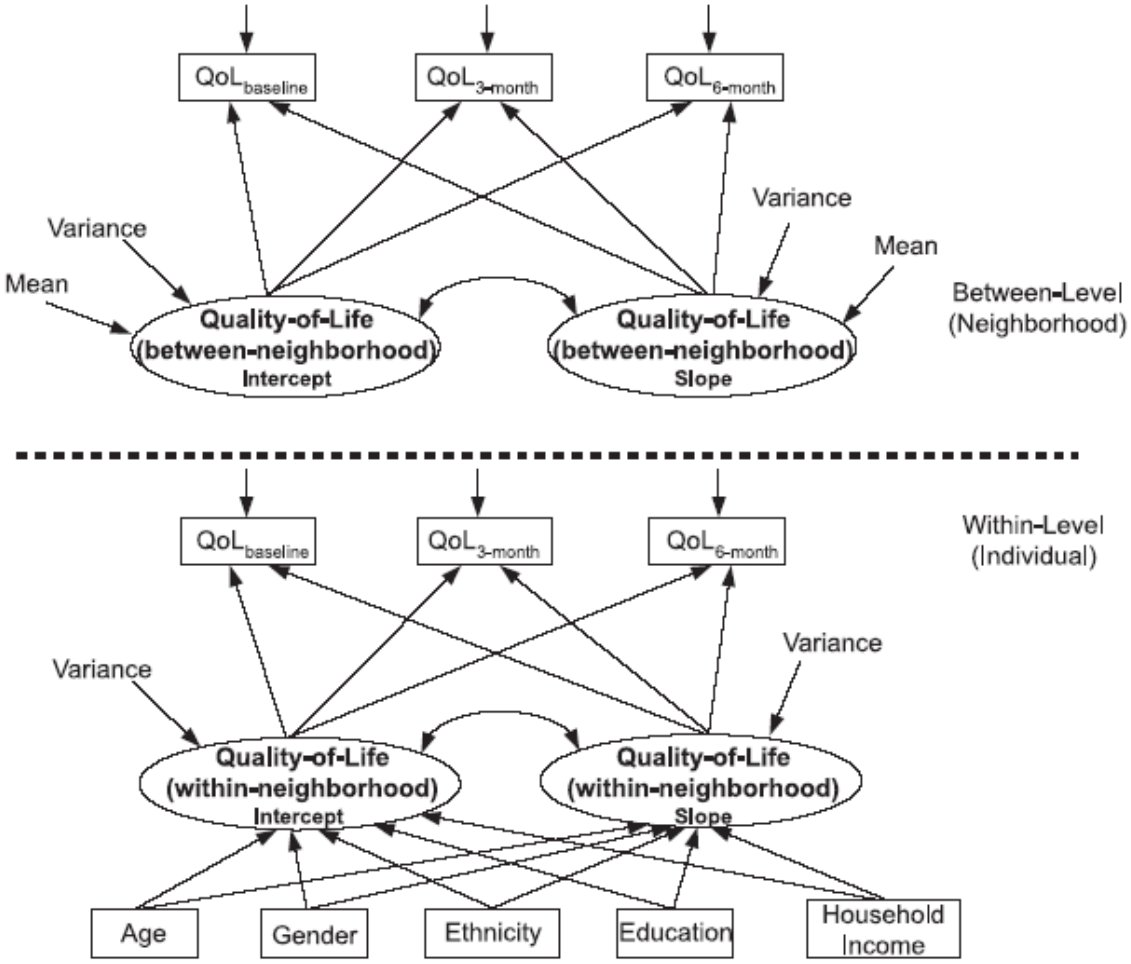

Figure 8.8: Multilevel LCM for QOL in older adults (taken from Fisher and Li, 2004).

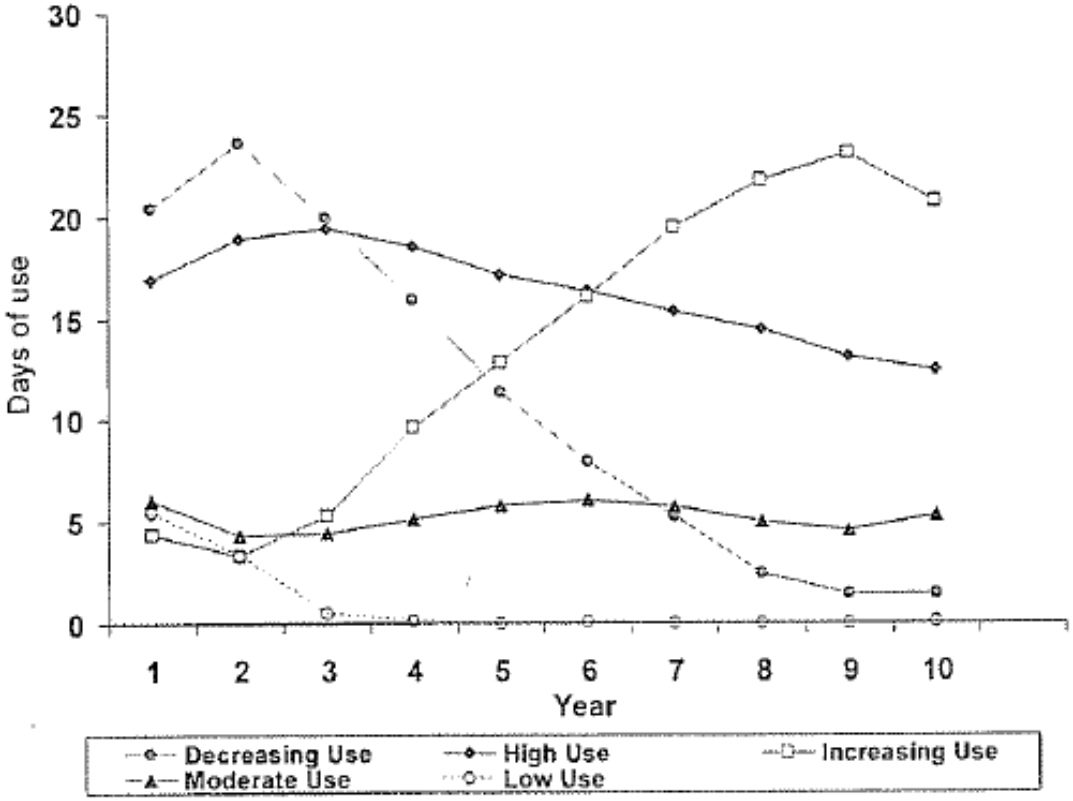

Figure 8.9: Five-group classification of drug use trajectories based on mixture LCM (taken from Hser *et al.*, 2008).