



Dynamics of Change and Change in Dynamics

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Abstract: A framework is presented for building and testing models of dynamic regulation by categorizing sources of differences between theories of dynamics. A distinction is made between the dynamics of change, i.e., how a system self-regulates on a short time scale, and change in dynamics, i.e., how those dynamics may themselves change over a longer time scale. In order to clarify the categories, models are first built to estimate individual differences in equilibrium value and equilibrium change. Next, models are presented in which there are individual differences in parameters of dynamics such as frequency of fluctuations, damping of fluctuations, and amplitude of fluctuations. Finally, models for within-person change in dynamics over time are proposed. Simulations demonstrating feasibility of these models are presented and OpenMx scripts for fitting these models have been made available in a downloadable archive along with scripts to simulate data so that a researcher may test a selected models' feasibility within a chosen experimental design.

Keywords: dynamical systems analysis, multi-timescale analysis, self-regulation, person-oriented analysis

Dynamics of Change

Much recent work has concentrated on psychological processes that are hypothesized to exhibit regulatory dynamics. These processes typically change relatively quickly: Variables measuring these processes change over time scales of as short as milliseconds to as long as days. Processes hypothesized to exhibit regulatory dynamics include perception–action processes (Jeka, Oie, & Kiemel, 2000; Wohlschläger, Gattis, & Bekkering, 2003), daily fluctuations in self-perceived mental health in recent widows (Bisconti, Bergeman, & Boker, 2004, 2006), self-disclosure and intimacy in married couples (Hamaker, Zhang, & van der Maas, 2009; Laurenceau, Barrett, & Rovine, 2005; Laurenceau, Feldman Barrett, & Rovine, 2005), symptoms of disordered eating in young women (Edler, Lipson, & Keel, 2007), and positive and negative affect (Chow, Ram, Boker, Fujita, & Clore, 2005; Deboeck, Monpetit, Bergeman, & Boker, 2009; Zautra, Affleck, Tennen, Reich, &

Davis, 2005). In each of these cases, some proportion of the observed short-term changes in observed scores are patterned in a time-dependent way that could reveal important clues about regulatory mechanisms. For the purposes of this article, the patterning of short-term change over time will be referred to as *dynamics of change* — the dynamical processes that are inferred from short term changes or fluctuations in time-intensive repeated observations. Most often, when authors write about the dynamics of a process, they mean the dynamics of change.

It is reasonable to think that there may be individual differences in the dynamics of change. That is to say, two individuals may regulate in somewhat different ways. Individual differences in the dynamics of change may take a variety of forms. The current article will categorize several types of individual differences in the dynamics of change and incorporate them into a common modeling framework. In this way, parameters can be estimated and statistical tests can be performed for hypotheses concerning relationships be-

tween dynamics of change and other person-specific characteristics.

The dynamics of change can be studied using *measurement bursts* (Nesselroade, 1991); in other words, one or more sequences of repeated observations closely spaced in time with longer intervals between them. For instance, daily diary studies (Laurenceau, Barrett, & Rovine, 2005, e.g.,) and experience sampling methods (Bolger, Davis, & Rafaeli, 2003; Cranford et al., 2006) can be considered examples of burst measurement with only one burst. Methods such as time-delay embedding (Sauer, Yorke, & Casdagli, 1991; Oertzen & Boker, 2010), latent differential equations (Boker, Neale, & Rausch, 2004), latent difference scores (McArdle, 2001; Hamagami & McArdle, 2007), Kalman filtering (Kalman, 1960; Molenaar & Newell, 2003; So, Ott, & Dayawansa, 1994), dynamic factor analysis (Molenaar, 1985), and state-space (Chow, Hamaker, Fujita, & Boker, 2009) methods have been used to estimate parameters of the dynamics of change from burst measurement data.

If characteristics of the dynamics of change are constant over time, the dynamics are said to exhibit stationarity (see, e.g., Hendry & Juselius, 2000; Shao & Chen, 1987, for discussions of stationarity). This is a convenient assumption, as it means that for a selected individual any interval of time can be considered to be a representative sample of the dynamics of change for that individual. However, an assumption of stationarity frequently does not hold for psychological processes. Some processes, such as the dynamics of head movements during conversation (Ashenfelter, Boker, Waddell, & Vitanov, 2009; Boker, Xu, Rotondo, & King, 2002), may be nonstationary during the same short time scales in which they exhibit their dynamics of change — that is to say these processes may exhibit *nonstationary regulation*. While such processes comprise an interesting category of psychological phenomena, they will be considered as outside the scope of the current article. We will focus on a large category of phenomena where nonstationarity is observed, but operates at a slower time scale than the dynamics of change for the process. This difference in temporal scale can be exploited to provide simultaneous estimates of the short-term dynamics of change and longer term change in dynamics.

Change in Dynamics

Many psychological phenomena exhibit slow nonstationarity relative to their regulatory processes. Adaptation, learning, or developmental processes are examples of nonstationarity that could play out over a time scale of hours to decades. For instance, day-to-day or minute-to-minute emotional regulation may itself exhibit a developmental trajectory, comprising within-individual differences in these regulatory mechanisms between childhood, midlife, and late life. Such a process may exhibit *approximate local stationarity*, that is to say over relatively short time scales, the characteristics of the dynamics of change may remain reasonably constant. However, over longer time scales, a process with approximate local stationarity can exhibit what will be referred to as *change in dynamics* —

relatively slow evolution of the parameters of the dynamics of change. We can take advantage of this difference in time scales to incorporate change in dynamics into existing modeling frameworks for individual differences in the dynamics of change so that hypotheses can be tested concerning the relationship of within-individual change in dynamics to other person-specific characteristics.

Two major categories of change in dynamics are: i) change in the equilibrium set for the process and ii) change in the attractor basin around the equilibrium set (Boker, 2013). The first category of change in dynamics is associated with the *equilibrium set*, the set of equilibrium states, for a system. One simple and commonly used type of equilibrium set is a *point equilibrium* (or homeostatic equilibrium) which is an equilibrium set with only one point in it. A point equilibrium means that there is one “best” or “optimal” or “goal” state for the system and that the system regulates relative to that state. There are many other types of equilibria (see, e.g., Hubbard & West, 1991, for a discussion) including, for instance, a *zone equilibrium* where many values for a variable are equally good (Boker, 2013). This so-called “comfort zone” is illustrated as the flat bottom of the leftmost of the three basins in Figure 1. One way that long-term change in dynamics may occur is that there may be changes in the equilibrium set. For instance, there might be long term developmental change in the location of the centroid of the equilibrium, e.g., an individual’s average level of overall arousal might decrease from adolescence to mid-life. Another possibility is that the type of equilibrium set might change, e.g., from a point equilibrium to a zone equilibrium.

The second category of change in dynamics is defined by changes in the basin of attraction of the regulatory system and can be estimated as change in the parameters of the differential equation or difference equation that define the short-term dynamics of change. For instance, individuals might change in how they regulate — in adolescence a person might tolerate large swings in overall arousal while in mid-life that same person might exhibit much greater regulatory control over their arousal levels as shown in Figure 1.

In the arousal example for both categories of change in dynamics, short-term dynamics have stable regulatory characteristics, but over a longer timescale, these regulatory dynamics may exhibit interesting and important change.

In order to estimate dynamics of change as well as change in dynamics, measurements must be sufficiently intensive in time to capture short term fluctuations as well as have sufficient longitudinal spread to be able to estimate developmental changes. Multiple burst designs are an efficient way to acquire these data. Sort bursts of time-intensive measurements (e.g., daily diary self-report or ecological momentary assessments) can be separated by longer intervals of time, e.g., months or years (e.g. Ong, Bergeman, & Boker, 2009). In this way, change in dynamics may be estimated from longitudinal changes in within-individual parameters of the dynamics of change estimated from each burst. However, it may not be immediately obvious what these changes in dynamics might mean. The

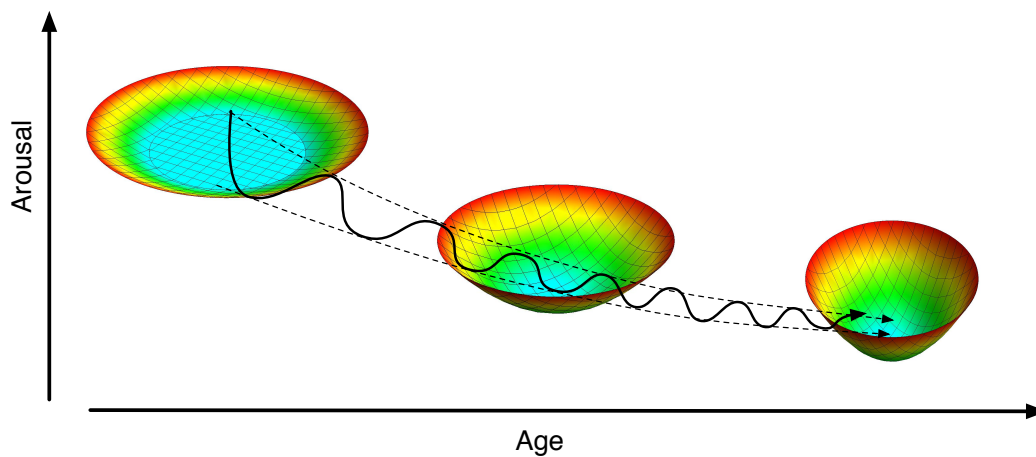


Figure 1. A hypothetical arousal regulation system exhibiting two categories of change in dynamics. i) the equilibrium at young ages is a zone equilibrium set such that many similar levels of relatively high arousal are equally good and resulting in wide swings of arousal. In contrast, at older ages a point equilibrium emerges where there is a single best, and somewhat lower, level of arousal. ii) the attractor basin in the younger age is shown as having a flat bottom and shallow sides indicating a low degree of regulation while in older ages a steeper basin emerges indicating a greater degree of regulation around the equilibrium thus resulting in smaller arousal fluctuations.

next section builds an individual differences model for the dynamics of change a step at a time and discusses the meanings for individual differences in each of the parameters of the model. In this way, the stage is set for the last portion of the article which will discuss the meaning of within-individual change in these parameters and will present a model framework for estimating changes in dynamics simultaneously with the dynamics of change.

Interindividual Differences in Dynamics of Change

Let us consider three ways in which individuals may differ in their dynamics of change: i) individual differences in equilibria; ii) differences in amplitude of fluctuations; and iii) individual differences in the parameters of a selected model of the dynamics. One strategy to estimate the magnitude of individual differences in the dynamics of change is to allow particular coefficients of a chosen model to take on individual values for each person in a burst sample. The specifics of this strategy will be presented in a later section, but first let us examine the implications of individual differences in dynamics of change.

First, individuals may differ in their equilibria. For a selected variable, each individual may have a value around which they fluctuate. As an example, consider a study of cognitive abilities. On a day to day basis, a selected individual may perform better or worse on a cognitive task than her or his mean performance. Thus, for a cognitive performance variable, the mean performance over repeated observations might be a reasonable estimate of a point equilibrium for each individual and the individual differences in within-person means could give an estimate of individual differences in equilibria. Another example might be a

variable such as degree of intimacy a married individual feels with her or his spouse. Each individual husband or wife might have a preferred level of intimacy. Greater or lesser intimacy than the preferred level might be felt on any selected day and regulatory processes might keep the degree of intimacy somewhere near the equilibrium. But there may be individual differences within the class of husbands as well as within the class of wives. Also, within couples, it may be that similarities or difference between the two spouses' equilibrium values may be predictive of marital satisfaction. Estimating and accounting for individual differences in equilibria is an important part of understanding regulatory systems.

Second, it may be that the amplitude of fluctuations around the equilibrium may differ across individuals. This might be due to individual differences in context or individual differences in regulatory dynamics. As an example of contextual differences, one person might be in a high-stress job whereas another person might be have a relaxed, low-stress occupation. The person in the high-stress job may have some days that are very difficult and troubling and other days that are fantastically rewarding. Each day for the person in the low-stress job may be quite like the day before: relaxing but not terribly rewarding either. Individual differences in contextual stressors for these two individuals might manifest as differences in amplitude of fluctuations in positive and negative affect. The affect of the person in the high-stress job is likely to show greater day-to-day fluctuations even if she regulates her affect in a very similar way to the person in the low stress job. It is possible that the individual with the high stress job has self-selected her occupation in part due to her ability to effectively regulate stress. Whereas the individual in the relaxed occupation may be less able to regulate his affective fluctuations due to stress. Thus, the amplitude of observed fluctuations in affect may be due to both the contextual ef-

fects (the differences in stress levels of the job) as well as individual differences in regulatory dynamics. If these two individuals were to be presented with equivalent contexts, the same amount of stress may lead to small fluctuations in affect in the person who effectively regulates affect whereas it might lead to large fluctuations in affect for the person who does not regulate effectively. When modeling dynamics of change, it is important to be able to distinguish between individual differences in context from individual differences in the model parameters controlling the dynamics.

Third, there may be individual differences in regulatory dynamics. That is to say, the dynamics by which one person regulates may be somewhat different than it is for another person even after accounting for differences in equilibrium and differences in exogenous input. For instance, a large magnitude stressor might resonate for one person for a relatively long time, resulting in large fluctuations in affect that take weeks to die away. Whereas, for a second individual fluctuations associated with the same stressor might be damped within a matter of a day or two. Or, one person's fluctuations might be rather slow relative to a second person. These individual differences in dynamics can be substantively important and be related to other variables. For instance, in a study of recently-bereaved widows, Bisconti and colleagues (2006) found that how quickly fluctuations were damped was related to the degree of reported emotion focused coping provided by the widows' social support network.

Intraindividual Change in Dynamics

Let us now consider the same three sources of differences, but interpreted as ways in which the characteristics of an individual's regulatory dynamics might slowly change. Models of change in dynamics must account for the fact that while individuals in a study are assumed to be independent of one another, a longitudinal model must be used to account for intraindividual change in dynamics.

The first way that intraindividual change in dynamics might occur is that the equilibrium value for the individual might shift over time. For instance, in a daily diary study, many recently bereaved widows exhibited a slow increase in a Mental Health Inventory (MHI) measure over a period of months (Bisconti et al., 2004). At the same time, these widows showed large daily fluctuations in MHI that could be modeled as a linear oscillator. The equilibrium point for the oscillations slowly changed over a period of months as the widows learned to deal with their loss.

A second possible way in which an individual's regulatory dynamics might change is that there could be a slowly evolving increase or decrease of amplitude of fluctuations about an equilibrium. For instance, an individual's context may be changing across time and thus creating new exogenous input to the system. A job might become more stressful over time or retirement might change the pattern and frequency of stressors. In such cases, the regulatory dynamics might stay constant while the observed amplitude

of fluctuations could increase or decrease. Changes in context are not necessarily examples of change in dynamics of regulatory systems. These changes might be entirely external to the regulatory system under study. However, when contexts are self-selected, one might consider contextual changes as part of a long-term adaptive strategy.

A third possibility is that one or more parameters for the selected dynamical systems model might change over time, implying that the regulatory dynamics are themselves changing. As an example, a younger individual might poorly regulate large fluctuations in mood, but later in life learn to become accomplished in such regulation.

Dynamical systems models are required to account for the regulatory process and longitudinal models are required in order to account for long term within-person change. These models must be able to discern the difference between changes in equilibrium, changes in external context, and changes in dynamics. Thus, a common framework is required that simultaneously models the dynamics of change and longitudinal change in dynamics. We will next provide a brief introduction of one common model for the dynamics of change, a second order linear differential equation. We will then extend this model to account for individual differences in equilibria, individual differences in the dynamics of change, and intraindividual change in dynamics.

Example Model: Second Order Linear Differential Equation

Differential equations can be used for the specification of models for dynamical systems in psychology (e.g., Boker, 2012). These models specify a set of relations between the time derivatives of the variables involved in the regulatory system. The time derivative of a variable, written as either dx/dt or as \dot{x} , is the amount of change occurring in the variable at a specific moment in time. Thus, these differential equation models are quite literally models of the dynamics of change.

Linear second order differential equations have recently been used to model a variety of psychological processes. This model is intuitively appealing due to its correspondence to physical systems that have been used as metaphors for psychological systems. Continuously variable thermostats, pendulums, and springs may all be modeled to a first approximation with linear second order differential equations. As an example, psychological resilience is often described as the ability to "bounce back" from adversity. Hooke's Law shows us that a simple model for elasticity with dissipation (akin to a bouncing ball that eventually comes to rest) is a linear second order differential equation (see Boker, Montpetit, Hunter, & Bergeman, 2010, for an extended discussion).

The linear second order differential equation for a variable x can be written as

$$\ddot{x}(t) = \eta x(t) + \zeta \dot{x}(t) \quad (1)$$

where x is the displacement of a variable from its equilib-

rium. That is to say, the variable x is centered at its equilibrium point. When a variable's equilibrium is fixed and known, we subtract that value from x to center the variable. When a variable's equilibrium is unknown, a number of strategies have been employed. We will discuss this question in greater detail in the next section.

Note that the parameters η and ζ have substantive meaning. If $\eta < 0$, then one may say that the farther the system is from equilibrium, the greater the acceleration that would turn it back towards equilibrium. Similarly if $\zeta < 0$, one may say that the faster the system is changing, the greater the deceleration. As a way of intuitively grasping the impact of these parameters, it might be helpful to think about an automatic driver for a car. If $\eta < 0$ then the farther the car is from its garage (i.e., equilibrium), the more it tends to accelerate towards the garage. If $\zeta < 0$, then the faster the car is going, the more it tends to apply the brake.

This linear second order system has the interesting property that if $\eta < 0$ and $\eta + \zeta^2/4 < 0$, then oscillations form. One may use the parameters of the equation to calculate the period of the oscillation (the time it takes for one full oscillation) as $\lambda = \frac{2\pi}{\sqrt{-(\eta+\zeta^2/4)}}$. As will be seen in a later section, this linear second order equation can be used to model a variety of dynamic behaviors.

Figure 2 plots a time series for 50 measurements for two simulated individuals, a and b who have the same parameters $\eta = -0.3$ and $\zeta = -0.1$. However, a and b start at two different initial values. That is to say, at time $t = 0$, a's score is 2.5 and b's score is 0. The smooth curves are "true score" values that obey the regulatory dynamics of a second order linear differential equation. The noisy curves are just the smooth curve at each measurement occasion $t > 0$ plus a time-independent normally distributed random value with mean of zero and standard deviation of one. Note that if one were to take an average of a large sample of these curves, the "true scores" would cancel entirely since the initial starting point for each curve is a random value. Thus, a standard latent growth curve approach would completely miss the dynamics in these data and report that there was only time-independent residual error.

In order to estimate parameters of differential equations models from observed data, a number of techniques can be employed. Exact discrete (Singer, 1993), approximate discrete (Oud & Jansen, 2000; Oud, 2007), and Kalman filter methods (Molenaar & Newell, 2003; Chow et al., 2009) can be used to estimate parameters of differential equation systems by first taking the integral and then estimating time lagged data. Another method, Latent Differential Equations (LDE) (Boker et al., 2004; Boker, 2007b, 2007a) will be used here. Although the modeling framework presented in this article does not preclude using these other estimation methods, the specifics of model setup would be quite different than the examples presented here. At the core of the LDE method is *time delay embedding*, a time series technique that came to prominence in nonlinear dynamical systems analysis in physics (Sauer et al., 1991; Takens, 1985; Whitney, 1936).

Time Delay Embedding

Time delay embedding is a preprocessing step that ensures that each row of one's data set has encapsulated within it sufficient information to model the relevant dynamics of change. Then, an estimation method such as LDE or others can be used to model these dynamics. In essence, a short time-sequential *snip* of data is placed on each row. The starting point for each row is varied across all of the possible starting points for each individual. The number of columns in a snip is model dependent and data dependent, so this step takes some careful thought.

Suppose a time series X has been centered around each individual's equilibrium values. If the original time series X is ordered by occasion j within individual i then the series of all observations $x_{(i,j)}$ for N people, each of whom have been sampled P times, can be written as a vector of scores

$$X = \{x_{(1,1)}, \dots, x_{(1,P)}, x_{(2,1)}, \dots, x_{(2,P)}, \dots, x_{(N,1)}, \dots, x_{(N,P)}\}.$$

If we choose to create 5 columns in each snip, we can now construct a 5-dimensional time delay embedded matrix $\mathbf{X}^{(5)}$ as

$$\begin{bmatrix} x_{(1,1)} & x_{(1,2)} & x_{(1,3)} & x_{(1,4)} & x_{(1,5)} \\ x_{(1,2)} & x_{(1,3)} & x_{(1,4)} & x_{(1,5)} & x_{(1,6)} \\ \vdots & \vdots & \vdots & \vdots & \vdots \\ x_{(1,P-4)} & x_{(1,P-3)} & x_{(1,P-2)} & x_{(1,P-1)} & x_{(1,P)} \\ x_{(2,1)} & x_{(2,2)} & x_{(2,3)} & x_{(2,4)} & x_{(2,5)} \\ x_{(2,2)} & x_{(2,3)} & x_{(2,4)} & x_{(2,5)} & x_{(2,6)} \\ \vdots & \vdots & \vdots & \vdots & \vdots \\ x_{(2,P-4)} & x_{(2,P-3)} & x_{(2,P-2)} & x_{(2,P-1)} & x_{(2,P)} \\ \vdots & \vdots & \vdots & \vdots & \vdots \\ x_{(N,1)} & x_{(N,2)} & x_{(N,3)} & x_{(N,4)} & x_{(N,5)} \\ \vdots & \vdots & \vdots & \vdots & \vdots \\ x_{(N,P-4)} & x_{(N,P-3)} & x_{(N,P-2)} & x_{(N,P-1)} & x_{(N,P)} \end{bmatrix}.$$

Note that if one looks at the time delay embedded matrix column-wise, each column contains the time series lagged by an amount that is dependent on the column number. For instance, the second column contains almost the same data as the first column, except that it has been shifted up one row within each individual's block of data.

The power of this method lies in the fact that each row of the matrix contains a sample of the time dependency information. One side-effect of this is that the ordering of the rows of a time delay embedded matrix does not affect an analysis since the time-dependent information has been captured within each row. This fact carries with it the great advantage that so-called phase resetting phenomena do not have large impacts on the parameter estimates of models fit from time delay embedded matrices (Deboeck & Boker, 2010). A phase reset might occur when a substantial external event creates an abrupt change in the target variable value. The variable's value is subsequently regulated back towards equilibrium. Such phase resets are common in psychological data. For instance, a participant might be enrolled in a daily diary study of affect. On some

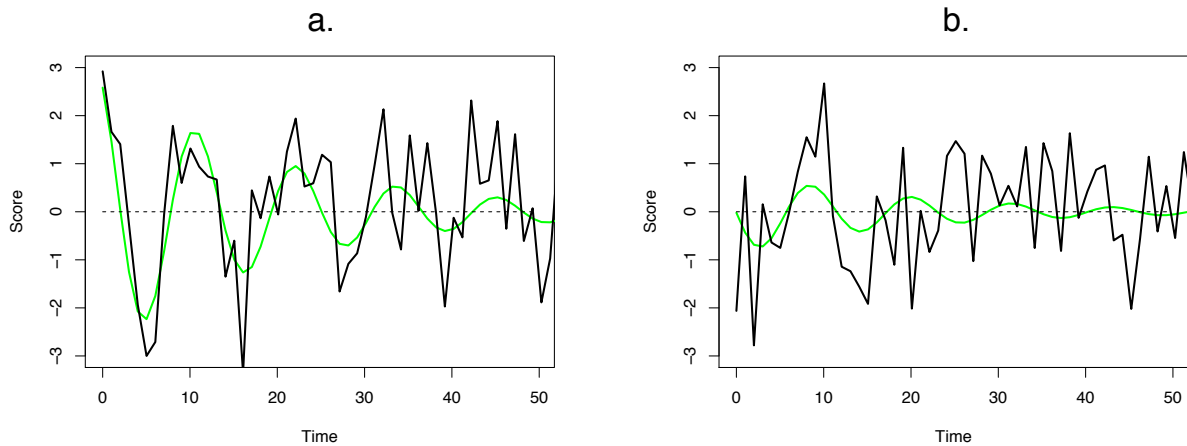


Figure 2. Time series plots of 50 measurements of two simulated individuals, a and b. The smooth line is the underlying dynamic and the noisy line has time-independent error.

random day during the study, the participant has a minor car crash, causing a marked and abrupt disturbance to her affect. Over the next few days she regulates back to equilibrium. But then after another random interval, she wins a prize, creating a second disturbance in her affect for a few days. When we model affect, we are not interested in accounting for the interval between the car crash and the prize. This interval is not part of the affective regulatory system. Time delay embedding isolates these exogenous effects and balances their impact so that they induce little bias in estimating parameters of regulation (Oertzen & Boker, 2010). However, time delay embedding carries with it a problem, in that the distribution of minus two times the log likelihood is not chi-square distributed. Until this problem is solved, standard error estimates for time delay embedding cannot be obtained by normal parametric methods, but must be estimated by methods such as bootstrapping.

Second Order Latent Differential Equation

As an example, we will restrict our discussion to variations on a linear second order Latent Differential Equation (LDE) for the dynamics of change of one variable (see Boker, 2007b, for a step by step introduction). By starting with a simple model, we can more easily focus later sections' discussion on extensions that are necessary to account for interindividual differences in dynamics and intraindividual change in dynamics. Figure 3 presents a path diagram of the second order LDE of a variable x using a 5-dimensional time delay embedded matrix $\mathbf{X}^{(5)}$. The latent second derivative with respect to time, \ddot{g} , for the j th row for person i in $\mathbf{X}^{(5)}$ is modeled as linear combination of the latent displacement, g , and its latent first derivative, \dot{g} ,

$$\ddot{g}_{ij} = \eta g_{ij} + \zeta \dot{g}_{ij} + e_{ij}, \tag{2}$$

where the residual term is assumed to be zero mean, independent, and identically distributed.

In matrix form, a second order LDE model for $\mathbf{X}^{(5)}$ can be specified as

$$\mathbf{X}^{(5)} = \mathbf{GL} + \mathbf{U}, \tag{3}$$

Where \mathbf{G} is a matrix of unobserved latent derivative scores, \mathbf{U} is a matrix of unobserved unique scores and \mathbf{L} is a fixed matrix defined as

$$\mathbf{L} = \begin{bmatrix} 1 & -2\Delta t & (-2\Delta t)^2/2 \\ 1 & -1\Delta t & (-1\Delta t)^2/2 \\ 1 & 0 & 0 \\ 1 & 1\Delta t & (1\Delta t)^2/2 \\ 1 & 2\Delta t & (2\Delta t)^2/2 \end{bmatrix}, \tag{4}$$

where Δt is the elapsed time between adjacent lagged columns in the time-delay embedded matrix $\mathbf{X}^{(5)}$.

Fitting the model to data involves using the model-implied covariances between the columns of \mathbf{G} to provide estimates of η and ζ , the residual variance for \ddot{g} and for the covariance matrix for \mathbf{U} , which is constrained to be diagonal with equal variances: a scalar matrix. We will use RAM structural equation modeling covariance algebra (McArdle & McDonald, 1984) and path diagram conventions (McArdle & Boker, 1990; Boker, McArdle, & Neale, 2002) to set up the model for the latent variables, specifying the covariance between columns of \mathbf{G} as a product of asymmetric and symmetric paths contained in two matrices \mathbf{A} and \mathbf{S} ,

$$\mathbf{A} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ \eta & \zeta & 0 \end{bmatrix} \tag{5}$$

$$\mathbf{S} = \begin{bmatrix} V_g & C_{g\dot{g}} & 0 \\ C_{g\dot{g}} & V_{\dot{g}} & 0 \\ 0 & 0 & V_{\ddot{g}} \end{bmatrix}, \tag{6}$$

where $V_g, V_{\dot{g}}, V_{\ddot{g}}$, and $C_{g\dot{g}}$ are the variances and covariances of the latent variables g, \dot{g} , and the residual variance for \ddot{g} .

Now, the model implied covariance matrix, $\text{Cov}(\mathbf{G})$, of the columns of the latent score matrix \mathbf{G} can be calculated

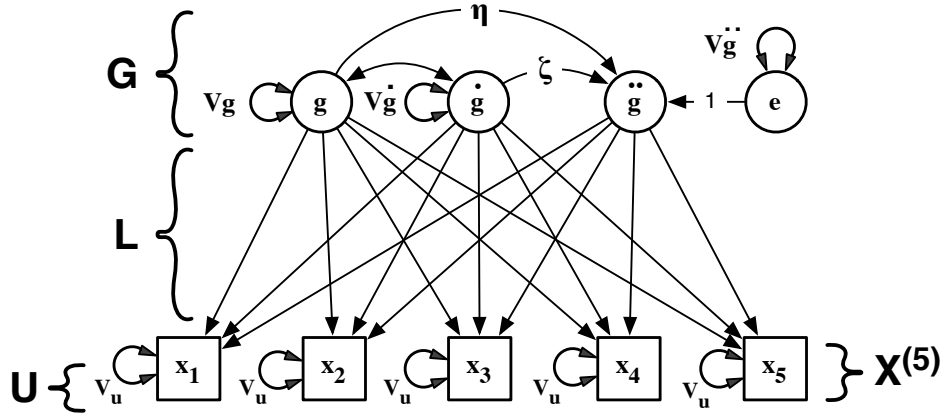


Figure 3. Path diagram of a second order linear latent differential equation model of a 5–dimensional time delay embedded matrix for a single variable. The latent second derivative, \ddot{g} , is a linear combination of the latent displacement, g , and latent first derivative, \dot{g} .

as

$$\text{Cov}(\mathbf{G}) = (\mathbf{I} - \mathbf{A})^{-1} \mathbf{S} (\mathbf{I} - \mathbf{A})^{-1'}. \quad (7)$$

When $\text{Cov}(\mathbf{G})$ is scaled by the loading matrix \mathbf{L} and added to the covariance matrix of \mathbf{U} , the expected covariance matrix of $\mathbf{X}^{(5)}$ can be written as

$$\mathcal{E}(\text{Cov}(\mathbf{X}^{(5)})) = \mathbf{L} \text{Cov}(\mathbf{G}) \mathbf{L}' + \text{Cov}(\mathbf{U}). \quad (8)$$

An example OpenMx (Boker et al., 2011) script to fit this second order linear LDE model using full information maximum likelihood is presented in Appendix A.

Individual Differences in Equilibrium

The model in the previous section assumes that each individual’s data is centered around his or her own equilibrium. This is can be reasonable if there is a known population equilibrium and there are no individual differences in this equilibrium value. However, for many psychological variables of interest, there are individual differences in equilibrium values and there is no a priori knowledge to guide us in centering individuals about their equilibria. Consider the data plotted in Figure 4 for two married couples’ self-reported intimacy and disclosure scores over 45 days (Laurenceau, Barrett, & Rovine, 2005). By inspection, each individual exhibits fluctuations about his or her own individual equilibrium for each variable.

One way to account for these individual differences in equilibrium value is to subtract a known or estimated equilibrium value from each individual’s time series. This process is sometimes called *prewhitening* or *detrending* in the time series literature. For instance, each individual’s mean over all occasions could be subtracted. But, it may be that the individual differences in equilibrium value are part of what one wishes to model. In this case it would be useful to be able to simultaneously estimate the equilibrium value and the parameters of the differential equation.

One way that individual differences in equilibrium values can be estimated simultaneously along with the parameters of an LDE is by adding a latent intercept term to the model as shown in Figure 5. This model is a hybrid of an intercept-only latent growth curve (LGC) model and a second order linear LDE and can be fit using full information maximum likelihood (FIML). The means for the data are modeled as a structured LGC while the variances and covariances for the data are modeled as an LDE. This technique essentially forces the LDE model for the dynamics to be fit to the residuals from the LGC means model where each individual can have their own estimated equilibrium value. Thus, we estimate the equilibrium value for each person that simultaneously maximizes the likelihood of the data given the chosen model for the dynamics. This simultaneous estimation is an improvement over separately estimating individual means and centering each person’s data about the mean in a prewhitening step.

In the RAM path diagram notation (McArdle & McDonald, 1984; McArdle & Boker, 1990), variances are always explicitly represented as double-headed arrows from a variable to itself. Note that the small circle near the bottom of Figure 5 has no variance. This small circle is simply a placeholder to denote a matrix operation during estimation and not an actual latent variable. Also note that the arrow pointing from the triangle (constant) to the small circle is labeled as $I|i$, denoting that there are separately estimated intercept means (I) grouped (the vertical bar, $|$) by individual (i). Thus, the individual differences in equilibrium value are subsumed into the individually estimated mean parameters, one for each individual. There is one intercept value for each individual even though there are many rows in $\mathbf{X}^{(5)}$ belonging to that individual. This is quite different than a standard growth curve model where the small circle would be taken to be a latent intercept with a variance term that represented the individual differences. Here, it is the vector of parameters $I|i$ that carry the individual differences variance.

The model in Figure 5 can be written as

$$\mathbf{X}_i^{(D)} = \mathbf{M}_i \mathbf{K} + \mathbf{G} \mathbf{L} + \mathbf{U}_i \quad (9)$$

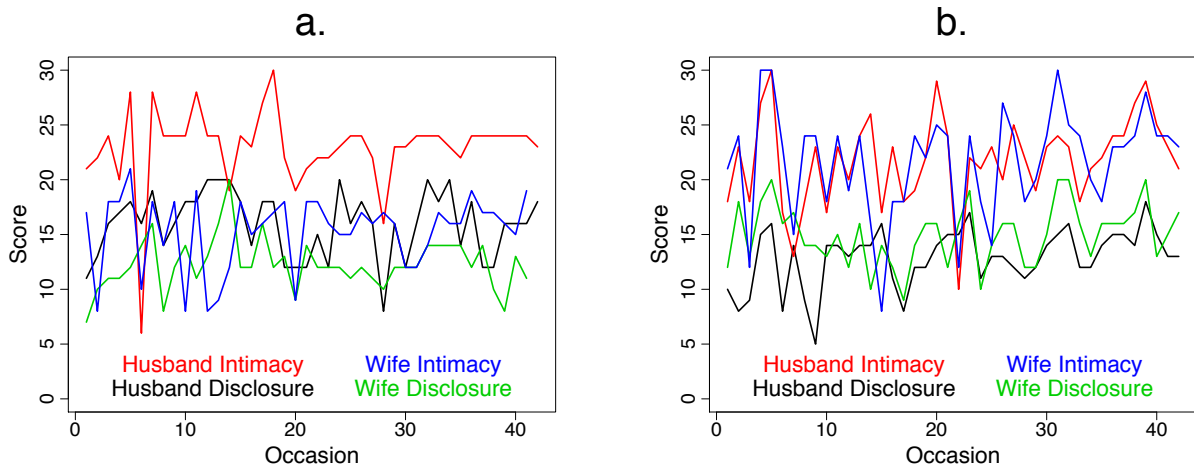


Figure 4. Intimacy and disclosure scores for two married couples (a and b) over 42 days of a daily diary study (data from Laurenceau, Barrett, & Rovine, 2005).

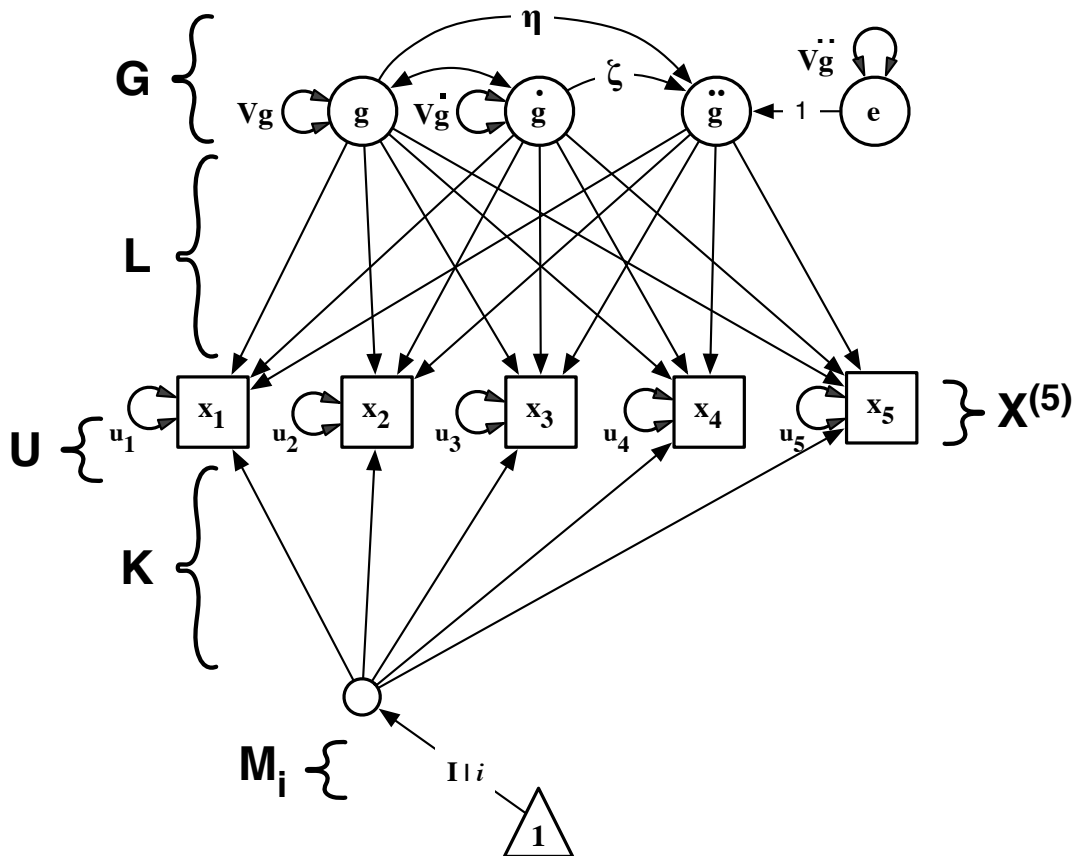


Figure 5. Hybrid linear second order LDE with individual differences in equilibrium intercept estimated by a latent intercept with mean grouped by individual.

where \mathbf{M}_i is the i th row of an $N \times 1$ matrix of means for the N persons in the sample, \mathbf{K} is a $1 \times D$ matrix with a fixed value of 1 in each cell, \mathbf{G} is the latent derivative score matrix, \mathbf{L} is the LDE loading matrix described in the previous section, and \mathbf{U}_i is a matrix of residuals for person i . In the example shown in Figure 5, $D = 5$, since this example uses a time delay embedding data matrix with 5 columns. In general the optimal number of columns for time delay embedding will be dependent on the data and process to be estimated (see, e.g., Hu, Boker, Neale, & Klump, 2014, for techniques for estimating optimal D).

An example OpenMx script implementing this approach is shown in Appendix B and the individual equilibria from this simulation are plotted in Figure 7–c. This script takes advantage of novel features available in OpenMx in order to create a random parameters matrix (called “Rand” in the script). The matrix Rand has a row for each individual in the data set. This allows us to constrain the latent growth curve intercept to be equal within–individual but allows it to differ between individuals. In this way, estimates from the LDE part of the model can affect the latent growth curve part of the model which is grouped by individual.

A simulation was performed in which data were generated to conform to the model in Figure 5 with small variation in parameters between each of 100 replications. The data were generated according to an experimental design where 50 individuals were each measured on 50 occasions. Each individual’s equilibrium value was drawn from a normal distribution with $\sigma = 2.0$. Each replication was then fit using the model script in Appendix B and results are summarized in Table 1 and Figure 6. One data set did not converge and three data sets resulted in parameter estimates that were extreme outliers (> 100 standard deviations from the mean point estimate). These parameter values were clearly impossible given the structure of the data and could be easily excluded as being optimization failures. No attempt was made to adjust starting values and refit for the non-converging and extreme cases, instead these 4 cases were removed from the summary results in Table 1.

Individual Differences in Equilibrium Change

One assumption of the model in the previous section is that there is no change in the equilibrium value during the burst of measurements. In many cases this assumption is reasonable, but in some cases it may not hold. For instance in Bisconti and colleagues’ study of recently bereaved widows (Bisconti et al., 2004), there is good reason to believe that the equilibrium value was likely to be lower at the beginning of the 90 day study than it was at the end. Also, there was evidence of individual differences in this change in equilibrium value. This makes sense from a substantive standpoint since there are so many different possible circumstances for a death. One death may be sudden and come as a great shock to surviving family members. Another death may be after years of protracted illness and pain and survivors may find a sense of relief that a loved

one’s pain is finally over.

Figure 7 presents time series plots of two samples of simulated data. In Figure 7–a there is no change in equilibrium value, consistent with the model from the previous section. But in Figure 7–b there are individual differences in not only the intercept, but also the slope of changing equilibria. Figures 7–c and 7–d show the results of fitting models allowing for individual differences in equilibrium and equilibrium change to the respective simulated data. The four plots in Figure 7 are produced as part of the scripts in Appendix A and Appendix B.

Figure 8 presents a path diagram of a model that can simultaneously account for regulatory fluctuations and longer–term linear changes in equilibrium. The linear second order LDE portion of the model is unchanged from the previous model, but now we add a slope parameter, $S|i$, which is grouped by individual i . That is to say, the mean slope is constrained to be equal within–individual, but allowed to vary across individuals.

The model can be written in a similar form to the previous model in Equation 9,

$$\mathbf{X}_i^{(D)} = \mathbf{M}_i \mathbf{K} + \mathbf{G} \mathbf{L} + \mathbf{U}_i, \quad (10)$$

but now \mathbf{M}_i is the i th row of an $N \times 2$ matrix of means for the N persons in the sample and which contains the individual–specific means for the latent intercept and slope. The matrix \mathbf{K} is a $2 \times D$ fixed matrix of values that allow the estimation of the latent intercept and slope. However, since the time delay embedded matrix $\mathbf{X}_i^{(D)}$ has many rows for the same individual, while the matrix \mathbf{K} is fixed for each row, its values must be calculated anew for each occasion j within each individual i ’s block of data in $\mathbf{X}_i^{(D)}$.

If Δt is the interval of time between successive measurements in the $D = 5$ columns of $\mathbf{X}_i^{(5)}$, then one may pre-specify matrices two fixed value matrices, \mathbf{C} and \mathbf{H} such that

$$\mathbf{C} = \begin{bmatrix} 1 & 1 & 1 & 1 & 1 \\ -2\Delta t & -\Delta t & 0 & \Delta t & 2\Delta t \end{bmatrix} \text{ and} \\ \mathbf{H} = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 \\ \Delta t & \Delta t & \Delta t & \Delta t & \Delta t \end{bmatrix}. \quad (11)$$

For the j th occasion of measurement within individual i ’s data, create a matrix \mathbf{J}

$$\mathbf{J} = \begin{bmatrix} 0 & 0 \\ 0 & j \end{bmatrix}.$$

Now, one may calculate \mathbf{K} as

$$\mathbf{K} = \mathbf{J} \mathbf{H} + \mathbf{C}. \quad (12)$$

These row–specific calculations can be performed within OpenMx using its *definition variable* facility. Thus, if the occasion of measurement for each person is stored as a column augmenting the time–delay embedded matrix, $\mathbf{X}_{ij}^{(5)}$, then the occasion of measurement, j , may be substituted into the matrix \mathbf{J} for each row of a full information maximum likelihood calculation. An example script is provided in Appendix C illustrating the use of individual–specific means and row–specific matrices to estimate this hybrid

Table 1. Simulation results for recovering individual differences in equilibrium value. Data were fit with the model shown in Figure 5 and scripted in Appendix B. The simulation includes 100 replications of an experiment with 50 individuals each measured on 50 equal-interval occasions. Standard deviations (sd) are the standard deviations of the simulated and estimated parameters.

	Simulated	(sd)	Estimated	(sd)
N	100		96	
Did Not Converge			1	
Extreme Outliers			3	
Eta Mean	-0.200	(0.014)	-0.189	(0.016)
Zeta Mean	-0.100	(0.015)	-0.110	(0.026)
Intercept Mean	-0.026	(0.289)	0.004	(0.311)
Intercept Variance	4.081	(0.830)	4.641	(4.411)

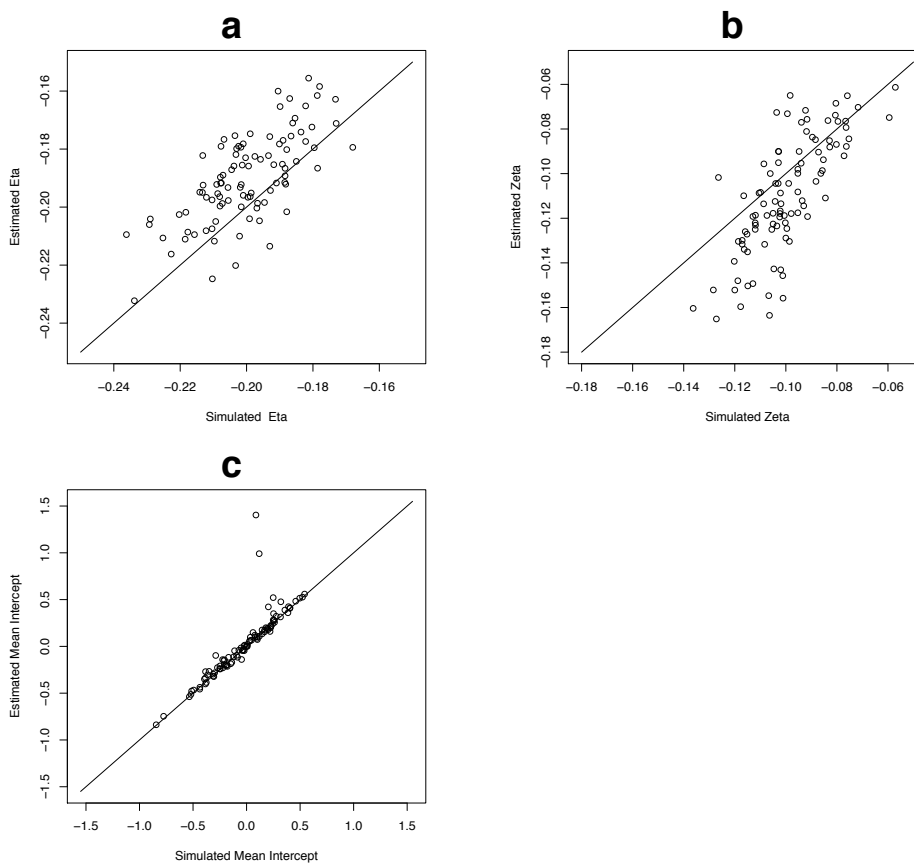


Figure 6. Scatterplots of simulated versus estimated parameters for a) η , b) ζ , and c) intercept for 96 replications of fitting the model shown in Figure 5 using the script in Appendix B.

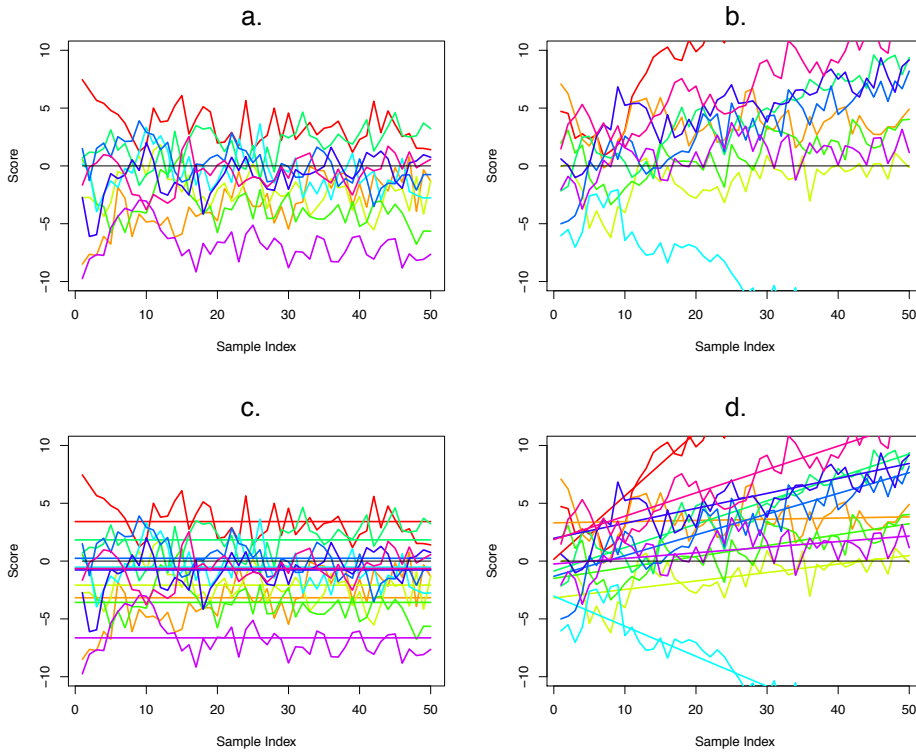


Figure 7. Time series plots of data with (a) individual differences in mean equilibrium and (b) individual differences in both intercept and slope of the equilibrium. Individual differences in equilibria are shown as lines resulting from (c) fitting the model from Equation 9 and (d) fitting the model from Equation 10. LDE parameters are simultaneously estimated from the residuals of these estimated equilibria.

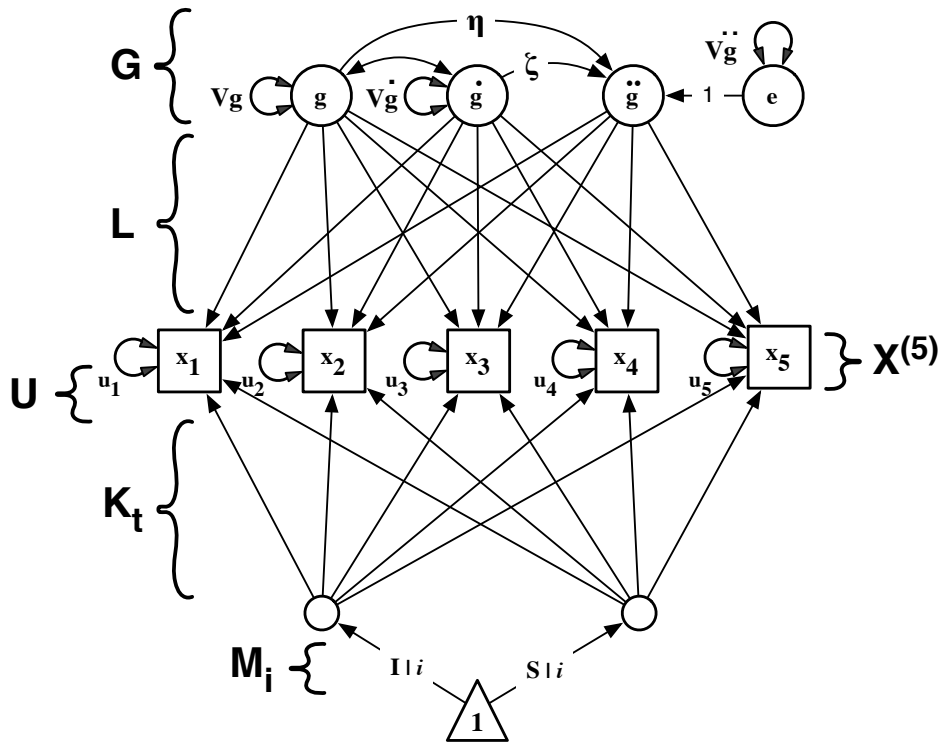


Figure 8. Hybrid linear second order LDE with individual differences in equilibrium intercept and slope estimated by latent intercept and slope terms with means grouped by individual.

LDE and LGC model and individual equilibria for the first 10 individuals from this simulation are plotted in Figure 7–d.

A second simulation was performed in which data were generated to conform to the model in Figure 8 with small variation in parameters between each of 100 replications. The data were once again generated according to an experimental design where 50 individuals were each measured on 50 occasions. Each individual's equilibrium intercept and slope was drawn from a normal distribution with intercept $\sigma = 2.0$ and slope $\sigma = 0.3$. Each replication was then fit using the model script in Appendix C and results are summarized in Table 2 and Figure 9. Five data sets did not converge and two data sets resulted in parameter estimates that were extreme outliers. Again, the non-converging and extreme outlier cases were removed from the summary results.

Individual Differences in Dynamics

The models presented in the three previous sections do not take into account the possibility that individuals may differ from one another in the way they self-regulate, that is to say there may be individual differences in the shapes of the systems' basins of attraction. In many regulatory systems it may be expected that there will be measurable individual differences in the parameters of models of regulation and that these individual differences would be substantively important. For instance, variables such as resiliency in older adults (Montpetit, Bergeman, Deboeck, Tiberio, & Boker, 2010), disclosure and intimacy in married couples (Laurenceau, Rivera, Schaffer, & Pietromonaco, 2004), and hormone cycles and disordered eating in young women (Edler et al., 2007) all have shown evidence of substantively important individual differences in the parameters of dynamical models.

Figure 10 presents nine examples of how different trajectories can result from parameter differences in a second order linear differential equation. In each of these time series plots, the same equation is used and only the parameters and starting values are changed. These plots illustrate how seemingly different behavioral trajectories may result from a single underlying differential equation process. Figures 10–a, 10–b, and 10–c are oscillating systems with damping. That is to say oscillations tend to die out over time if they are not perturbed by external events. Figures 10–d, 10–e, and 10–f are oscillating systems with amplification; systems that tend to amplify perturbations from exogenous sources. Finally, Figures 10–g, 10–h are overdamped, and 10–i is nearly overdamped. These systems show trajectories that seem very similar to a first order linear system with exponential decay.

We can make an adjustment to the model from the previous section in order to estimate individuals' parameters of the LDE as shown in the path diagram in Figure 11. This adjustment requires individual level subscripts on the LDE

model equations which can be written,

$$\ddot{g}_{ij} = \eta_i g_{ij} + \zeta_i \dot{g}_{ij} + e_{ij} \quad (13)$$

$$\mathbf{X}_{ij}^{(5)} = \mathbf{M}_i \mathbf{K} + \mathbf{G}_i \mathbf{L} + \mathbf{U}. \quad (14)$$

This model can be implemented in OpenMx using the same mechanism that we used to group the intercepts and slopes by individual. We add two new columns to the random parameters matrix and use the individual ID as an index into the random parameters matrix in order to constrain η_i and ζ_i within-individual while allowing individual differences in these parameters. An OpenMx script implementation is shown in Appendix D.

A third simulation was performed in which data were generated to conform to the model in Figure 11 where each individual's parameters of the second order differential equation were drawn from a normal distribution with means and variances shown in Table 3. The data were again generated according to an experimental design where 50 individuals were each measured on 50 occasions. Each individual's equilibrium intercept and slope was drawn from a normal distribution with intercept $\sigma = 2.0$ and slope $\sigma = 0.3$. Each replication was then fit using the model script in Appendix D and results are summarized in Table 3 and Figure 12. All data sets converged and one data set resulted in parameter estimates that were extreme outliers. Again, the non-converging and extreme outlier cases were removed from the summary results.

Individual Differences in Variability

Another important and often studied source of individual differences is variability. Variability is sometimes operationalized as the within-person variance (or standard deviation) of repeated observations of a variable. However, this definition misses an important distinction: the difference between the overall amplitude of the displacement from equilibrium and the amplitude of the first derivatives (Deboeck et al., 2009). This distinction is illustrated in Figure 13.

Both of these types of variability may be estimated by allowing individual differences in the variances of the displacement and first derivatives. Thus, the path model from the previous section can be relaxed further as shown in Figure 14 where $Vg|i$ and $V\dot{g}|i$ are the variances of the displacement from equilibrium and first derivatives respectively grouped by individual i . This model can be implemented in OpenMx in the same manner as was used in modeling individual differences in the parameters of the differential equation. While we do not show this in an Appendix, a script implementing this is included in the downloadable archive file.

Second Level Predictors

When there are individual differences in parameters of a dynamical systems model, it may be of interest to predict

Table 2. Simulation results for recovering individual differences in equilibrium value and equilibrium change. Data were fit with the model shown in Figure 8 and scripted in Appendix C. The simulation includes 100 replications of an experiment with 50 individuals each measured on 50 equal-interval occasions. Standard deviations (sd) are the standard deviations of the simulated and estimated parameters.

	Simulated	(sd)	Estimated	(sd)
N	100		93	
Did Not Converge			5	
Extreme Outliers			2	
Eta Mean	-0.200	(0.015)	-0.203	(0.016)
Zeta Mean	-0.099	(0.014)	-0.110	(0.022)
Intercept Mean	0.021	(0.272)	0.044	(0.332)
Intercept Variance	3.992	(0.967)	5.492	(7.946)
Slope Mean	0.002	(0.037)	0.003	(0.037)
Slope Variance	0.087	(0.017)	0.086	(0.017)

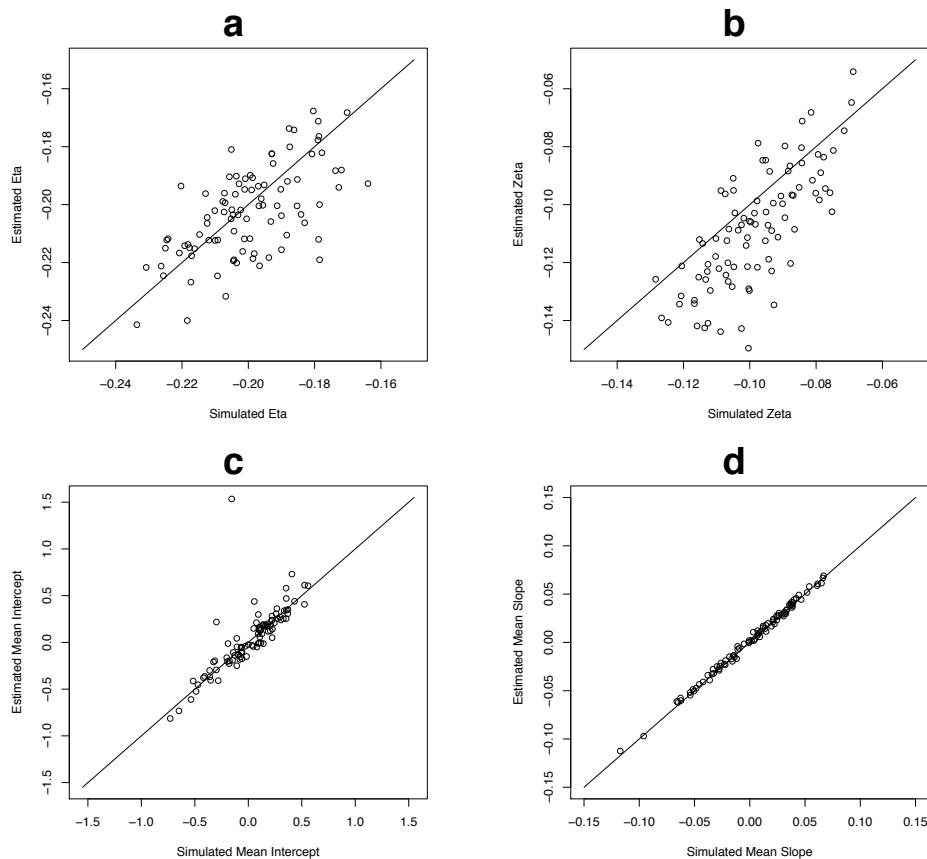


Figure 9. Scatterplots of simulated versus estimated parameters for a) η , b) ζ , c) intercept, and d) slope for 93 replications of fitting the model shown in Figure 8 using the script in Appendix C.

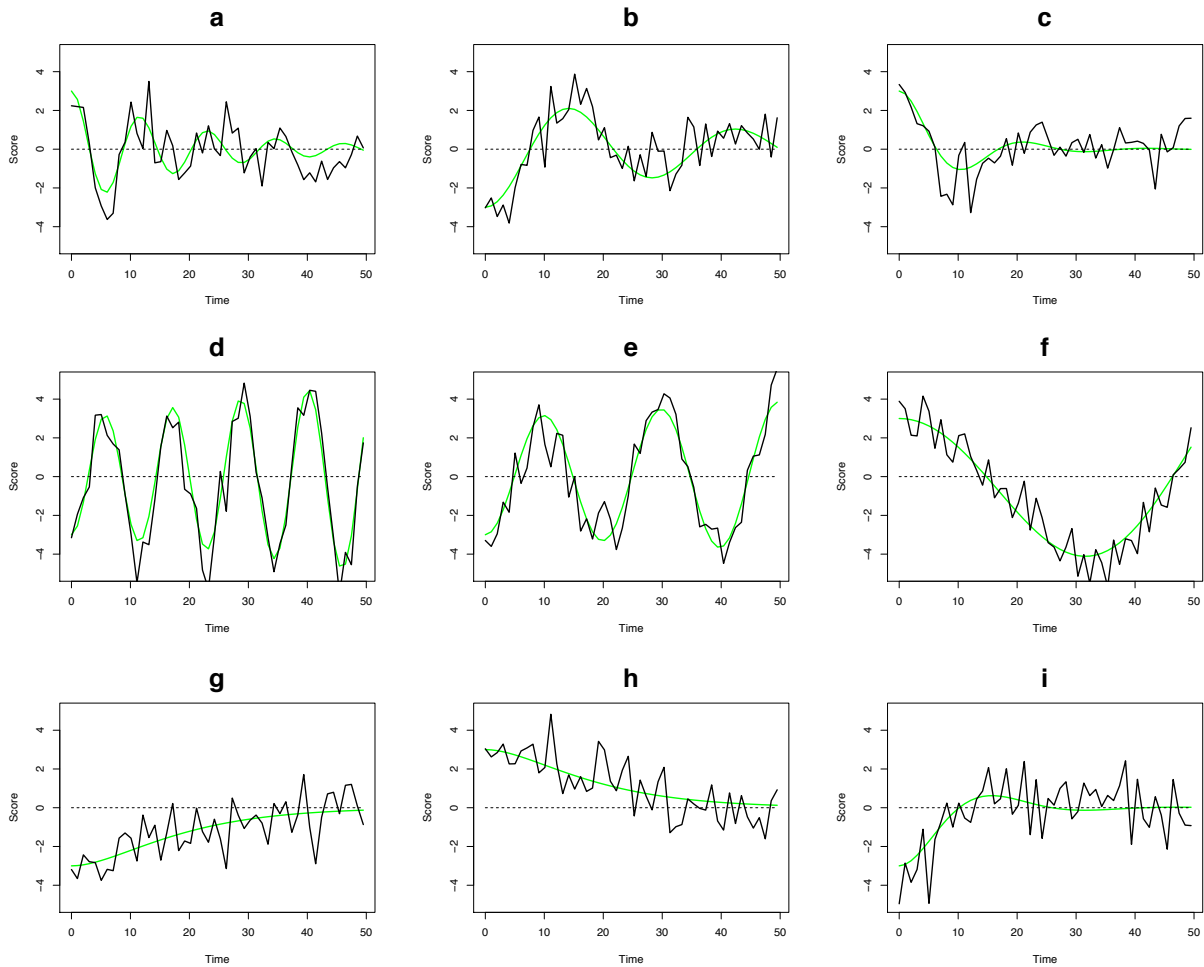


Figure 10. Nine time series resulting from the equation $\ddot{g}_j = \eta g_j + \zeta \dot{g}_j + e_j$. Smooth lines are the underlying dynamics ($V_u = 0$) and noisy lines include time-independent additive error ($V_u = 1$). (a) $\eta = -0.3, \zeta = -0.1$ (b) $\eta = -0.05, \zeta = -0.05$ (c) $\eta = -0.1, \zeta = -0.2$ (d) $\eta = -0.3, \zeta = 0.02$ (e) $\eta = -0.1, \zeta = 0.1$ (f) $\eta = -0.01, \zeta = 0.02$ (g) $\eta = -0.01, \zeta = -0.2$ (h) $\eta = -0.01, \zeta = -0.1$ (i) $\eta = -0.05, \zeta = -0.2$

Table 3. Simulation results for recovering individual differences in dynamics of a second order linear differential equation with individual differences in equilibrium value and equilibrium change. Data were fit with the model shown in Figure 11 and scripted in Appendix D. The simulation includes 100 replications of an experiment with 50 individuals each measured on 50 equal-interval occasions. Standard deviations (sd) are the standard deviations of the simulated and estimated parameters.

	Simulated	(sd)	Estimated	(sd)
N	100		99	
Did Not Converge			0	
Extreme Outliers			1	
Eta Mean	-0.201	(0.013)	-0.231	(0.015)
Eta Variance	0.010	(0.002)	0.016	(0.004)
Zeta Mean	-0.099	(0.014)	-0.119	(0.025)
Zeta Variance	0.010	(0.002)	0.034	(0.014)
Intercept Mean	0.037	(0.281)	0.042	(0.312)
Intercept Variance	3.845	(0.787)	4.863	(3.417)
Slope Mean	-0.007	(0.044)	-0.005	(0.044)
Slope Variance	0.088	(0.018)	0.088	(0.017)

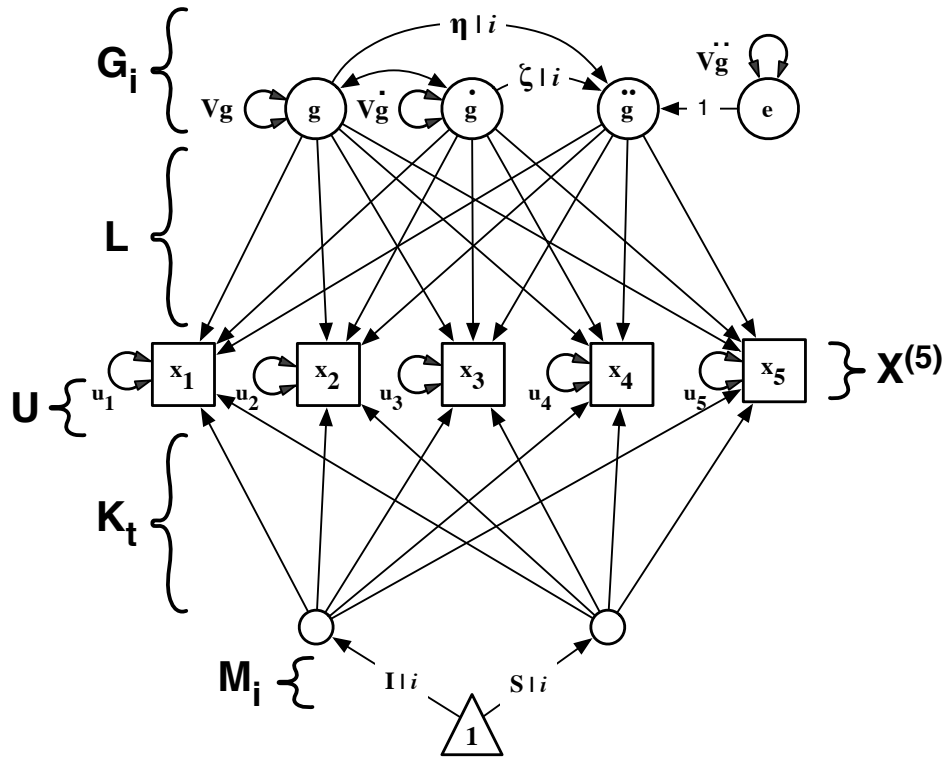


Figure 11. Path diagram of a hybrid second order linear LDE and LGC with individual differences in equilibrium level, equilibrium change and dynamic parameters.

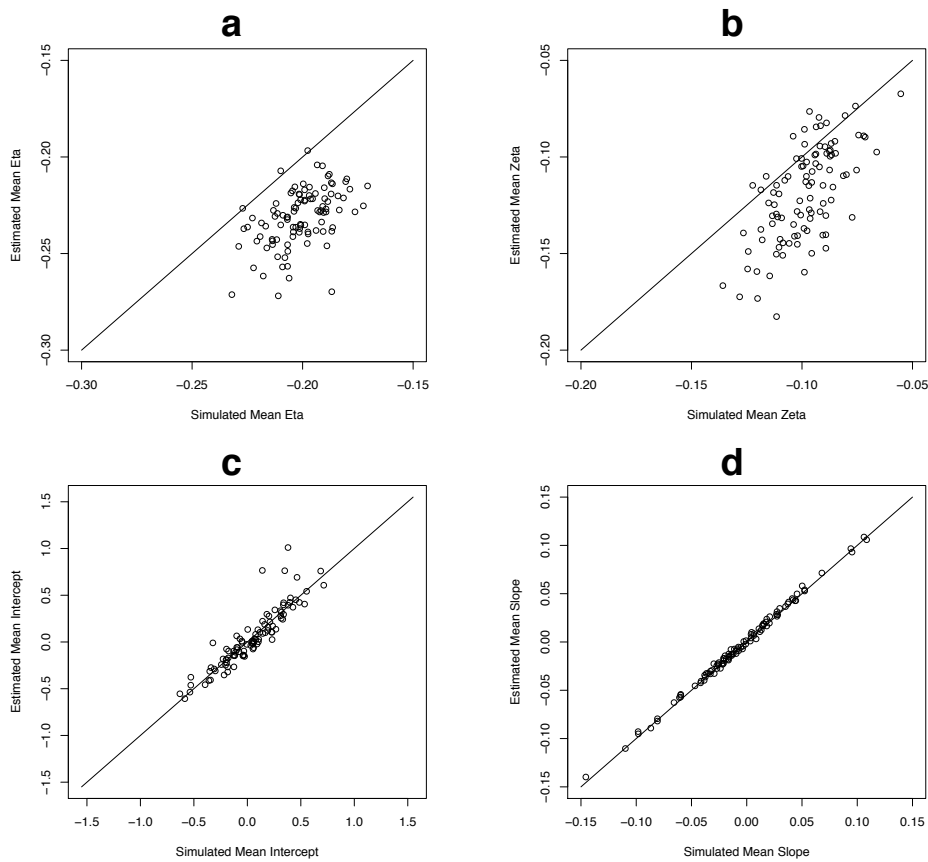


Figure 12. Scatterplots of simulated versus estimated parameters for a) η , b) ζ , c) intercept, and d) slope for 93 replications of fitting the model shown in Figure 11 using the script in Appendix D.

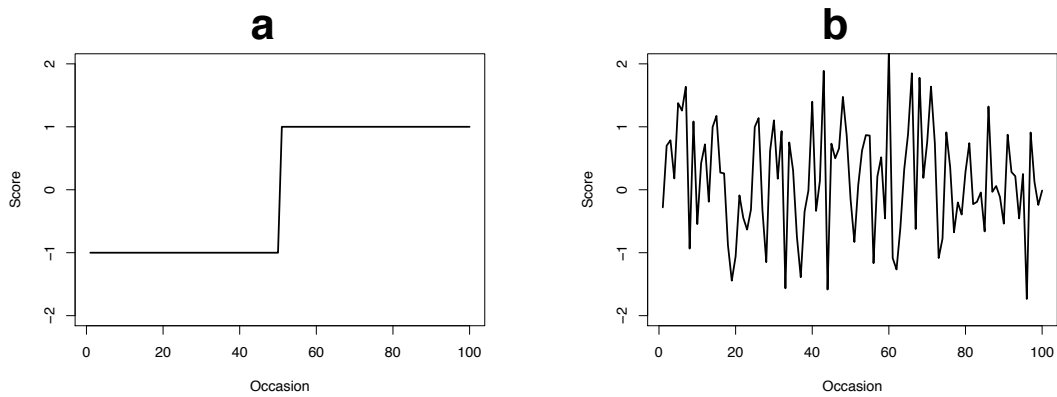


Figure 13. Illustration of the difference between standard deviation of the displacement and standard deviation of the first derivative. The time series in a) and in b) each have 100 observations and standard deviation of the displacement $\sigma = 1.0$. However, the standard deviation of the first derivative is much smaller in a), $\sigma = 0.14$, than it is in b), $\sigma = 0.74$.

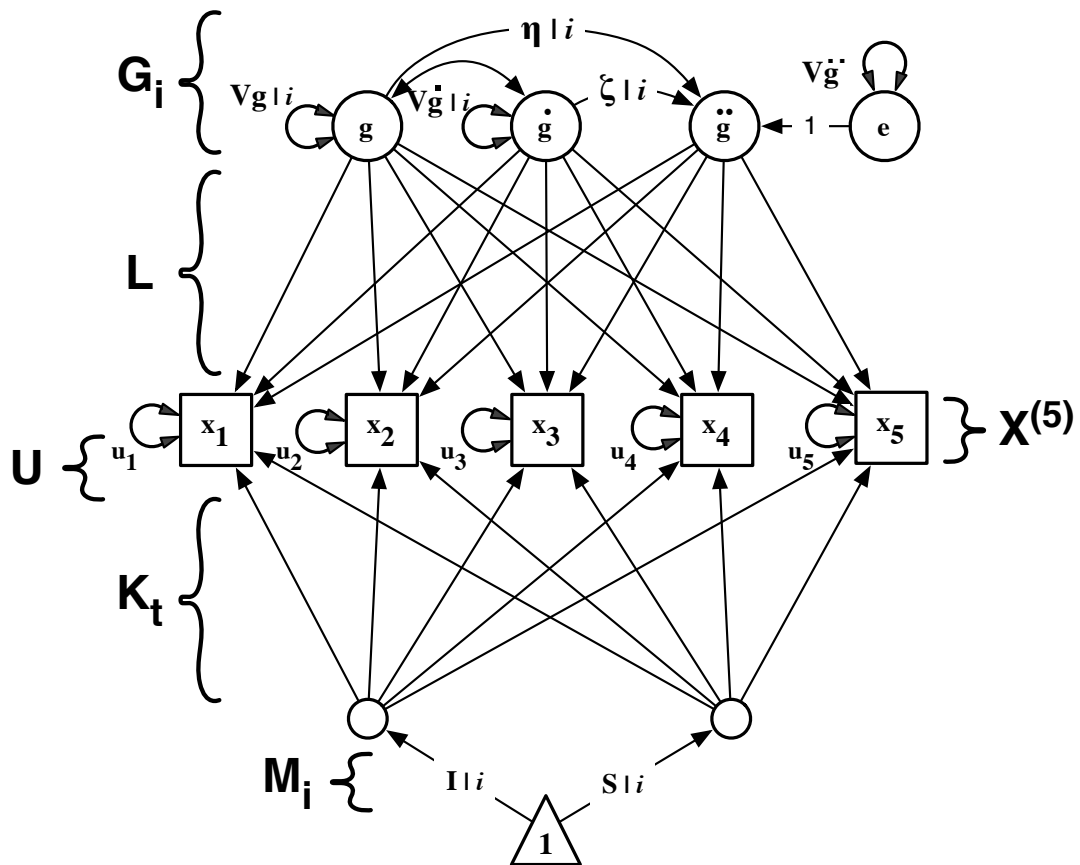


Figure 14. Path diagram of a hybrid second order linear LDE and LGC with individual differences in equilibrium level, equilibrium change, dynamic parameters, and amplitude of fluctuations.

these differences from other measured characteristics or traits of the individual. For instance, fluctuations in self-perceived mental health in recently bereaved widows were damped more quickly in widows who reported a high level of social support from their family members, while widows who cycled at a faster rate were reported by family members to have lower levels of perceived control (Bisconti et al., 2006). Individual differences in equilibrium level and slope may also be predictable from other individual characteristics (Boker, 2013) and could be modeled in the same manner as illustrated here.

$$\begin{aligned}\ddot{g}_{ij} &= \eta_i g_{ij} + \zeta_i \dot{g}_{ij} + e_{ij} \\ \eta_i &= \eta + \eta_z z_i \\ \zeta_i &= \zeta + \zeta_z z_i\end{aligned}\quad (15)$$

$$\mathbf{X}_i^{(5)} = \mathbf{M}_i + \mathbf{GL} + \mathbf{U}_i \quad (16)$$

Note that the subscript i is no longer present on the latent structure matrix, \mathbf{G} . This model can be fit using a two column indexed parameter matrix containing only the individual-level means and slopes. The assumption that we make is that the interindividual differences in the latent differential equation structure is characterized by the between-persons variance in the individual characteristic variable z_i . Positive consequences of this assumption are that the model can estimate the effect of z on the parameters of the dynamic (as shown in Equation 15) and that the model can be estimated more quickly than one with individual-level parameters. The approach shown in Appendix E can be used to estimate the mean effects and associated individual second-level effects.

A fourth simulation was performed in which data were generated to conform to the model in Figure 15 where each individual's parameters of the second order differential equation were predicted by a trait-level variable z drawn from a normal distribution with mean $\mu = 0.0$ and standard deviation $\sigma = 1.0$. The data were again generated according to an experimental design where 50 individuals were each measured on 50 occasions. Each individual's equilibrium intercept and slope was drawn from a normal distribution with intercept $\sigma = 2.0$ and slope $\sigma = 0.3$. Each replication was then fit using the model script in Appendix D and results are summarized in Table 3 and Figure 12. All replications converged and there were no extreme outliers.

Longitudinal Change in Dynamics

Over longer periods of time, the regulatory dynamics exhibited by an individual may change. Examples of substantively interesting changes in dynamics include developmental mechanisms, changes in an individual's context, or changes due to learning and/or plasticity. Long-term changes in dynamics can be estimated using data from a

multiple burst design. For instance, the Notre Dame Study of Health and Well-Being (Ong et al., 2009) includes three 52-day bursts of daily self-report, where bursts are separated by an interval of two years.

Figure 17 plots simulated data for four individuals for a 3-wave burst design. In these simulated data, equilibrium intercepts and slopes as well as the parameters of regulatory dynamics change across waves. One way to model this change is to construct a variable that represents the temporal interval in such a way that individuals are temporally aligned on that variable. For instance, for younger individuals, age in years might be an appropriate variable. In samples of elders when mortality data are available, another time-aligning variable that has been used is years to mortality (e.g., Gerstorf, Ram, Röcke, Lindenberger, & Smith, 2008). Or, in some cases it might make sense to align all individuals by burst, such as in an intervention study where the first burst might be pre-intervention with a second burst after intervention and a third burst as a long term followup. In each of these situations, the constructed time-aligned variable can be substituted into the model in Figure 15 as the variable z , allowing the estimation of between-persons time-dependent changes in dynamics that can be detected when the bursts are time-aligned.

Given that this simulated example has only three bursts, the most complex change in dynamics we can estimate is linear change. When there are more than three bursts of data, more sophisticated questions could be posed about the evolving nature of within-person change in dynamics. If there is a theoretic reason for a particular form of nonlinear change (e.g., negative exponential growth to an asymptote as might be postulated in a training study) then the variable z can be constructed so that the theoretic nonlinear basis is built into its values. In that case, the model in Figure 15 can be used to estimate a nonlinear change in dynamics.

Summary

The article presented structural equation models for simultaneously modeling equilibria and the dynamics governing regulation about the equilibria. We used a modified latent growth curve approach to create a structured means model that estimates the level and linear change in the equilibrium. The covariances of the residuals from this structured means model were in turn modeled as a latent differential equation. By combining these two approaches we can obtain estimates for the parameters describing the equilibrium and the dynamics that simultaneously maximize the likelihood of the observed data. This is an improvement over methods that first subtract means and/or trends from data and then later estimate models for the dynamics of these residuals.

We presented a framework for thinking about and developing models of individual differences in equilibria, dynamics, and intraindividual variability. These models started with a restricted model that assumed no individual differences and relaxed this assumption in a structured way: first

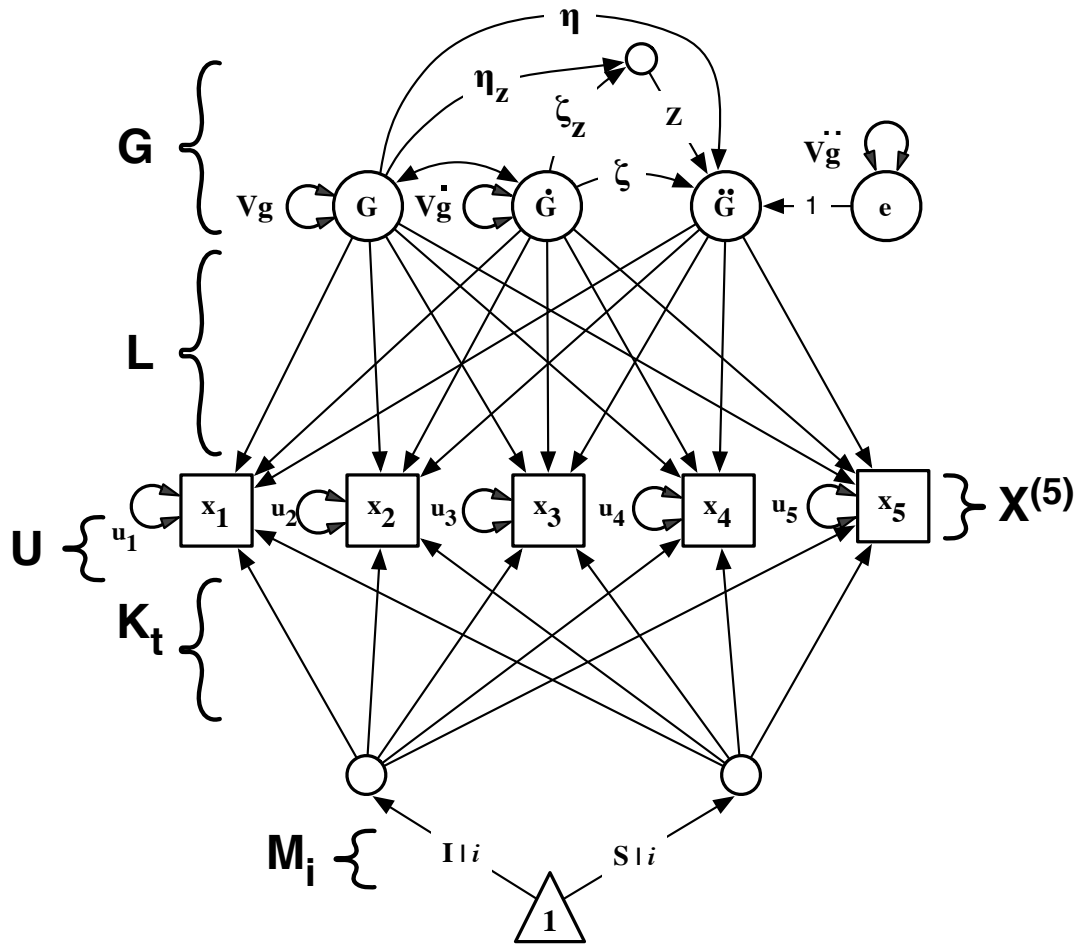


Figure 15. Path diagram of a hybrid second order linear LDE and LGC with individual differences in equilibrium level, equilibrium change and a second level predictor of η and ζ .

Table 4. Simulation results for recovering second level effects on parameters of dynamics of a second order linear differential equation with individual differences in equilibrium value and equilibrium change. Data were fit with the model shown in Figure 15 and scripted in Appendix E. The simulation includes 100 replications of an experiment with 50 individuals each measured on 50 equal-interval occasions. Standard deviations (sd) are the standard deviations of the simulated and estimated parameters.

	Simulated	(sd)	Estimated	(sd)
N	100		100	
Did Not Converge			0	
Extreme Outliers			0	
Eta Mean	-0.200	(0.000)	-0.198	(0.008)
Zeta Mean	-0.100	(0.000)	-0.119	(0.014)
Eta Interaction	0.100	(0.000)	0.098	(0.016)
Zeta Interaction	0.100	(0.000)	0.108	(0.020)
Intercept Mean	-0.027	(0.225)	-0.073	(0.238)
Intercept Variance	3.892	(0.779)	4.089	(0.787)
Slope Mean	-0.004	(0.034)	-0.002	(0.034)
Slope Variance	0.088	(0.018)	0.087	(0.017)

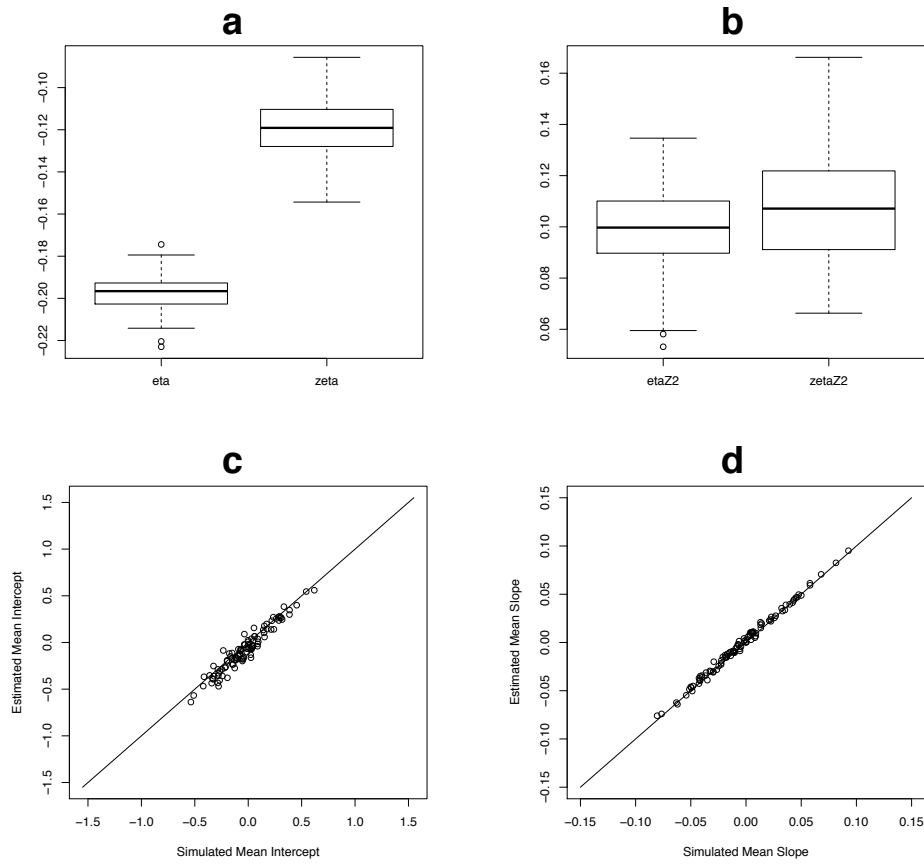


Figure 16. Boxplots and scatterplots of simulated versus estimated parameters for a) η and ζ , b) the interaction between z and η and ζ , c) intercept, and d) slope for 100 replications of fitting the model shown in Figure 15 using the script in Appendix E.

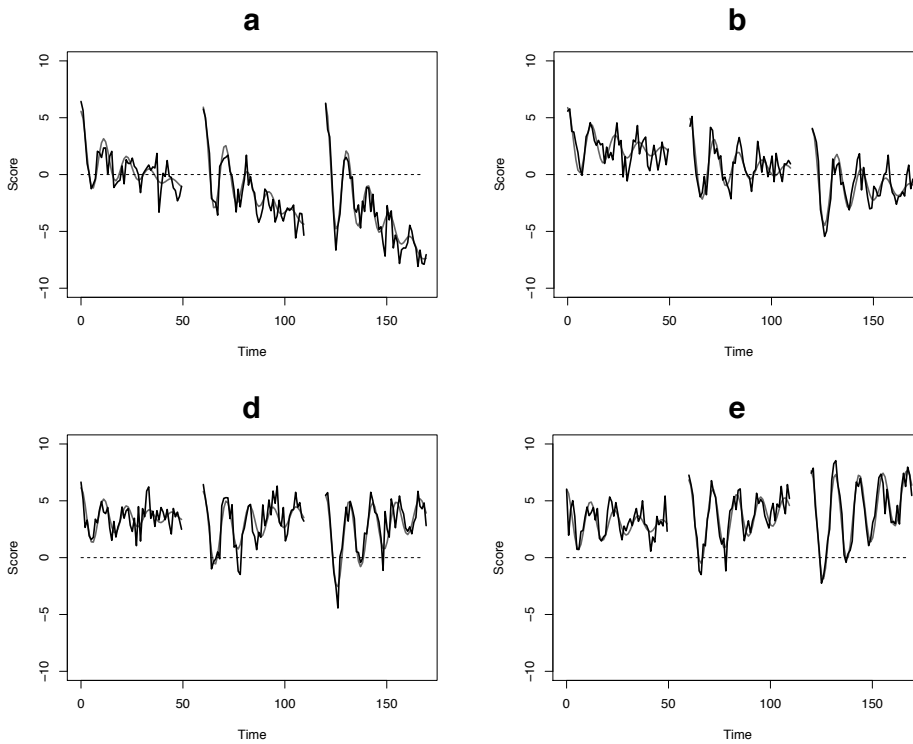


Figure 17. Multiple bursts with longitudinal change in dynamics in both equilibrium intercept and slope.

allowing individual differences in the intercept and slope of the equilibrium; next allowing individual differences in the parameters of the system dynamics; next allowing individual differences in the variability of the displacement and first derivative; and finally adding a second level interaction term to estimate regression relationships between trait level variables and parameters of dynamics. Finally we proposed an experimental design and model that could estimate change in dynamics simultaneously with change in equilibrium over time.

Overall, the estimates of individual differences in intercept and slope were exceptionally good for all models presented here, with the exception of a few extreme outliers. While the dynamic parameters showed more variability with respect to their originally simulated values, the estimates were reasonable given the relatively short (50 occasions) time series for each individual and relatively low simulated signal to noise ratio of 1/1. Data with higher reliability and/or more observations per individual would have improved these parameter estimates. Estimates of damping showed ($\approx 10\%$) bias towards stronger damping (ζ) than was simulated. The only model that showed appreciable ($\approx 10\%$) bias in frequency (η) was the individual differences in dynamics model from Appendix D.

All of the model scripts and data simulation scripts described in the article are available for free download from the corresponding author's web site: <http://people.virginia.edu/~smb3u/ChangesInDynamics.zip>. We encourage interested readers to modify the simulation scripts to apply to their own experimental design and assure themselves that the results that they obtain from fitting these models provide tolerable estimates. The simulation scripts may also be useful in calculating power prior to beginning an intensive longitudinal experiment. In particular, we recommend power estimates be obtained for longer bursts and fewer individuals relative to shorter bursts and more individuals. The particulars of an experimental question will determine in which of these two directions the data design is best steered.

Limitations

In order to estimate derivatives for the second order differential equation models presented here, one will need on the order of a minimum of 400 observations. These observations could be configured in a variety of ways, e.g., as 20 individuals with 20 observations each or as 10 individuals with 40 observations each. As with any simplistic rule-of-thumb such as suggested here, actual power will be dependent on the characteristics of the data: the reliability of the within-person measurements (Hu et al., *in press*); the degree of within-person equilibrium change over time; and the degree of between-person differences. The more one wishes to know about the person-oriented variables, the more data one will want from each person. At the extreme, when one disregards between-person differences and can only afford P observations, more power is always obtained from more observations from fewer individuals,

and so one is left with the maximum power when all P observations are from a single individual. If one is willing to ignore within-person change in dynamics and is only interested in between-person differences, maximum power is obtained when one has the minimum number of observations per person that have degrees of freedom to estimate the derivatives and residuals of a single fluctuation. Assuming an *optimal sampling interval* that is 5 observations per person, so maximum power would be obtained with 5 observations sampled from $P/5$ individuals. However, one is unlikely to know the optimum sampling interval, since that depends on each individual's dynamics. Thus one will wish to oversample at the individual level. We have often recommended a minimum of 30 observations sampled at a shorter interval than required by a hypothesized fluctuation. As an example, suppose a hypothesis of a weekly cycle. The optimum might be 5 observations per week. But in order to also cover the possibility of daily and also monthly cycles, one might consider sampling 5 times a day for a month: 150 observations per person. There are two downsides to such intensive sampling: participant fatigue and less power to detect between-person differences. But the gain is substantial — If there are changes in daily dynamics that play out over the course of a month, the within-person model has sufficient data to detect them.

The framework presented here distinguishes between some useful types of regulatory dynamics, but is by no means comprehensive. Important sources of individual differences in behavioral and physiological regulation that were not addressed include some types of nonstationary systems. For instance, some regulatory mechanisms may exhibit nonstationary change in dynamics that occur within the same time scale as the regulatory dynamics of change. The proposed modeling framework cannot capture these multiple co-occurring sources of nonstationarity. We believe that the current framework can be extended to estimate such sources of nonstationarity, but identification conditions for such models are not yet well established.

A second type of potentially important systems is one where the dynamics of change are occurring at multiple time-scales. This type of system might have faster regulation embedded within slower regulatory systems. For instance, while there is evidence that affect has one dynamic that results in fluctuations with an approximately 6 to 7 day cycle (e.g., Chow et al., 2005), it is self-evident that emotional and affective states can change quickly — within minutes. Estimating models that could test hypotheses of how multiple timescales of affective regulation interrelate would require a framework that would be able to accommodate multi-timescale delay embedding. This is an active area of inquiry and much work needs to be accomplished before such models can be reliably brought into a framework of change in dynamics and dynamics of change.

Some of the modeling procedures outlined in this article, in particular the indexed parameter matrix method, require free parameters for each individual in the data set. While this type of model is tractable for smaller data sets, a few hundred individuals, the estimation time for these models grows approximately with the cube of the number of indi-

viduals. Thus, numerical fitting procedures for this type of model quickly become intractable as the number of individuals becomes large. Methods need to be developed that overcome this computational limitation without losing the flexibility of the current implementation.

Conclusions

We have presented a framework to help organize development of models for the dynamics of change exhibited by regulatory systems while taking into account some plausible sources of change in dynamics. Change in dynamics was partitioned into categories that include change in equilibria, change in basins of attraction, and change in variance. These types of change were modeled in a framework in which the change in dynamics can be estimated as an individual differences characteristic, a within-individual adaptation, or both. A method was proposed for modeling dynamics of change and change in dynamics from multiple burst designs. While the presentation remained within the context of simulated data, we expect these methods to be immediately applicable to an increasing number of studies as it becomes more widely recognized that individual-level dynamics and adaptation provide important understanding of human behavior, physiology, and development over the lifespan.

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Appendices A-E

Please refer to the included R script files `ChangesInDynamics_Appendix[A-E].R`, `GLLAfunctions.R`, and the data files `ExampleDataAppendix[A-E].R`.

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