Psychophysiology, 38 (2001), 951–960. Cambridge University Press. Printed in the USA.

Piecewise latent growth curve modeling of systolic blood pressure reactivity and recovery from the cold pressor test

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Abstract

Latent growth curve methodology was used to model systolic blood pressure reactivity and recovery from the cold pressor test. A piecewise regression approach permitted the separate but simultaneous modeling of the two components (reactivity and recovery) of the stress process. Data came from a study of 99 participants classified on the basis of gender, ethnicity, and family history of hypertension. Their systolic blood pressure was assessed at rest, during the cold pressor test, and during a task recovery period. A measure of task appraisal and readings from ambulatory blood pressure monitoring during a workday were also examined. The article illustrates a step-by-step approach to modeling reactivity and recovery. Results indicated that both reactivity and recovery were associated with subsequent systolic blood pressure at work.

Descriptors: Cardiovascular reactivity, Recovery, Latent growth modeling, Piecewise regression, Cold pressor, Blood pressure

Recent advances in applications of structural equation modeling to the measurement of change over time may be used to reframe the quantification of reactivity and recovery from stress. Specifically, latent growth curve (LGC) modeling provides a tool for addressing multiple issues raised in the reactivity literature but to our knowledge, has not been applied in such a context. Our objective is to illustrate the potential of LGC methodology by modeling systolic blood pressure (SBP) reactivity and recovery from the cold pressor test in the context of a complex model of disease risk. In doing so, important questions about predictors of reactivity and recovery, about the similarity or uniqueness of these constructs, and about their potential for predicting disease risk may be addressed.

Sympathetically mediated hyperreactivity to stressors is postulated to be a marker for subsequent hypertension (Manuck & Krantz, 1986). Accumulating evidence also suggests that poststress recovery may predict subsequent hypertension (Borghi, Costa, Boschi, Mussi, & Ambrosioni, 1986). Like hyperreactivity, prolonged recovery may indicate altered cardiovascular functioning (Gerin & Pickering, 1995; Hocking Schuler & O'Brien, 1997) and may provide additional information on the role of behavioral factors in the development of hypertension (Gerin & Pickering, 1995; Gerin, Pieper, & Pickering, 1994). The primary value of estimating parameters of reactivity and recovery lies in their potential for clarifying the underlying mechanisms of disease and their utility in predicting subsequent measures of disease risk or disease itself. It is also important to identify individual characteristics such as demographic, health, or situational variables that contribute to the specific pattern of reactivity and recovery exhibited by an individual. Likewise, it is useful to compare different populations exhibiting different risk profiles for disease. In this manner, comprehensive models of disease risk may be examined that more accurately reflect the role of reactivity and recovery in the stress–disease relation.

Past research has relied on the simple change score or delta as the common way of quantifying reactivity primarily because of its simplicity (Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991), although concerns about reliability have been raised. There seems to be less agreement as to the most appropriate way to quantify recovery. Several alternatives have been proposed and used by researchers, including calculating delta from baseline, delta from task, or the time it takes to reach some criteria of recovery (Haynes, Gannon, Orimoto, O'Brien, & Brandt, 1991). Baseline and task levels have not been controlled in a consistent manner. And although recovery studies make repeated assessments that are linked to time, many operational definitions have used arbitrary times or ignored time altogether. Any quantification method of recovery that does not take time into account is missing critical information.

Conventional analytic methods present limitations when studying reactivity and recovery in the context of more complex models. Such methods do not correct for measurement error, lose information by averaging or using arbitrary times, and examine only one parameter at a time. LGC modeling provides a means for attending

This research was supported by National Institutes of Health Grants HL36588 and HL04726.

The authors wish to acknowledge our colleagues Jean-Phillip Laurenceau and L. Allison Jones for their feedback on the manuscript.

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to measurement error, incorporating appropriate controls for baseline and task, and using all of the available information in one comprehensive analysis. In using this model, improved relations with external criteria may be detected.

Growth modeling is applicable to data where individuals are measured repeatedly over a period of time. The questions of interest focus on the trajectory or pattern of the change (or growth). Parameters of the trajectory, such as the slope, can then be related to other participant characteristics. In growth models there are two levels of analysis. At Level 1, the unit of analysis is the repeated observations within a subject. The Level 2 units are the participants themselves. In LGC methodology, a structural equation modeling (SEM) approach is used to estimate the parameters of the growth model. This approach capitalizes on the measurement model aspect of SEM to specify the Level 1 model, and on the structural model aspect of SEM to specify the Level 2 models. Growth models may also be analyzed within the context of hierarchical or multilevel models (e.g., Bryk & Raudenbush, 1992; Goldstein, 1995), or mixed linear models (Jenrich & Sluchter, 1986; Laird & Ware, 1982; Lindstrom & Bates, 1988; Rao, 1958) as is done in PROC MIXED in SAS (Littell, Milliken, Stroup, & Wolfinger, 1996). Each approach has certain advantages and limitations. One key distinction is that the values for the time variable are part of the data set when using a multilevel program such as HLM (Bryk & Raudenbush, 1992) or when using PROC MIXED in SAS. Thus the values of time need not be constant across participants. However, the time values become the fixed loadings in the measurement model for the latent slope when analyzing the data using SEM software such as LISREL (Joreskog & Sorbom, 1993). Therefore, time of measurement must be constant across participants when using many SEM programs for latent growth modeling. This may be a limitation of the SEM approach for situations where the design of the study does not permit manipulation of the time variable. The problem may be circumvented with software such as Mplus (Muthen & Muthen, 1998) with missing data capabilities. On the other hand, the SEM framework allows the testing of structural models where the latent growth parameters may be embedded in more complex structures such as mediation models. Thus with the SEM approach, the latent growth parameters may function as predictors, outcomes, or both. [For a more comprehensive explanation of the SEM approach see Willett and Sayer (1994) and earlier work by Meredith and Tisak (1990). Numerous applications and extensions have been published by McArdle and colleagues and Muthen (e.g., McArdle & Epstein, 1987; Muthen, 1997, 1998). Duncan & Duncan (1995) provide a nice introduction.]

Our application of LGC modeling used cold pressor test data from a study of blood pressure (BP) regulation in adult men and women (see Saab et al., 1997). We limited our focus to the cold pressor test and, therefore, describe only certain aspects of the protocol. We first modeled the reactivity and recovery periods separately, then combined them using a piecewise regression approach. We then tested a model using gender, ethnicity, family history of hypertension, and task appraisal as predictors of SBP baseline, reactivity and recovery, and subsequent work SBP as a marker of cardiovascular disease risk. We included work ambulatory BP as a marker of risk, because ambulatory BP levels have been related to several cardiovascular outcomes, including left ventricular hypertrophy (Devereux et al., 1983; Prisant & Carr, 1990), measures of target organ damage (Parati, Pomidossi, Albini, Malaspina, & Mancia, 1987), and risk of future morbidity and mortality (Mann, Millar, & Raftery, 1985; Perloff, Sokolow, & Cowan, 1983).

The cold pressor test has been used in cardiovascular research for over 60 years (e.g., Hines & Brown, 1936). Our decision to examine SBP reactivity to the cold pressor test was based on evidence supporting a prospective relationship with the development of hypertension (Menkes et al., 1989). Research in the past 15 years has shown that responses to the cold pressor test are useful in detecting differences in cardiovascular reactivity between Black and White Americans (Anderson, Lane, Muranaka, Williams, & Houseworth, 1988; McAdoo, Weinberger, Miller, Fineberg, & Grim, 1990; Saab et al., 1992, 2000; Tischenkel et al., 1989; Treiber et al., 1990), as well as groups with differential risk of hypertension (Fredrickson & Matthews, 1990).

Models that link laboratory reactivity to hypertension (Manuck & Krantz, 1986) should predict associations between laboratory responses and BP assessments in naturalistic settings. However, Turner et al. (1994) documented a mixed pattern of results across studies. A critical review of the literature relevant to the reactivity–hypertension relation led Pickering and Gerin (1990) to recommend the examination of reactivity in the context of more complex models of hypertension with the inclusion of relevant controls, such as family history of hypertension and attention to reliability. It has been suggested that both reactivity and recovery require evaluation because they may each provide unique information about mechanisms involved in BP regulation (Haynes et al., 1991). Thus, omitting recovery from reactivity protocols presents an incomplete picture, one that is inconsistent with early models of stress (Linden, Earle, Gerin, & Christenfeld, 1997). LGC modeling allows examination of these more complex models that incorporate additional risk factors and controls, in the simultaneous and parsimonious evaluation of reactivity, recovery, and their prediction of BP in natural settings.

Methods

Participants

Participants were 99 healthy adults, 25–54 years of age. Their resting BP levels were $M = 118/73$, $SD = 15.9/11.1$. The BP levels ranged from 90 to 155 and 54 to 98 for SBP and diastolic BP, respectively. No participant was on antihypertensive medication. They were deemed healthy based upon medical history, physical examination, fasting blood chemistry analysis, and 12-lead ECG. Participants were recruited through local newspaper advertising and were paid for their participation. The sample included 21 Blacks and 78 Whites, 48 men and 51 women, and 58 with positive and 41 with negative family history of hypertension.

Procedures

Laboratory. The present study utilized a 6-day protocol. Psychological and preliminary physical assessments were made on the first 2 days. Participants were instructed to eat a light meal prior to the third day's session, but were reminded to refrain from smoking (for 2 hr), and were instructed not to consume coffee (for 18 hr), tea, cocoa, or caffeinated soft drinks (for 6 hr) prior to their appointment.

On Day 3 of the protocol, BP was recorded while participants were at rest and while engaged in the cold pressor test. BP was obtained using a Critikon Dinamap (model 1846SX) Adult/Pediatric Vital Signs Monitor. The Dinamap monitor measures BP using the oscillometric method. Three baseline measurements were assessed at 2-min intervals at the conclusion of a 15-min rest period. Upon instruction, participants then placed their left foot into a bucket of ice water (half ice, half water, with temperature

about 4°C) for 90 s. Participants were instructed to keep their foot in the water until they were told to remove it. BP was assessed approximately at 0.75 and 1.5 min into the task. Upon instruction again, participants removed their feet from the water and rested. Three recovery readings were assessed at 1.5, 3.75, and 5.75 min after the task. Thus the sampling of BP across baseline, reactivity, and recovery periods occurred at approximately the following times in minutes for all participants:

$$\underline{0 \;\; 0.75 \;\; 1.5 \;\; 3.0 \;\; 5.25 \;\; 7.25}$$
$$\text{time in minutes}$$

Upon completion of the cold pressor task, participants responded to a questionnaire. The task appraisal questionnaire consisted of six items designed to assess the participant's appraisal of the "stressfulness" of the task. The items used a 5-point Likert type scale and included questions on task difficulty, challenge, pain, frustration, effort, and mood. A lower score on the questionnaire is associated with greater appraisal of stress.

Ambulatory. Ambulatory BP assessments were made on Days 4 and 5 of the protocol. Participants were fitted with an Accutracker II (Suntech Medical Instruments, Raleigh, NC) ambulatory BP monitor. The monitor was programmed to make assessments every 15 min. Participants were instructed to wear it until bedtime and to return to the lab with the monitor the next day. All participants in this study were fitted with the monitor around 9:00 a.m. and wore it until approximately 11:00 p.m. The participants were given a set of 50 diary cards and instructed to complete a diary card following each deflation of the cuff. Participants then recorded the time and indicated the place (such as work) on the card. They then left the laboratory and went about their normal daily activities. A work BP reading was then calculated for each participant by computing the mean of all the readings taken at work.

Plots. Prior to modeling the data, it is instructive to examine the plots of the outcome variable as a function of time. Figure 1 presents plots for 10 participants selected at random from our sample of $n = 99$. Although there are obvious individual differences across the 10 participants, the plots reveal a general pattern of increase in SBP from the baseline to the task and a subsequent decrease with an eventual plateau at the end of the recovery period. The plots guide the choice of the appropriate mathematical function, in this case a linear function for the reactivity period and possibly a curvilinear function for recovery. Other functional forms are possible to accommodate the pattern suggested by other stressors.

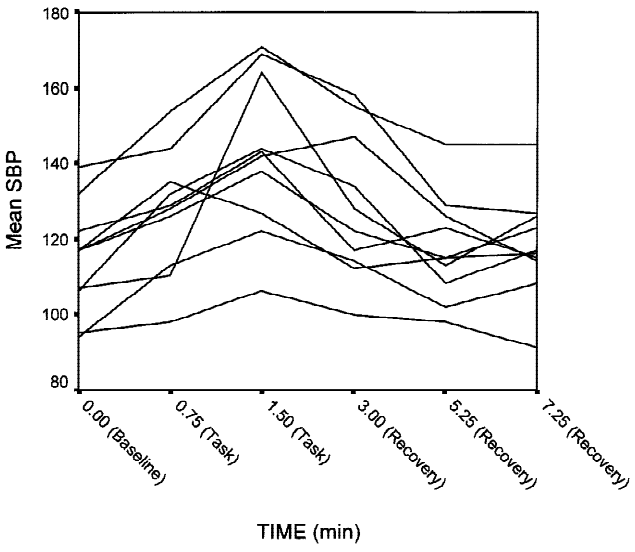

Figure 1. Systolic blood pressures during baseline, cold pressor task, and recovery for 10 randomly selected subjects.

Modeling Reactivity

At Level 1, BP for each individual is expressed as a function of the time period. For reactivity, we expected that this Level 1 model would take the form of a line; there would be a steady increase in BP from baseline to the end of the task. The corresponding linear model may be specified as

$$Y_{ij} = \pi_{0j} + \pi_{1j}\, t_{ij} + r_{ij}, \tag{1}$$

where Y_{ij} represents the BP measure for person j at time i. π_{0j} is the intercept for the BP trajectory for participant j or the predicted BP value at baseline, where time = 0 represents the baseline. π_{1j} is the slope of the trajectory for participant j, or the true change from baseline to stress level per minute change in time. t_{ij} is the time, i, corresponding to each measurement for person j. And r_{ij} represents the random error or unexplained deviations from the line for person j. This stochastic or error term contains measurement error combined with any time-specific error. Thus the intercept and slope parameters are estimated separately from the measurement error and are free from such error. Having one such equation for each person, we can then treat the true intercept (π_{0j}) and slope (π_{1j}) parameters as random variables that may be modeled at Level 2. A simple level 2 model may be

$$\pi_{0j} = \beta_0 + u_{0j}$$

$$\pi_{1j} = \beta_1 + u_{1j},$$

where β_0 represents the mean intercept and β_1 represents the mean slope. These are fixed parameters in the Level 2 model. u_{0j} and u_{1j} are random and represent the residual difference between the mean of all participants and each person's intercept or slope, respectively. Estimates of the variance associated with each random variable may be obtained as well as their covariance. Examination of the magnitude of the variance components in this simple model can serve as a guide in determining whether there is sufficient individual variability to warrant the inclusion of additional predictors at Level 2.

Although typically multiple baseline values are collected at specific time intervals during a baseline period, the assumption is that the baseline values will be stable during that period. If so, there is nothing to be gained by treating them as if they were different, or by taking the time of their measurement into account. In fact, this is the assumption underlying the practice of calculating an average baseline value. Thus all baseline readings were assigned the same time value of 0. This is analogous to averaging the baseline values, as is typically done, but improves on that by accounting for measurement error around that mean value.[1]

[1]It is only when multiple measurements are available for a given time that random measurement error may be estimated separately from time-specific error. When only one measurement is available at each time, measurement and time specific errors are confounded.

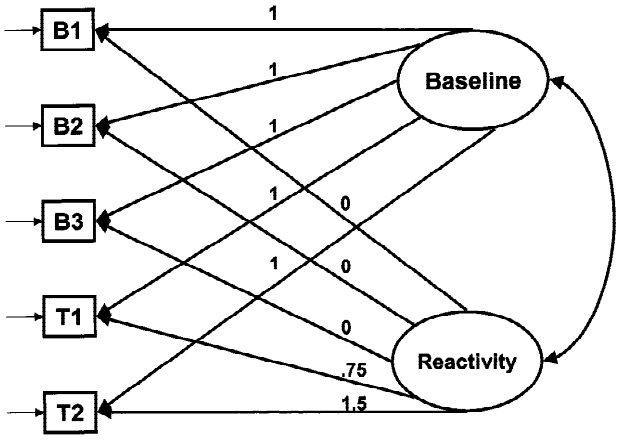

Figure 2. Latent growth curve model of reactivity.

Figure 2 shows a path diagram of the LGC model of reactivity. Factor loadings are shown in the arrows pointing from the latent (ovals) to the observed (boxes) variables. Unlike the typical application of SEM where the parameters to be estimated include the loadings, in LGC modeling, the loadings are fixed to values that convey information about the nature of the latent variable. The mean and variance (and covariance) of the latent variables are the parameters estimated in LGC. In the reactivity model, the latent variables are the intercept and slope of the model defined in Equation (1). As shown in Figure 2, the loading for each latent intercept is 1, indicating the constant coefficient for the intercept in the model. The loadings for the latent slope are the corresponding times when the BP measures were taken.

Any SEM software may be used to obtain estimates of the model parameters and their standard errors, as well as a test of model fit. Common estimation methods assume multivariate normality, and we found the assumption to be tenable for these data. Mplus was used for all model estimation and testing. Table 1 shows the values, standard errors, and *t*-values for the fixed parameters and the variance and covariance components of the random parameters for SBP. The results indicate the predicted mean SBP was 118 mmHg at baseline. During cold pressor reactivity, there was an average increase of 12.6 mmHg in SBP per minute into the task. There was also significant variability in baseline values and reactivity across participants. This means that, although a pattern characterized by the mean values was consistent with the data, there were individual differences in both baseline and reactivity worthy of further investigation at Level 2. Also the correlation (standardized covariance) between baseline and reactivity was .30 and significant. The linear model provided adequate fit to the data with $\chi^2(13) = 10.73$, $p = .63$ for SBP. The root mean square error of approximation (RMSEA) value was .001.[2] We should note that the model fit was assessed when constraining the measurement error variances for the baseline measures to be equal to each other and the measurement error variances for the reactivity measures also to be equal to each other.[3] Measurement errors were uncorrelated across measures. One advantage of the SEM approach is the flexibility and ease in specifying these error variance-covariance structures. Error variances and covariances may be estimated freely or specified to conform to a predetermined pattern, such as is done in traditional repeated measures analysis of variance where sphericity is assumed.

Table 1. *Fixed and Random Parameter Estimates for SBP Reactivity Model*

	Estimate (mmHg)	*SE*	*t*
Fixed parameters			
Baseline	117.773	1.586	74.235
Reactivity	12.648	1.022	12.31
Random parameters			
Variances			
Baseline	241.560	35.425	6.819
Reactivity	77.240	15.044	5.134
Covariances			
Baseline/reactivity	40.597	16.518	2.458

t-values > 2 are significant, $p < .05$.

Modeling Recovery

For recovery, we expected an initial decline that stabilized over the recovery period. In this fashion, we may wish to model a quadratic polynomial as shown below:

$$Y_{ij} = \pi_{0j} + \pi_{1j} t_{ij} + \pi_{2j} t_{ij}^2 + r_{ij}.$$

If we code the last time during the cold pressor as time = 0, then π_{0j}, the intercept for the blood pressure trajectory for participant j, represents the predicted blood pressure value at the last task reading. π_{1j} is the linear coefficient of the trajectory for participant j, or the instantaneous slope at time = 0. The change in meaning for the slope parameter results from fitting a quadratic function (parabola), where the slope changes as a function of time. π_{2j} is the quadratic coefficient, reflecting the direction and degree of curvature of the parabola, and contributing to the conditional slope as described in the results below. t_{ij} is the time i corresponding to each measurement for person j. And r_{ij} contains random measurement error or the residual deviation from the curve for person j.

At Level 2, there are now three parameters that may be modeled. For example,

$$\pi_{0j} = \beta_0 + u_{0j}$$

$$\pi_{1j} = \beta_1 + u_{1j}$$

$$\pi_{2j} = \beta_2 + u_{2j}.$$

Each parameter may be modeled by a fixed component expressing the mean across all participants, and a random component representing individuals' deviations from the mean. For the random variables, u_{ij}, variance and covariance components may be estimated. Figure 3 shows the path diagram for recovery, which

[2]Nonsignificant χ^2 values indicate consistency between the model and the data. RMSEA values below .06 are also indicative of close model fit. For more information about these and other fit indices and relevant criteria see Hu and Bentler, 1999.

[3]The conventional hierarchical linear model approach assumes all the error variances to be equal. In our situation, that assumption was not consistent with the data. Because within the baseline and the reactivity periods all procedures are constant, it made logical sense to assume equal measurement errors within periods, but different between periods.

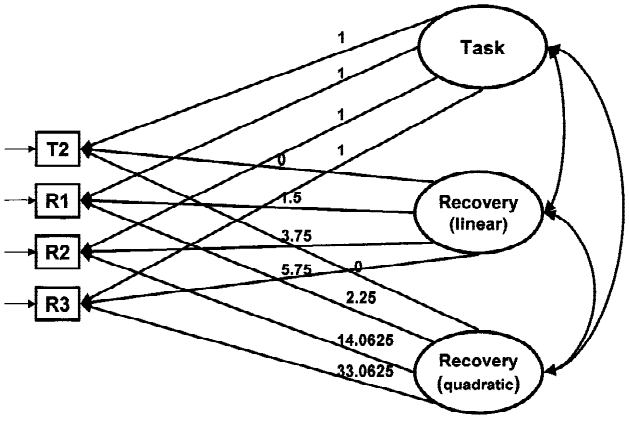

Figure 3. Latent growth curve model of recovery.

includes three latent variables. An added feature of this diagram is the set of coefficients for the loadings of the quadratic latent variable. These coefficients are the squares of the times in the design.

Table 2 shows the results of the parameter estimates for SBP recovery. As shown in the table, the predicted mean SBP at the end of the cold pressor test was 137 mmHg. Upon completion of the task, SBP instantaneously declined by an average of 8.2 mmHg and this decline is reduced by an average of 1.68 mmHg per minute (2×0.84, or the coefficient in the first derivative). The first derivative of the quadratic polynomial of SBP as a function of time gives us an equation of the conditional slope (conditional on time). The equation is $-8.2 + 1.68t$. Thus the estimation of the slope in the context of a quadratic function takes into account both the linear and the quadratic parameters, as well as the specific time where the changing slope is calculated. Setting the conditional slope equation equal to 0 yields the average time associated with the end of the decline. This may be considered the time when full recovery has taken place. For our data, this time is 4.85 min into the recovery period.

Estimates of the variance components showed significant variability in all three parameters, but the magnitude of the quadratic variance (0.58) was small relative to the variance component for the task level (552.92) and the linear parameter (34.35). This indicates limited variability in curvature, and therefore seeking external predictors of the quadratic parameter in subsequent equations may not be productive. There was also significant and substantial covariation among all three parameter estimates, particularly between the linear and quadratic components. A test of model fit indicated that the quadratic polynomial provided good fit for the SBP cold pressor recovery data $\chi^2(3) = 1.44$, $p = .70$, RMSEA = .001.[4]

Table 2. *Fixed and Random Parameter Estimates for SBP Recovery Model*

	Estimate (mmHg)	*SE*	*t*
Fixed parameters			
Task	137.015	2.459	55.721
Recovery	−8.151	0.739	−11.036
Recovery^2	.836	0.102	8.204
Random parameters			
Variances			
Task	552.915	85.365	6.477
Recovery	34.346	8.671	3.961
Recovery^2	.580	0.191	3.035
Covariances			
Task/recovery	−100.436	22.186	−4.527
Task/recovery^2	9.998	2.828	3.536
Recovery/recovery^2	−4.195	1.237	−3.392

t-values > 2 are significant, $p < .05$.

Piecewise Regression of Reactivity and Recovery

Ideally we should model the reactivity and recovery data jointly, in order to assess their interrelation. Rather than using a single function to model the responses across both periods, it is optimal to model each period separately but simultaneously using a piecewise regression approach. The piecewise approach is more appropriate than using a single function for at least two reasons. An obvious one is that the design characteristics are different between the two periods. For the reactivity period, the stressor is introduced, whereas for recovery, the stressor is removed. More importantly, the parameters of interest (i.e., the slope) for each period are lost when modeling the combined data, if we assume that time is the only changing factor.

Piecewise regression may be used for both continuous and discontinuous functions. Our two functions are continuous because they share a common time at the last task reading. The reactivity period began with the baseline values and ended with the last task reading. The recovery period began with the last task reading and ended at the last reading taken. The time variable may be centered at that common point, making that the point of the intercept. Times associated with the reactivity period will then have the appropriate negative values, and recovery times will be positive. Alternatively, time may be more meaningfully kept centered at baseline. Because we expect baseline values to remain stable during the baseline period, and modeling the baseline period is not of interest, all of the baseline values were assigned the same values for time.

Our approach is a modification of the coding described by Neter, Kutner, Nachtsheim, and Wasserman (1996) for piecewise regression. In their model, they included time and the interaction of time by a dummy coded vector. They also centered time at the common point, meaning that at the common point, time = 0. Willett, Singer, and Martin (1998) described the potential application of piecewise regression to multilevel models using the same coding approach and a linear function. The approach is to first compute the interaction of a dummy vector and time. The dummy vector is coded such that

$$\text{dummy} = 0, \quad \text{if time} \leq 0$$

$$\text{dummy} = 1, \quad \text{if time} > 0,$$

where 0 is the common point (i.e., time is centered at the common point).

The resulting equation is $Y = b0 + b1 \text{ time} + b2 \text{ time} * \text{dummy}$. Thus, for the initial period, where time ≤ 0, the third term disap-

[4] An alternative approach may be used to capture the nonlinearity in the data. Rather than specifying a quadratic component, the last two times during the recovery period are not assigned fixed time values, but are allowed to be estimated so as to maximize fit by the program. This approach fits a linear spline to the data. The final decision should be based on the optimal model fit for a given application.

pears and $b1$ provides an estimate of the slope for that period. The difference between the slope for the first period and the slope for the second period is indicated by $b2$.

To estimate the two slopes, one for each period, rather than one slope and the difference between the two slopes, we included two dummy vectors and omitted the original time variable. Once we centered the time variable at the common point, we then multiplied it by two dummy coded vectors, $D1$ and $D2$.

$$D1 = 0, \text{ if time} \geq 0$$

$$D1 = 1, \text{ if time} < 0$$

$$D2 = 0, \text{ if time} \leq 0$$

$$D2 = 1, \text{ if time} > 0.$$

Centering at the common point, which is the last reactivity value, yields the following time values:

$$\underline{-1.5 \;\; -.75 \;\; 0 \;\; 1.5 \;\; 3.75 \;\; 5.75}$$
$$\text{time}$$

Multiplying time by $D1$ and $D2$ will yield

$$\text{REACT} = D1 * \text{time: } -1.5 \;\; -.75 \;\; 0 \;\; 0 \;\; 0 \;\; 0$$

$$\text{RECOV} = D2 * \text{time: } 0 \;\; 0 \;\; 0 \;\; 1.5 \;\; 3.75 \;\; 5.75$$

The two resulting interaction vectors, REACT and RECOV, contain information about the times for a specific period and the constant 0, for the other period. The common point has a value of 0 for both vectors; that is where time is centered. The centering may later be changed to coincide with any time where the value of 0 may be meaningful. In our application, it was meaningful to consider the baseline value as the point where time should equal 0. To effect this change we simply added 1.5 to each value in REACT. The recentered vectors are:

$$\text{REACT: } 0, .75, 1.5, 1.5, 1.5, 1.5$$

$$\text{RECOV: } 0, 0, 0, 1.5, 3.75, 5.75$$[5]

The equation is

$$Y_{ij} = \pi_{0j} + \pi_{1j}\text{REACT}_{ij} + \pi_{2j}\text{RECOV}_{ij} + r_{ij}.$$

When time = 0, both REACT and RECOV are 0, and the response value represents the intercept. The intercept is the value associated with the predicted average baseline reading. During the reactivity period, REACT contains information about the times for that period, but holds constant the times for the recovery period. Conversely, during the recovery period, RECOV contains information about the times for the recovery period, but holds constant the times for the reactivity period. Taken together, the two vectors, REACT and RECOV, convey information about the common intercept, as well as their respective time values. Note that the time variable was left out of the equation. Including it would have resulted in an overparameterized model. We then included a quadratic term for the recovery period only, by adding a squared term for RECOV to the equation.

Table 3. *Fixed and Random Parameter Estimates for Piecewise Model of Reactivity and Recovery*

	Estimate (mmHg)	*SE*	*t*
Fixed parameters			
Baseline	117.778	1.592	73.961
Reactivity	12.645	0.998	12.671
Recovery	−8.014	0.726	−11.038
Recovery^2	0.820	0.102	8.058
Random parameters			
Variances			
Baseline	243.464	35.691	6.821
Reactivity	74.807	14.162	5.282
Recovery	35.125	8.376	4.193
Recovery^2	0.647	0.192	3.368
Covariances			
Baseline/reactivity	42.084	16.235	2.592
Baseline/recovery	−32.632	11.944	−2.732
Baseline/recovery^2	3.242	1.643	1.973
Reactivity/recovery	−42.227	9.319	−4.531
Reactivity/recovery^2	4.312	1.190	3.624
Recovery/recovery^2	−4.489	1.221	−3.678

t-values > 2 are significant, $p < .05$.

Results of the piecewise regression model are shown in Table 3. Model fit indices for the piecewise model of SBP, with a linear term for the reactivity period and a quadratic term for the recovery period, suggest a good fit to the data, $\chi^2(27) = 34.53$, $p = .151$, RMSEA = .053.[6] Consistent with the separate models previously shown, the significant fixed parameters indicate that participants started at a predicted SBP baseline level of 118 mmHg and increased by an average of 12.6 mmHg per minute during the cold pressor task. Upon completion of the cold pressor task, participants' SBP initially declined by an average of 8.0 mmHg, but the decline was gradually reduced by an average of 1.68 mmHg per minute. Minor differences in parameter estimates when models were fitted separately result from the nature of the iterative estimation algorithm. We consider these differences to be negligible. There was significant individual variability in all parameters but, relative to the others, the quadratic variance component was small. Correlations among parameter estimates showed that baseline was significantly related to both reactivity (.31) and recovery (−.35), with higher baseline values generally associated with increased reactivity and steeper recovery. There was also considerable correlation between reactivity and recovery (−.82), raising the question of the uniqueness of these two related constructs. The quadratic

[5] An alternative way to think about the coding is to specify two coded vectors, REACT and RECOV, each with appropriately varying intervals for the period it measures, and constant values for the period it does not measure. So for instance, to code the reactivity period we specify the values 0, 0.75, 1.5 for the three times during the reactivity period. The recovery times are then maintained at the constant 1.5. To code the recovery period we begin with the constant 0 and then assign the appropriate interval, 1.5, to the first recovery time because there was an interval of 1.5 between the last reactivity time and the first recovery time. Likewise the other recovery times will be assigned 3.75 and 5.75 representing the difference between the last reactivity time and each recovery time.

[6] Piecewise LGC models may require the specification of starting values for convergence. This is particularly true for the random variance components. Fitting the pieces individually as a first step provides estimates that may then be used as starting values in the more complex model.

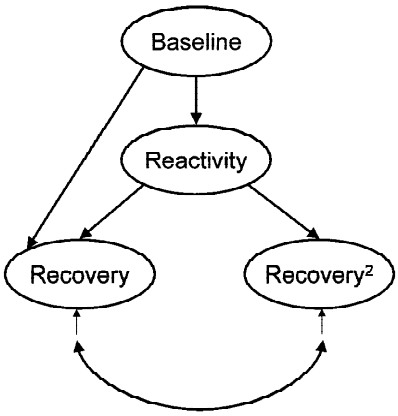

Figure 4. Piecewise structural model of reactivity and recovery with direct effects.

component was correlated with reactivity (.62) and recovery (−.94) as expected, but not with baseline (.26).

This piecewise LGC model was restated to incorporate our view of the directionality of effects among the latent parameters and thereby illustrates this unique capability of LGC modeling. As shown in Figure 4, baseline levels had a direct effect on both reactivity and the linear component of recovery. Reactivity also directly influenced both aspects of recovery. The two components of recovery were correlated. This path model differs from the previous piecewise model (reported in Table 3 but not shown in a figure) in that no effect is presumed between baseline and the quadratic component of recovery, and therefore is nested in the previous model. The difference in χ^2 (1) = 0.402 was not significant, indicating that the more restricted model was not worse. The fit of this restated model was quite good $\chi^2(28) = 34.93$, $p = .17$, RMSEA = .05. Whether relations between variables are specified as direct effects or simply correlated cannot be distinguished on statistical grounds, but rather depends on our conceptualization of the stress process.

Introducing Level 2 Variables

Predictors of the Level 1 model parameters may be introduced at Level 2. Initially, gender, ethnicity, and family history of hypertension were used to predict baseline SBP levels, and together with task appraisal, predict reactivity and recovery. In addition, all four exogenous variables together with baseline, reactivity, and recovery were used to predict work SBP. The structural aspect of this model is close to being saturated in that most effect parameters are estimated freely. It was used as a starting point against which other nested models could be compared. This model fit the data well, $\chi^2(49) = 57.50$, $p = .165$, RMSEA = .042. An inspection of the effect parameter estimates indicated several nonsignificant parameters that could potentially be considered equal to zero without loss of fit. This information, together with previous findings in the literature, was used to revise the model with certain parameters set to zero. The revised model is presented in Figure 5. Modifications to the model were done in three steps, although intermediate results are not presented. First, the paths from the exogenous variables to the latent parameters were revised. Then, the paths from the exogenous variables to work SBP were revised. Finally, the paths from the latent variables to work SBP were systematically revised and interpreted.

At the first modification step, gender differences in reactivity and recovery were set to zero, as were the effects of the exogenous variables on the quadratic component of recovery. All these parameters were nonsignificant. Also, gender differences in SBP have not been observed for the cold pressor test (Saab, 1989) unless gender relevant instructions are given (Lash, Gillespie, Eisler, & Southard, 1991), which was not the case in the present study. With respect to the quadratic component, we initially noted very little variability in this parameter, and anticipated difficulty detecting its predictors. The resulting model showed good fit to the data, $\chi^2(55) = 66.25$, $p = .14$, RMSEA = .045. The fit did not diminish relative to the more saturated model, $\Delta\chi^2(6) = 8.75$, $p > .10$. An examination of the parameter estimates for the effects of exogenous variables (gender, ethnicity, family history, and task appraisal) on work SBP revealed that only gender retained a significant direct effect.

Direct effects from ethnicity, family history, and task appraisal to work SBP were then set to zero. It is important to note that these variables retained their indirect effects to work SBP through baseline, reactivity, and recovery. Again, there was good model fit, $\chi^2(58) = 67.64$, $p = .18$, RMSEA = .041. Examination of the direct effects from baseline, reactivity, and recovery to work SBP revealed that, when all direct effects were specified, the partial effects of reactivity and recovery were nonsignificant, but the baseline effect was significant. Because of the shared variability between reactivity and recovery, it may be the case that their relation to work SBP was not unique. Therefore, we restated the model, specifying the effect to work SBP from the recovery parameters only, with the reactivity effect as indirect. The parameters of this model, which showed good fit to the data, $\chi^2(59) = 67.69$, $p = .20$, RMSEA = .039, are the ones shown in Table 4 and interpreted below. We should mention that an alternative model with the direct effect to work SBP stemming from reactivity was also tested and found to fit well, $\chi^2(58) = 68.99$, $p = .15$, RMSEA = .044. The parameter estimates from both of these last two models were almost identical, with the exception, of course, of the effects from reactivity or recovery to work SBP.

The results shown in Table 4 indicate that most direct effects in the model were statistically significant. However, ethnic differences were not detected for baseline or reactivity, only in the linear component of recovery. Family history of hypertension predicted

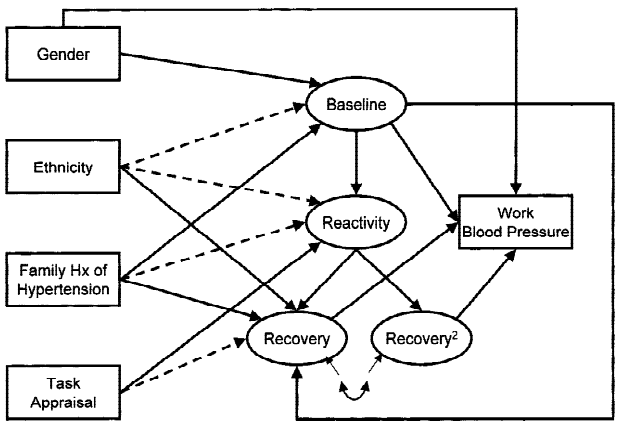

Figure 5. Piecewise structural model of reactivity and recovery with gender, ethnicity, family history of hypertension, and task appraisal as predictors and work SBP as outcome.

Table 4. *Effect Parameters of Final Model*

Criterion	Predictor	Estimate (mmHg)	*SE*	*t*
Baseline	Male gender	11.496	2.908	3.953
	Black ethnicity	5.924	3.568	1.661
	Positive family Hx	7.759	2.901	2.675
Reactivity	Black ethnicity	1.467	2.218	0.662
	Positive family Hx	−2.224	1.904	−1.168
	Task appraisal	−0.578	0.147	−3.920
	Baseline	0.207	0.061	3.375
Recovery	Black ethnicity	0.505	0.231	2.218
	Positive family Hx	−0.605	0.201	−3.009
	Task appraisal	−0.003	0.018	−0.155
	Baseline	−0.016	0.007	−2.132
	Reactivity	−0.558	0.058	−9.602
$Recovery^2$	Reactivity	0.057	0.011	5.410
	Recovery	−1.994[a]	0.743	−2.683
Work SBP	Male gender	7.546	1.775	4.251
	Baseline	0.476	0.068	7.042
	Recovery	−1.638	0.573	−2.856
	$Recovery^2$	−10.119	4.318	−2.344

[a]Value represents the covariance.
t-values > 2 are significant, $p < .05$.

recovery but not reactivity, whereas appraisal of the task predicted reactivity, but not recovery. This model explained 19% of the variance in baseline values, 29% of the variance in reactivity, and 69% and 40% of the variances in the linear and quadratic components of recovery, respectively. The model explained 65% of the variance in work SBP.

Close examination of the partial effect parameters indicated that, after controlling for the other exogenous variables, the average baseline SBP of the men was 11.5 mmHg higher than that of the women. The average SBP of the Blacks was about 6 mmHg higher than that of the Whites, although this study did not have sufficient power to detect this effect as significant. Also, the average SBP of participants with a positive family history of hypertension was 7.8 mmHg higher than that of those without such family history.

Baseline SBP levels related to reactivity such that every 1.0 mmHg increase in baseline level was associated with a 0.2 mmHg increase in reactivity. Also, the appraisal of the cold pressor test as stressful, painful, or difficult was associated with increases in reactivity, after controlling for baseline level. Every unit increase in appraisal of stressfulness of the task was associated with more than 0.5 mmHg in reactivity (low scores represent high stress appraisal).

When controlling for other predictors including baseline, reactivity, family history, and task appraisal, the instantaneous decline in SBP after the cold pressor test was steeper for Whites than for Blacks by about 0.5 mmHg. With similar controls including ethnicity, participants with a positive family history of hypertension had steeper declines (−0.605) in their instantaneous slope than those without a positive family history. Also, although both baseline and reactivity directly influenced recovery in the same direction, the partial effect of reactivity (−0.558) was greater than that of baseline (−0.016). The quadratic component of recovery was also influenced by reactivity, such that every millimeter of mercury increase in reactivity was associated with 0.057 rate of change in recovery. Because the recovery slope was initially negative, the positive rate of change implies a lessening of the negative slope. The linear and quadratic components of recovery retained a significant correlation (−.42), even when controlling for other variables in the model.

With respect to work SBP, the average for men was 7.5 mmHg higher than for women. As one would expect, higher baseline values were also associated with higher work values. Both components of recovery influenced work SBP such that higher values at work are associated with steeper instantaneous slopes and smaller changes in slope, a pattern consistent with higher reactivity. The magnitude of the unstandardized partial coefficients cannot be compared between the linear and quadratic components of recovery because they occur in different units, essentially representing different variables (analogous to velocity and acceleration).

Discussion

LGC modeling provided a powerful method for testing a complex model of SBP reactivity and recovery from the cold pressor test. Unlike more conventional approaches, such as regression, LGC modeling controlled for measurement error in reactivity and recovery, permitted their simultaneous analysis, used all individual data points, and allowed reactivity and recovery to function as both predictors and outcomes of other variables.

The model included common predictors of BP identified in the literature: gender, ethnicity, and family history of hypertension, as well as a measure of appraisal of the stressfulness of the task. In addition, work SBP was used as an outcome variable: a marker of disease risk. A stepwise approach was used to develop and test the model. This approach is recommended because it facilitates the identification of problems common to SEM model fitting. Information from prior literature, assumptions about the stress process, and the data, all contributed to the final model. The results showed good fit of the model to the data.

Consistent with prior literature, family history of hypertension was a significant predictor of baseline SBP and the initial recovery from the cold pressor test, but not reactivity. The importance of including family history as a control variable in reactivity studies has been underscored by Pickering and Gerin (1990), based on the number of studies reporting their association (see Fredrickson & Matthews, 1990; Matthews & Rakacsky, 1986 for reviews). Two factors may explain the lack of direct association in the present study. One possible explanation is that in the model studied, baseline BP was included as a mediator in the family history–reactivity relation. Family history may influence reactivity because it influences initial BP levels. This possibility points to what is already commonly mentioned but not practiced in the literature: that reactivity studies control for baseline levels. A second possibility lies in the choice of cardiovascular parameter for the present study. SBP was chosen because of its emergence in a prospective study of cold pressor reactivity and subsequent hypertension (Menkes et al., 1989). However, the family history influence on reactivity may be more easily detected on measures of peripheral resistance. Multivariate models of cardiovascular reactivity may illuminate this possibility.

Gender differences in SBP, commonly reported in the literature (Saab, 1989) were evident in this study. The differences were observed in baseline levels and in levels at work, but not in reactivity or recovery. It seems that gender differences in reactivity are specific to the task used, and that the cold pressor test is not one to evoke them. Lash et al. (1991) suggested that part of the reactivity gender effect is a function of cognitive appraisal of a stressor as gender specific. They, in fact, detected gender differ-

ences in SBP reactivity to the cold pressor test when "masculine" rather that gender-neutral instructions were provided. The standard administration of the cold pressor test, however, is not gender specific. As a result, gender differences in reactivity to the cold pressor have not been widely reported (Saab, 1989).

Ethnic differences in baseline and reactivity SBP were not strong enough to be detected in the present study with a small number of Black participants, and emerged only with respect to the linear component of recovery. The difference in recovery slopes was small in magnitude, once baseline, reactivity, and family history were controlled. Given the greater prevalence of hypertension in Blacks when compared to Whites, stronger ethnic effects were anticipated. However, ethnic differences in BP levels, reactivity, or the mechanism by which recovery from stress operates may be more evident in DBP. In their meta-analysis of hypertension risk factors and recovery from stress, Hocking Schuler & O'Brien (1997) noted heterogeneity across studies in the effect of ethnicity on SBP recovery, with some studies reporting "more complete recovery" for Blacks when compared to Whites. We recognize that the small number of Black participants in our data may have prevented us from more fully understanding the ethnicity effect.

The predictor that had the strongest effect on reactivity was task appraisal. This measure included an item on the respondent's perception of pain from the cold pressor. Studies from our laboratory documented the role of pain perception on reactivity to the cold pressor test (Peckerman et al., 1991, 1994, 1998). Peckerman et al. (1994) provided evidence that large magnitude increases in BP reactivity to the cold pressor test are strongly related to the magnitude of perceived pain ($r = .79$). Furthermore, the pain and nonpain-related increases in BP were analyzed as residual effects of concurrent changes in cardiac output and peripheral resistance. The partial effect associated with pain included positive changes in both cardiac output and peripheral resistance, whereas the nonpain effect was specifically related to peripheral resistance only. Peckerman et al. (1994) concluded that elevated BP reactivity to this test is closely related to its psychological properties. The results of the present study support that conclusion.

SBP at work was used as a marker of disease risk. Ideally our model would have included a more direct measure such as left ventricular hypertrophy. The results showed an effect from either reactivity or recovery on this marker. When both components were included together, their shared variation did not allow the detection of their effect. But in the context of a path model, the effect was seen when the reactivity effect was mediated by recovery. It was clear from our results that both reactivity and recovery influence work SBP, even after controlling for baseline levels. Although the inferences that may be drawn from this study pertain to disease risk rather than disease per se, the potential for the methodology to uncover important relations that may underlie disease is quite clear. Future applications of LGC methodology, which include better disease markers, will go a long way toward unraveling the role of reactivity in the pathogenesis of hypertension.

Are reactivity and recovery different processes? The strong inverse relation between them would suggest that they reflect opposite processes. However, there are a few distinct features detected in this study that would suggest the need to differentiate them. At a basic level, they are modeled by different functions, linear for reactivity and quadratic for recovery. And although the quadratic component of recovery did not display a lot of individual variability in this sample, it was a significant predictor of work SBP, suggesting that both aspects may indeed be important for predicting disease. Another distinction between reactivity and recovery was in their predictors with direct effects. Beyond what is explained by baseline levels, reactivity was predicted by appraisal of the task, whereas the linear component of recovery was influenced by ethnicity and by family history of hypertension. But although some differentiation was evident with the cold pressor test, other stressors might differentiate them further. A single stressor may not identify all of the uniqueness of reactivity and recovery. In fact, although the cold pressor has been shown to have a psychological component, pain (Peckerman et al., 1994), this may not be the psychological mechanism associated with delayed recovery.

Using LGC methodology raised sampling considerations in these kinds of studies. How frequently and when should we sample the outcome? The number of measurements determines the shape of the curve that can be modeled. The spacing of the measurements determines the change that may be captured. Frequent sampling should coincide with the time period when the greatest change is taking place. The expected trajectory may be used to guide sampling decisions. If change from baseline is expected right away, more frequent sampling should occur early. Certainly, more frequent sampling will allow the estimation of more complex curves and better separation between the latent variables and error. The issue of how many measurements are sufficient to estimate the different parameters with precision is beyond the scope of this paper, but worthy of further investigation with LGC methodology.

Despite the limitations of the study in terms of the measure of disease risk, the use of a single stressor, and the univariate nature of the cardiovascular parameter, LGC methodology proved a powerful tool for studying the processes of BP reactivity and recovery from stress and the prediction of BP at work.

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(Received January 9, 2001; Accepted June 19, 2001)