CHAPTER 3

MOOD CHANGES ASSOCIATED WITH SMOKING IN ADOLESCENTS

An Application of a Mixed-Effects Location Scale Model for Longitudinal Ecological Momentary Assessment Data

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INTRODUCTION

Modern data collection procedures, such as ecological momentary assessments (EMA; Smyth & Stone, 2003; Stone & Shiffman, 1994), experience sampling (Feldman Barrett & Barrett, 2001; Scollon, Kim-Prieto, & Diener, 2003), and diary methods (Bolger, Davis, & Rafaeli, 2001) yield relatively large numbers of subjects and observations per subject, and data from such designs are sometimes referred to as intensive longitudinal data (Walls & Schafer, 2006). Analysis of EMA data using mixed models (also known as

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multilevel or hierarchical linear models) is well described by Schwartz and Stone (2007). Additionally, Moghaddam and Ferguson (2007) analyzed EMA data using mixed models to examine smoking-related changes in mood. These articles focus on the effects of covariates, either subject-varying or time-varying, on the EMA mean responses. Here we extend this approach by examining the degree to which covariates influence the within-subjects variation inherent in the EMA data.

A few articles have described approaches for examining determinants of between- and within-subjects variance from EMA studies. Penner, Shiffman, Paty, and Fritzsche (1994) used basic descriptive statistical methods to examine relations among within-subject variation in several mood variables. Jahng, Wood, and Trull (2008) described generalized mixed models to analyze within-subject differences in sequential EMA mood responses, specifically characterizing these as mean square successive differences. Hedeker, Mermelstein, Berbaum, and Campbell (2009) described a mixed model that included determinants of the between-subjects variance, while Hedeker, Mermelstein, and Demirtas (2008) developed a model that additionally allowed determinants of the within-subjects variance plus a random subject scale effect. This model is referred to as a *mixed-effects location scale model* because subjects have both random location and scale effects. Models with random scale effects have been described in several articles where interest centers on variance modeling and/or accounting for heterogeneous variation across individuals or clusters (Chinchilli, Esinhart, & Miller, 1995; Cleveland, Denby, & Liu, 2002; James, Venables, Dry, & Wiskich, 1994; Leckie, 2010; Lin, Raz, & Harlow, 1997; Myles, Price, Hunter, Day, & Duffy, 2003).

In this chapter, we extend the mixed-effects location scale model to focus on the variation of mood change that is associated with smoking across measurement waves, and the degree to which subject and wave characteristics influence the variation in mood changes. Also, while Hedeker et al. (2008) only considered random subject intercepts for the one wave of EMA data, here we allow random subject time trends for the multiple waves of EMA data. We further consider a three-level model that treats observations nested within waves within subjects. To aid in making this class of models accessible, sample computer syntax is provided in the Appendix.

ADOLESCENT SMOKING, MOOD, AND VARIABILITY

Many prominent models of cigarette smoking maintain that smoking is reinforcing, and that smoking can relieve negative affect (Kassel, Stround, & Paronis, 2003; Khantzian, 1997). Indeed, both adults and adolescents often claim that smoking is relaxing and reduces emotional distress (Chassin, Presson, Rose, & Sherman, 2007; Kassel & Hankin, 2006). However, although the relation between mood and smoking has received substantial empirical attention for adult smokers, much less is known about the acute changes in mood with smoking among adolescents. The present study, with its focus on real-time assessments of mood and smoking among adolescents, helps to shed light on this important topic.

Although there is substantial consensus among both smokers and researchers that smoking helps to regulate affect, most of the empirical work investigating the smoking-mood relation has focused on the examination of changes in mean levels of mood with smoking. Surprisingly, although affect regulation inherently implies the modulation of variability in mood as well, the examination of variability in mood and smoking has largely been neglected. As Hertzog and Nesselroade (2003) noted, describing mean levels of variables is not always adequate for examining key features of developmental change. Variation also conveys important information about the phenomenon of interest. In the case of adolescent smoking and the development of dependence, variation in mood and mood changes may help to explain more of the development of tolerance. Examining individual variability may enhance our ability to predict changes in smoking behavior above and beyond what can be achieved by examining mean information alone.

Important, too, in the examination of mood and smoking is the distinction between within-person and between-person variability. Kassel and colleagues (Kassel & Hankin, 2006; Kassel et al., 2003) have argued persuasively for the need to differentiate causal within-person mechanisms from between-person data. Whether smoking relieves negative affect is essentially a within-person question, and thus analytic models need to similarly differentiate between within-subject and between-subject effects.

Much of the research on mood and smoking has also been limited to assessments of negative affect, while ignoring positive affect. This neglect is particularly problematic given the theoretical importance of differentiating between negative reinforcement models of smoking and positive reinforcement models, especially in the development of dependence among adolescents (Tiffany, Conklin, Shiffman, & Clayton, 2004). There is also considerable evidence to support the notion that positive and negative affect are distinct constructs, and not just opposite ends of a continuum (Watson & Tellegen, 1985; Watson, Wiese, Vaidya, & Tellegen, 1999). Thus, in the current study, we assessed both positive and negative affect.

Finally, there may well be individual differences in the extent to which adolescents' moods vary and whether these moods vary with smoking. Identifying potential moderator variables may also help in the prediction of smoking escalation among relatively novice smokers. Indeed, in a previous paper (Hedeker et al., 2009) it was observed that adolescent smoking level was associated with variation in mood changes associated with smoking, diminishing this variance for both positive and negative affect. While this finding was noteworthy, it represents a between-subjects effect of smoking level, rather than addressing the point of whether variation in mood changes associated with smoking diminishes *as a person increases their smoking level* (a within-subjects effect). Here, we aim to assess this within-subjects effect by modeling the EMA data across several measurement waves as a subject changes his or her smoking level. We hypothesized that the between-subjects effect of smoking level that we previously reported would also be observed as a within-subjects effect. Namely, as adolescents increase their level of smoking across time, their variation in mood changes associated with smoking would diminish. Thus, following along the lines of the development of tolerance with dependence, we hypothesized that as smoking level or experience increased, mood responses to smoking would decrease, as would variability in overall mood.

METHODS

Subjects

The data are drawn from a natural history study of adolescent smoking. Participants included in this study were in 9th or 10th grade at baseline, 55.1% female, and reported on a screening questionnaire 6–8 weeks prior to baseline that they had smoked at least one cigarette in their lifetimes. The majority (57.6%) had smoked at least one cigarette in the past month at baseline. Written parental consent and student assent were required for participation. A total of 461 students completed the baseline measurement wave. Of these, 57% were white, 20% Hispanic, 16% black, and 7% of other race.

The study utilized a multimethod approach to assess adolescents including self-report questionnaires, a week-long time/event sampling method via hand-held computers (EMA), and detailed surveys. Adolescents carried the hand-held computers with them at all times during a data collection period of 7 consecutive days and were trained both to respond to random prompts from the computers and to event-record (initiate a data collection interview) in conjunction with smoking episodes. Random prompts and the self-initiated smoking records were mutually exclusive; no smoking occurred during random prompts. Questions concerned place, activity, companionship, mood, and other subjective variables. The hand-held computers dated and time-stamped each entry. Following the baseline assessment, subjects completed additional EMA sessions at 6-, 15-, and 24-month follow-ups, for a total of four EMA measurement waves. Subject retention was good, with 405, 360, and 385 subjects completing the EMA sessions at these three follow-ups, respectively. Since estimation of model parameters is based on a full-likelihood approach, the missing data are assumed to be "ignorable" conditional on both the model covariates and the observed responses of the dependent variable (Laird, 1988). In longitudinal studies, ignorable nonresponse falls under the "missing at random" (MAR) mechanism of Rubin (1976), in which the missingness depends only on observed data. As Molenberghs et al. (2004) indicate, MAR is a relatively weak assumption, especially as compared to the more stringent missing completely at random (MCAR) assumption, and one that we will make here. For the interested reader, extended not missing at random (NMAR) approaches are described in Chapter 14 of Hedeker and Gibbons (2006).

Because of our interest in comparing mood *within subjects* from smoking events across measurement waves, we restricted our analysis to subjects who provided two or more waves of data, where at each wave the subject provided at least two smoking events. In all, there were 130 such subjects with data from a total of 3,388 smoking events. Of these, 47, 39, and 44 subjects provided data at two, three, and four waves, respectively. The number of subjects at each measurement wave equaled 116 (baseline), 91 (6 months), 92 (15 months), and 88 (24 months), and the average number of smoking events equaled 7.14 (range = 2–42), 7.65 (range = 2–32), 9.97 (range = 2–43), and 10.76 (range = 2–49) at these same four waves, respectively.

Measures

Negative and Positive Affect

Two mood outcomes were considered: measures of the subject's negative and positive affect (denoted NA and PA, respectively) at a smoking episode. Both of these measures consisted of the average of several individual mood items, each rated from 1 to 10, with "10" representing very high levels of the attribute that were identified via factor analysis. Specifically, PA consisted of the following items that reflected subjects' assessments of their positive mood: I felt happy, I felt relaxed, I felt cheerful, I felt confident, and I felt accepted by others. Similarly, NA consisted of the following items: I felt sad, I felt stressed, I felt angry, I felt frustrated, and I felt irritable. For the smoking events, participants rated their mood "before" and "now after smoking." Considering the five items of the "before" (and "now after smoking") PA mood assessments, Cronbach's alpha was equal to .84 (.77), .81 (.78), .85 (.83), and .83 (.82) at baseline, 6, 9, and 24 months, respectively. Similarly, in terms of the NA mood assessments, Cronbach's alpha equaled .90 (.90), .92 (.91), .88 (.91), and .93 (.90) at baseline, 6, 9, and 24 months, respectively. Because of our interest in smoking-related mood change, we used the difference (now – before) as our measure of reported mood change associated with smoking.

Gender and Wave

As covariates, we considered gender and measurement wave with the variables `Male` (coded 0 = female or 1 = male) and `Wave` (coded 0 = baseline, 1 = 6 months, 2.5 = 15 months, and 4 = 24 months). In our selected sample of 130 subjects, 46% were males.

Smoking Level

As a time-varying (within-subjects) measure of a subject's smoking level, we used the number of smoking events that a subject reported at a given measurement wave (denoted as `NumSmk`). To separate the between- and within-subjects effects of this time-varying variable on mood change, as described in Begg and Parides (2003), we also included the subject's mean of `NumSmk` as a covariate (denoted as `AvgSmk`). By including both the wave-varying `NumSmk` and the subject-varying `AvgSmk`, we can estimate, respectively, both the within-subjects and between-subjects effects of smoking level on mood change. The between-subject effect represents the association of a person's average smoking level with their average change in mood (both averages being taken over time). Conversely, the within-subjects effect indicates how a person's mood change differs as their level of smoking varies over waves. The latter is of primary interest here as it represents the degree to which a person's mood response to smoking (now – before) changes as their smoking level varies across time. Finally, because of the relatively large numerical range of these variables, to ease computation and interpretation, we divided both by a factor of 10 so that the coefficients of these variables represent changes attributable to 10 smoking events (rather than a single smoking event). Also, to increase the interpretation of the intercept-related parameters we centered these two smoking-level variables at the value of 10 smoking reports.

DATA ANALYSIS

Consider the following mixed-effects regression model for the measurement y, either smoking-related change in NA or PA, of subject i ($i = 1, 2, \ldots, N$ subjects) on occasion j ($j = 1, 2, \ldots, n_i$ occasions):

$$y_{ij} = (\beta_0 + \upsilon_{0i}) = (\beta_1 + \upsilon_{1i})\texttt{Wave}_{ij} + \beta_2\texttt{Male}_i + \beta_3\texttt{AvgSmk}_i + \beta_4\texttt{NumSmk}_{ij} + \varepsilon_{ij} \quad (3.1)$$

Here, the occasions refer to the multiple smoking events that a subject provides, which, based on our inclusion criteria, are obtained at two or more measurement waves for each subject. The random subject effect υ_{0i} indicates the influence of individual i on his or her mood change at baseline, while υ_{1i} represents how a subject's mood change varies over time. Both of these reflect individual deviations relative to the population intercept and slope, β_0 and β_1. The inclusion of the random slope υ_{1i} is important here because the data are collected across multiple waves. With two random subject-specific effects, the population distribution of intercept and slope deviations is assumed to be a bivariate normal $N(0, \Sigma_\upsilon)$, where Σ_υ is the 2×2 variance–covariance matrix given as:

$$\Sigma_\upsilon = \begin{bmatrix} \sigma^2_{\upsilon_0} & \sigma_{\upsilon_0\upsilon_1} \\ \sigma_{\upsilon_0\upsilon_1} & \sigma^2_{\upsilon_1} \end{bmatrix}.$$

The model includes the intercept and `Wave` effect at both the individual (υ_{0i} and υ_{1i}) and population (β_0 and β_1) levels. Thus, we are controlling for baseline mood change and mood change across time at both of these levels. The model additionally includes the subject-varying covariates `Male` and `AvgSmk` to allow for the effects of gender and overall smoking level on mood change. The final regressor, `NumSmk`, which varies within subjects and across waves, represents the within-subjects effect of smoking level; the effect of this variable indicates the degree to which smoking-related mood change (now – before) varies as a subject changes his or her smoking level across time.

The errors ε_{ij} are assumed to be normally distributed in the population with zero mean and variance σ^2_ε, and independent of the random effects. Here, σ^2_ε is the within-subjects (WS) variance, which indicates the degree of variation in mood change within a subject. Because our interest is in allowing covariates to influence mood change variation, in addition to the effects on the mean level of mood change, we posit the following log-linear model of the WS variance:

$$\sigma^2_{\varepsilon_{ij}} = \exp(\mathbf{w}'_{ij}\boldsymbol{\tau}). \tag{3.2}$$

This type of log-linear representation is common in the context of heteroscedastic (fixed-effects) regression models (Aitkin, 1987; Davidian & Carroll, 1987; Harvey, 1976). The WS variance is subscripted by i and j to indicate that it varies depending on the values of the covariates in vector $\mathbf{w}_{ij}$ (and their coefficients). The number of parameters associated with these variances does not vary with i or j. The covariate vector $\mathbf{w}_{ij}$ includes a (first) column of ones for the reference WS variance (τ_0); the WS variance equals $\exp(\tau_0)$ when the covariates $\mathbf{w}_{ij}$ equal 0, and is increased or decreased as a function of these covariates and their coefficients $\boldsymbol{\tau}$. For a particular covariate w^*, if $\boldsymbol{\tau}^* > 0$, then the WS variance increases as w^* increases (and vice versa if $\boldsymbol{\tau}^* < 0$). Note that the exponential function ensures a positive multiplicative factor for any finite value of τ, and so the resulting variance is guaranteed to be positive.

As in Hedeker et al. (2008), the WS variance can vary across subjects, above and beyond the contribution of covariates, namely,

$$\sigma^2_{\varepsilon_{ij}} = \exp(\mathbf{w}'_{ij}\boldsymbol{\tau} + \omega_i), \tag{3.3}$$

where the random subject (scale) effects ω_i are distributed in the population of subjects with mean 0 and variance σ^2_ω. The idea for this is akin to the inclusion of the random (location) effects in equation 3.1, namely, covariates do not account for all of the reasons that subjects differ from each other. The parameters υ_{0i} and υ_{1i} in equation 3.1 indicate how subjects differ in terms of their means and the ω_i parameters in equation 3.3 indicate how subjects differ in variation, beyond the effect of covariates. Notice that taking logs in equation 3.3 yields $\log(\sigma^2_{\varepsilon_{ij}}) = \mathbf{w}_{ij}\boldsymbol{\tau} + \omega_i$, which indicates that if the distribution of ω_i is specified as normal, the random scale effects serve as log-normal subject-specific perturbations of the WS variance. The skewed, nonnegative nature of the log-normal distribution makes it a useful choice for representing variances and it has been used in many diverse research areas for this purpose (Fowler & Whitlock, 1999; Leonard, 1975; Reno & Rizza, 2003; Shenk, White, & Burnhamb, 1998; Vasseur, 1999).

In this model, υ_{0i} and υ_{1i} are random effects that influence an individual's mean, or location, and ω_i is a random effect that influences an individual's variance, or (square of the) scale. Thus, the model with both types of random effects is called a *mixed-effects location scale model.* These three random effects are all allowed to be correlated with covariance parameters $\sigma_{\upsilon_0\upsilon_1}$ (intercept and slope), $\sigma_{\upsilon_0\omega}$ (intercept and scale), and $\sigma_{\upsilon_1\omega}$ (slope and scale). Details on model estimation can be found in Hedeker et al. (2008). A nice feature of the model is that standard software (i.e., SAS PROC NLMIXED) can be used for estimation, and we provide sample computer syntax in the Appendix.

Visually, Figure 3.1 presents the model for EMA data without the error variance model (i.e., only equation 3.1), while Figure 3.2 illustrates the addition of the error variance model and random scale effects (i.e., equations 3.1 and 3.3). These figures present artificial data in order to better highlight and describe the model features. In both figures, the average across all subjects is depicted with solid horizontal lines, and the lines of two subjects are presented as dotted horizontal lines. In a given dataset, there will be dotted lines for each subject, but for simplicity here we only plot two representative subjects. Also, for simplicity, only two waves of data are plotted. The slanted solid lines represent the population time-trends (averaged over

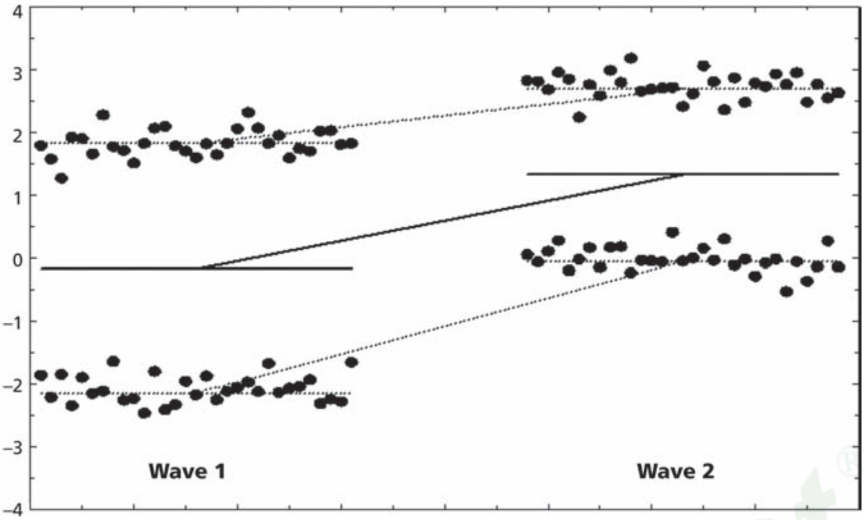

Figure 3.1 Mixed-effects model for longitudinal EMA data across two waves. Mean trend (solid lines), subject trends (dotted lines), and EMA observations (dots) for two representative subjects.

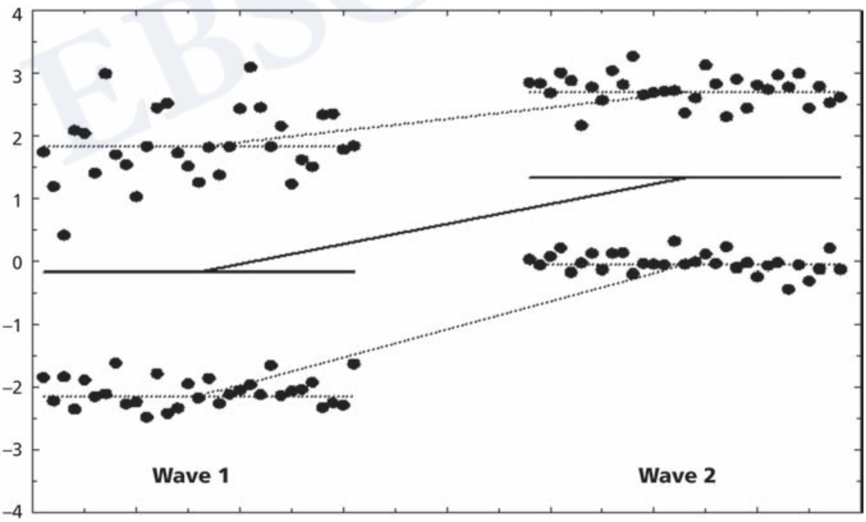

Figure 3.2 Mixed-effects location scale model for longitudinal EMA data across two waves. Mean trend (solid lines), subject trends (dotted lines), and EMA observations (dots) for two representative subjects.

subjects), while the slanted dotted lines represent the time-trends for the two subjects. Data points for these two subjects are also included in the plot; these represent the outcomes for a subject at a wave.

Considering Figure 3.1 first, the solid horizontal line at Wave 1 (i.e., baseline) corresponds to the population intercept and the slanted solid line is the population slope (β_0 and β_1, respectively). Covariates (besides Wave) can affect this mean response by either raising or lowering the slanted solid line (main effect) or change its slope (time interaction) in the usual way. The dotted lines of the two subjects at Wave 1 are indicative of a person's random intercept υ_{0i}, which indicates how a subject deviates from the mean response at Wave 1. In the figure, one subject is above and another subject is below the mean line. The heterogeneity in these dotted lines at Wave 1 is indicative of BS intercept variance ($\sigma^2_{\upsilon_0}$): if the dotted lines are close together then there is not much subject heterogeneity; conversely, if the dotted lines are spread out, then more heterogeneity is indicated. Similarly, the heterogeneity of the dotted trend lines is reflected by the BS slope variance ($\sigma^2_{\upsilon_1}$); in the plot these slopes vary between the two subjects, which corresponds to the notion that subjects vary in their time trajectories. Finally, the degree of variation of a person's data points around each of their horizontal dotted lines is the WS variance (σ^2_ε). In Figure 3.1, as in a standard mixed model, this is the same for all subjects at all waves.

Figure 3.2 illustrates the concept of the random scale effect and error variance modeling inherent in equation 3.3. Notice that the dispersion of the observations around each of the horizontal dotted lines varies. At both Waves 1 and 2, there is a great deal more WS variance for the subject above the mean line, relative to the subject below it. This disparate WS variation across subjects is precisely what the random scale effect ω_i captures, and the variance associated with this random effect (σ^2_ω) indicates the degree of subject heterogeneity in the WS variance. Notice also that the WS variance for both subjects is lessened at Wave 2 relative to Wave 1. This illustrates the effect that covariates $\mathbf{w}$ (and their coefficients $\boldsymbol{\tau}$) can have on the WS variance expressed in equation 3.3. As illustrated, the coefficient for `Wave` would be negative (i.e., as `Wave` increases, WS variance diminishes). Covariates of the WS variance could be occasion-varying like `Wave`, or subject-varying like `Male`, in which case the WS variation (across all occasions) of males would be more/less relative to females.

Second and/or Third Thoughts

The two-level model in equation 3.1 treats all observations at level 1 as nested within subjects at level 2. However, the observations are obtained across several measurement waves, and so a three-level structure of observations (level 1) within waves (level 2) within subjects (level 3) would seem more appropriate. For this, consider the multilevel decomposition of the model, where, for simplicity, we only include the covariate `Wave` (here, $i = 1, 2, \ldots, N$ subjects; $j = 1, 2, \ldots, n_i$ waves; and $k = 1, 2, \ldots, n_{ij}$ observations within a wave j for subject i):

Level 1 (within subjects, within waves)

$$y_{ijk} = b_{0ij} + \varepsilon_{ijk} \tag{3.4}$$

Level 2 (within subjects, between waves)

$$b_{0ij} = b_{0i} + b_{1i}\texttt{Wave}_{ij} + [\upsilon_{0ij}] \tag{3.5}$$

Level 3 (between subjects)

$$b_{0i} = \beta_0 + \upsilon_{0i} \tag{3.6}$$
$$b_{1i} = \beta_1 + \upsilon_{1i}$$

Here, b_{0ij} represents the subject means across time (i.e., the averages for a subject of the observations at a particular wave), and b_{0i} and b_{1i} are the subject intercepts and time trends of these means, respectively. The parameters in equation 3.6 of the level-3 model are as described above. In the proposed two-level model in equation 3.1, we have not included a wave-specific random effect, which is specified in equation 3.5 in brackets as $[\upsilon_{0ij}]$ for emphasis. Notice that without this term in the model, one is assuming that the subject means across time follow a line perfectly. This would seem to be a rather stringent assumption for subjects with more than two waves of data.

To test this assumption, therefore, we also estimated three-level models that include a random wave effect, in addition to the random subject effects. The three-level models also incorporate the modeling of the error variance given in equation 3.3. Namely, our full three-level mean model is specified as:

$$y_{ijk} = (\beta_0 + \upsilon_{0i} + \upsilon_{0ij}) + (\beta_1 + \upsilon_{1i})\texttt{Wave}_{ij} + \beta_2\texttt{Male}_i + \beta_3\texttt{AvgSmk}_i + \beta_4\texttt{NumSmk}_{ij} + \varepsilon_{ijk} \tag{3.7}$$

with the corresponding error variance model as:

$$\sigma^2_{ijk} = \exp(\tau_0 + \tau_1\texttt{Wave}_{ij} + \tau_2\texttt{Male}_i + \tau_3\texttt{AvgSmk}_i + \tau_4\texttt{NumSmk}_{ij} + \omega_i). \tag{3.8}$$

The random effects at the same level are allowed to be correlated; however, random effects at different levels are independent. Thus, the subject-level random scale effect and subject-level random location effects are correlated, but the random wave effect is independent.

RESULTS

First, to get a sense of the data, Table 3.1 lists the results of wave-stratified random intercept models of PA and NA, treating observations nested within subjects, namely, $y_{ij} = \beta_0 + \upsilon_{0i} + \varepsilon_{ij}$. The intercept of this model reflects the dependent variable mean, adjusting for the different numbers of observations per subject. Similarly, the variances are separated in terms of the between- and within-subjects components. As can be seen, the positive affect means are all positive, while the negative affect means are all negative, indicating the mood benefit attributed to smoking (both of these variables are mood assessments of now – before smoking). The benefit to positive affect does seem to diminish somewhat over time. What is also apparent is the general decline across time in both the between- and within-subjects variances.

Next, Table 3.2 lists the results of several two- and three-level models of smoking-related changes in positive and negative affect. For each of the models, the number of variance-covariance parameters, deviance (–2 log likelihood value), and Akaike information criterion (AIC) are provided. All models included the variables `Wave`, `Male`, `AvgSmk`, and `NumSmk` as regressors in both the mean and error variance structure.

The first two rows in this table are for the two-level models (labeled 2a–2b), while the remaining four rows are for three-level models (labeled 3a–3d). Computational issues arose for two of the three-level models, in that either a random effect correlation equaled unity in absolute value (model 3b), or

TABLE 3.1 Smoking-Related Positive and Negative Affect Change: Wave-Stratified Model-Based Descriptive Results

			Positive affect			Negative affect		
			Mean	BS var	WS var	Mean	BS var	WS var
Wave	***N***	$\sum_i n_i$	$\hat{\beta}_0$	$\hat{\sigma}^2_\upsilon$	$\hat{\sigma}^2_\varepsilon$	$\hat{\beta}_0$	$\hat{\sigma}^2_\upsilon$	$\hat{\sigma}^2_\varepsilon$
Baseline	116	828	0.730	0.792	2.240	–0.439	0.902	2.495
6 months	91	696	0.538	0.371	2.020	–0.445	0.350	2.399
15 months	92	917	0.353	0.457	1.574	–0.318	0.380	1.771
24 months	88	947	0.404	0.243	1.460	–0.391	0.267	1.507

Note: N equals the total number of subjects, $\sum_i n_i$ equals the total number of observations.

TABLE 3.2 Smoking-Related Positive and Negative Affect Change: Two- and Three-Level Model Results (Deviance and AIC Values)

Model	Subject-level random	Wave-level random	Variance-covariance parameters	Positive affect		Negative affect	
				Deviance	AIC	Deviance	AIC
2a	I, W		3	11763	11789	11999	12025
2b	I, W, S		6	11246	11278	11154	11186
3a	I	I	2	11756	11780	11997	12021
3b	I, W	I	4	I,W correlation = –1			
3c	I, S	I	4	11228	11256	11150	11178
3d	I, W, S	I	7	W variance = 0			

Note: I, intercept, W, wave, S, scale; Deviance = –2 log likelihood; AIC: Akaike information criterion. Regressors = `Wave`, `Male`, `AvgSmk`, `NumSmk` in both mean and error variance models.

a random effect variance equaled zero (model 3d). These computational issues occurred for both positive and negative affect. Of the remaining models, there is clear evidence for the three-level models, and also for the models including random scale effects. Thus, the model of choice is the three-level random scale model (model 3c). Estimates from this model are provided in Table 3.3.

In terms of the mean model, the intercept is highly significant for both mood change outcomes: positive for `PA` ($\hat{\beta}_0 = .547, p < .0001$) and negative for `NA` ($\hat{\beta}_0 = -.339, p < .0001$). This indicates that smoking had a beneficial effect by increasing positive affect change and decreasing negative affect change (when the covariates all equal 0, or for an average female at baseline with 10 smoking events). `Wave` had a diminishing effect on smoking-related `PA` mood change ($\hat{\beta}_1 = -.059, p < .005$), but no significant effect on `NA`. Namely, as time increased the smoking-related benefit to positive affect change decreased, while the negative affect change remained. Neither gender nor smoking level significantly influenced smoking-related mood change.

In terms of the error variance, `Wave` has a consistent significant effect in reducing variation for both `PA` change ($\hat{\tau}_1 = -.124, p < .0001$) and `NA` change ($\hat{\tau}_1 = -.095, p < .0001$); the variation in smoking-related mood change diminished across time. The BS effect of smoking level (`AvgSmk`) also significantly reduced variation in smoking-related `PA` mood change ($\hat{\tau}_3 = -.259, p < .018$). Thus, subjects who, on average, smoke more across time also exhibit less variation in smoking-related positive affect change, averaged across time. Controlling for these effects, the WS effect of smoking (`NumSmk`) significantly reduced variation in smoking-related mood change of both `PA` ($\hat{\tau}_4 = -.080, p < .046$) and especially `NA` ($\hat{\tau}_4 = -.220, p < .0001$). Thus, controlling for the effect of time and the between-subjects effect of

TABLE 3.3 Smoking-Related Positive and Negative Affect Change Estimates, Standard Errors (SE), and *p*-Values[a]

	Positive affect			Negative affect		
	Estimate	**SE**	***p*** **<**	**Estimate**	**SE**	***p*** **<**
Mean model						
Intercept β_0	0.547	0.078	0.0001	–0.339	0.064	0.0001
`Wave` β_1	–0.059	0.020	0.005	0.025	0.017	0.14
`Male` β_2	0.112	0.099	0.27	–0.114	0.079	0.15
`AvgSmk` β_3	–0.111	0.077	0.16	0.016	0.063	0.81
`NumSmk` β_4	–0.042	0.045	0.36	0.034	0.039	0.38
Error variance model						
Intercept τ_0	0.654	0.111	0.0001	0.650	0.152	0.0001
`Wave` τ_1	–0.124	0.020	0.0001	–0.095	0.021	0.0001
`Male` τ_2	0.217	0.151	0.16	0.166	0.214	0.44
`AvgSmk` τ_3	–0.259	0.107	0.018	–0.198	0.145	0.19
`NumSmk` τ_4	–0.080	0.040	0.046	–0.220	0.042	0.0001
Random effect (co)variances						
Subject intercept $\sigma^2_{\upsilon(3)}$	0.162	0.041	0.001	0.082	0.027	0.004
Subject scale $\sigma^2_{\omega(3)}$	0.560	0.091	0.0001	1.28	0.188	0.0001
Subject int,scale $\sigma_{\upsilon\omega(3)}$	0.139	0.041	0.001	–0.204	0.048	0.0001
Wave intercept $\sigma^2_{\upsilon(2)}$	0.071	0.024	0.004	0.033	0.017	0.06

[a] *p*-values are based on Wald statistics (Estimate/SE ~ standard normal distribution).

smoking level, as a person increases his or her level of smoking across time the variation in his or her smoking-related mood change is reduced.

Turning to the variance estimates, the subject-level intercept variance is seen to be significant for both outcomes ($\hat{\sigma}^2_{\upsilon(3)} = .162, p < .001$ for `PA`, $\hat{\sigma}^2_{\upsilon(3)} = .082, p < .004$ for `NA`); subjects do vary in their levels of smoking-related mood changes. The wave-level variance is observed to be significant for `PA` ($\hat{\sigma}^2_{\upsilon(2)} = .071, p < .004$), and near-significant for `NA` ($\hat{\sigma}^2_{\upsilon(2)} = .033, p < .06$), indicating that the data from subjects within a wave are also correlated, over and above the overall subject effect. The subject-level scale variance is observed to be highly significant for both outcomes ($\hat{\sigma}^2_{\omega(3)} = .560, p < .0001$ for `PA`, $\hat{\sigma}^2_{\omega(3)} = 1.28, p < .0001$ for `NA`), which indicates the importance of including the random subject scale effect. Subjects clearly vary in terms of the within-subject within-wave variance (over and above the influence of the covariates in the error variance model). In terms of the covariance, for both outcomes, the association of the random subject intercept and scale terms is seen to be significant. For `PA` it is positive ($\hat{\sigma}_{\upsilon\omega(3)} = .139, p < .001$), which indicates that subjects with higher positive affect levels (in terms of smoking-related mood change) have greater mood (change) variation. Conversely, for NA this covariance is negative ($\hat{\sigma}_{\upsilon\omega(3)} = -.204, p < .0001$), which suggests that subjects with higher negative affect levels (smoking-related mood change) exhibit less mood (change) variation. Expressed as correlations, these are .47 for PA and –.63 for NA. As the outcomes are change scores, these suggest that as the change score levels go toward zero (lower PA change and higher NA change), the scale variance is reduced. It is worth noting that zero is not a boundary value for these change scores, which varied from –9 to 9, and so these correlations do not necessarily reflect a floor effect of measurement.

DISCUSSION

This chapter has illustrated how mixed models for EMA data can be used to model differences in WS variances, and not just means. As such, these models can help to identify predictors of within-subjects variation, and to test psychological hypotheses about these variances. While estimation of the model goes beyond standard mixed model software (e.g., SAS PROC MIXED, SPSS MIXED, HLM, MLwiN, SuperMix), SAS PROC NLMIXED can be used for this purpose. In the Appendix, we provide sample syntax for maximum likelihood estimation of our mixed location scale models, making this class of models accessible to researchers.

Here, we focused on the degree of change in mood variation associated with smoking events (now – before), and whether covariates influenced this variation among adolescent smokers. One of the key concepts in dependence is the development of tolerance, or the diminishing of effects of a substance with continued use. A common experience reported by both adults and adolescents is mood change after smoking a cigarette, and the equally common notion is that these subjective feelings diminish over time as one's experience with smoking increases and tolerance may develop. However, heretofore, researchers have examined changes in these subjective experiences primarily through paper-and-pencil, retrospective questionnaire reports. Thus, it has been difficult to document adequately whether adolescents experience mood changes with smoking and also how symptoms of dependence develop or with what level of smoking experience. Overall, following smoking, adolescents reported higher positive affect and lower negative affect than before their smoking report. Additionally, our analyses indicated an increased consistency of subjective mood responses as a person's smoking experience increased over time and a diminishing of the mood change associated with smoking. Our data thus provide one of the few ecologically valid examinations of the development of tolerance.

Our study is one of the first to examine real-time subjective mood responses to smoking among adolescents who are still relatively early in their smoking careers and light or infrequent smokers (less than 9% of the sample smoked more than five cigarettes a day). As such, this study helps to add important information about the relatively early development of symptoms of dependence, a potential development of tolerance to the mood-regulating effects of smoking.

More potential applications of this class of models clearly exist in substance abuse and psychological research. For example, many questions of both normal development and the development of psychopathology address the issue of variability or stability in emotional responses to various situations and contexts. Often, an interest is with the variability of responses an individual gives to a variety of stimuli or situations, and not just with the overall mean level of responsivity. The models presented here also allow us to examine hypotheses about consistency of responses as well.

In order to reliably estimate variances, and the effects of covariates on these variances, a fair amount of both within-subjects and between-subjects data is required. Modern data collection procedures, such as EMA and real-time data captures, provide this opportunity. These procedures follow the "bursts of measurement" approach described by Nesselroade (1991). As noted by Nesselroade, such bursts of measurement increase the research burden in several ways; yet they are necessary for studying individual variation and allow researchers to examine important questions that were previously unanswerable. Along with these modern data collection procedures, it is useful to have statistical models that can effectively analyze the unique features of these datasets. Hopefully, this chapter has provided models for this purpose.

APPENDIX

Below are syntax samples for the two- and three-level mixed-effects location scale models. Expressions with all uppercase letters denote SAS-specific syntax, while expressions including lowercase letters are for user-defined entities. The dependent variable `NAchange` is the change in negative affect associated with a smoking event (now – before) and, for simplicity, we only consider the covariate `Wave`. The variable `id` is a subject-level identifier. For the random subject effects, `u0` is the intercept and `u1` is the trend across waves ("u" is used for the Greek upsilon of our equations), while `omega` is for the random scale effect.

The mean response model is given by `mean`, with regression coefficients named `b0` and `bWave`. The model for the within-subjects (error) variance is denoted `vare`, with `t0` for the reference variance (i.e., the variance when

Wave equals 0), in natural log units, and tWave as the coefficient for Wave. Finally, v0, v1, and vs represent the variances of the two random location and one random scale effects, with covariances c01, c0s, and c1s.

```
PROC NLMIXED GCONV=1e-12;
PARMS b0=-.3 bWave=.01 t0=.6 tWave=-.1
      v0=.2 v1=.1 vs=.005 c01=0 c0s=0 c1s=0;
mean = (b0 + u0) + (bWave + u1)*Wave;
vare = EXP(t0 + tWave*Wave + omega);
MODEL NAchange ~ NORMAL(mean,vare);
RANDOM u0 u1 omega ~ NORMAL([0,0,0], [v0,c01,v1,c0s,c1s,vs])
      SUBJECT=id;
```

Users must provide starting values for all parameters on the PARMS statement. To do so, it is beneficial to run the model in stages using estimates from a prior stage as starting values and setting the additional parameters to zero or some small value. For example, one can start by estimating a random-trend model using standard mixed model software to yield starting values for the fixed effects (β), random intercept variance (v0), random trend variance (v1), intercept-trend covariance (c01), and error variance (t0). Then, one can add covariates to the error variance model, perhaps one at a time, with starting values of zero. Finally, the full model with the parameters associated with the random scale effect (vs, c0s, c1s) can be estimated. In practice, this approach works well with PROC NLMIXED, which sometimes has difficulties in converging to a solution for complex models. Also, in our experience, it seems that specifying a small starting value for the random scale effect variance (vs) helps model convergence. Furthermore, for complex models, it is sometimes the case that the default convergence criterion is not strict enough. In the above syntax, the convergence criterion is specified as GCONV=1e-12 on the PROC NLMIXED statement. The results in this chapter did change a bit as this stricter criterion was applied, relative to the default specification; however, the results did not change beyond this level. It would seem that this level is reasonable; however, it probably should be examined on a case-by-case basis.

Three-Level Extension

PROC NLMIXED is set up for two-level models; however, it can be used for three-level analysis in some situations. Li (2010) developed a recursive conditional likelihood approach that can be used for this purpose. An alternative approach was described by Dale McLerran in a Web post at http://listserv.uga.edu/cgi-bin/wa?A2=ind0506b&L=sas-l&F=&S=&P=55. For the current example, we created four indicator variables for the four measurement waves, w1, w2, w3, and w4. These are then included in the mean model and specified as random effects (named below as d1, d2, d3, d4) with mean zero. Furthermore, they are constrained to have the same variance (vwave), and to be independent of each other and the subject random effects.

```
PROC NLMIXED GCONV=1e-12;
PARMS b0=-.3 bWave=.01 t0=.6 tWave=-.1
      v0=.2 v1=.1 vs=.005 vwave=.1 c01=0 c0s=0 c1s=0;
mean = (b0 + u0) + (bWave + u1)*Wave
       + d1*w1 + d2*w2 + d3*w3 + d4*w4;
vare = EXP(t0 + tWave*Wave + omega);
MODEL NAchange ~ NORMAL(mean,vare);
RANDOM u0 u1 omega d1 d2 d3 d4 ~ NORMAL([0,0,0,0,0,0,0],
       [v0,c01,v1,c0s,c1s,vs,
        0, 0, 0, vwave,
        0, 0, 0, 0, vwave,
        0, 0, 0, 0, 0, vwave,
        0, 0, 0, 0, 0, 0, vwave]) SUBJECT=id;
```

As noted by McLerran, the feasibility of this approach depends on the size of the problem because the number of random effects in the model can greatly increase the computational demands. In our case, with only four waves, including the random wave effect was not problematic, in and of itself. However, as noted in the chapter, once the random wave effect was included (vwave), the random subject wave variance (v1) went to zero, and so was removed from the final model.

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REFERENCES

Aitkin, M. (1987). Modeling variance heterogeneity in normal regression using GLIM. *Applied Statistics, 36,* 332–339.

Begg, M. B., & Parides, M. K. (2003). Separation of individual-level and cluster-level covariate effects in regression analysis of correlated data. *Statistics in Medicine, 22,* 2591–2602.

Bolger, N., Davis, A., & Rafaeli, E. (2003). Diary methods: capturing life as it is lived. *Annual Review of Psychology, 54,* 579–616.

Chassin, L., Presson, C. C., Rose, J., & Sherman, S. J. (2007). What is addiction?: Age-related differences in the meaning of addiction. *Drug and Alcohol Dependence, 87,* 30–38.

Chinchilli, V. M., Esinhart, J. D., & Miller, W. G. (1995). Partial likelihood analysis of within-unit variances in repeated measurement experiments. *Biometrics, 51,* 205–216.

Cleveland, W., Denby, L., & Liu, C. (2002). Random scale effects (Bell Labs technical report). Retrieved from *http://cm.bell-labs.com/cm/ms/departments/sia/doc/randomscale.pdf.*

Davidian, M., & Carroll, R. (1987). Variance function estimation. *Journal of the American Statistical Association, 82,* 1079–1091.

Feldman Barrett, L., & Barrett, D. (2001). An introduction to computerized experience sampling in psychology. *Social Science Computer Review, 19,* 175–185.

Fowler, K., & Whitlock, M. C. (1999). The distribution of phenotypic variance with inbreeding. *Evolution, 53,* 1143–1156.

Harvey, A. C. (1976). Estimating regression models with multiplicative heteroscedasticity. *Econometrica, 44,* 461–465.

Hedeker. D. & Gibbons, R.D. (2006). *Longitudinal data analysis.* New York: Wiley.

Hedeker, D., Mermelstein, R. J., Berbaum, M. L., & Campbell, R. T. (2009). Modeling mood variation associated with smoking: An application of a heterogeneous mixed-effects model for analysis of ecological momentary assessment (EMA) data. *Addiction, 104,* 297–307.

Hedeker, D., Mermelstein, R. J., Demirtas, H. (2008). An application of a mixed-effects location scale model for analysis of Ecological Momentary Assessment (EMA) data. *Biometrics, 64,* 627–634.

Hertzog, C., & Nesselroade, J. R. (2003). Assessing psychological change in adulthood: an overview of methodological issues. *Psychology and Aging, 18,* 639–657.

Jahng, S., Wood, P. K., & Trull, T. J. (2008). Analysis of affective instability in ecological momentary assessment: Indices using successive difference and group comparison via multilevel modeling. *Psychological Methods, 13,* 354–375.

James, A. T., Venables, W. N., Dry, I. B., & Wiskich, J. T. (1994). Random effects and variances as a synthesis of nonlinear regression analysis of mitochondrial electron transport. *Biometrika, 81,* 219–235.

Kassel, J. D., & Hankin, B. L. (2006). Smoking and depression. In A. Steptoe (Ed.), *Depression and physical illness* (pp. 321–347). Cambridge, UK: Cambridge University Press.

Kassel, J. D., Stroud, L. R., & Paronis, C. A. (2003). Smoking, stress, and negative affect: Correlation, causation, and context across stages of smoking. *Psychological Bulletin, 129,* 270–304.

Khantzian, E. J. (1997). The self-medication hypothesis of substance use disorders: A reconsideration and recent applications. *Harvard Review of Psychiatry, 4,* 231–244.

Laird, N. M. (1988). Missing data in longitudinal studies. *Statistics in Medicine, 7,* 305–315.

Leckie, G. (2010). *Modelling the variance in multilevel models.* Colloquium presentation, Institute for Health Research and Policy, University of Illinois at Chicago.

Leonard, T. (1975). A Bayesian approach to the linear model with unequal variances. *Technometrics, 17,* 95–102.

Li, X. (2010). *A three-level mixed-effects location scale model with an application in ecological momentary assessment data.* Unpublished doctoral thesis, University of Illinois at Chicago, School of Public Health.

Lin, X., Raz, J., & Harlow, S. (1997). Linear mixed models with heterogeneous within-cluster variances, *Biometrics, 53*, 910–923.

Moghaddam, N. G., & Ferguson, E. (2007). Smoking, mood regulation, and personality: An event-sampling exploration of potential models and moderation. *Journal of Personality, 75,* 451–478.

Molenberghs, G. M., Thijs, H., Jansen, I., Beunckens, C., Kenward, M. G., Mallinckrodt, C., et al. (2004). Analyzing incomplete longitudinal clinical trial data. *Biostatistics, 5*, 445–464.

Myles, J. P., Price, G. M., Hunter, N., Day, M., & Duffy, S. W. (2003). A potentially useful distribution model for dietary intake data. *Public Health Nutrition, 6*, 513–519.

Nesselroade, J. R. (1991). The warp and woof of the developmental fabric. In R. Downs, L. Liben, & D. Palarmo (Eds.), *Visions of development, the environment, and aesthetics: The legacy of Joachim F. Wohlwill* (pp. 213–240). Hillside, NJ: Erlbaum.

Penner, L. A., Shiffman, S., Paty, J. A., & Fritzsche, B. A. (1994). Individual differences in intraperson variability in mood. *Journal of Personality and Social Psychology, 66,* 712–721.

Reno, R., & Rizza, R. (2003). Is volatility lognormal?: Evidence from Italian futures. *Physica A: Statistical Mechanics and its Applications, 322,* 620–628.

Rubin, D. B. (1976). Inference and missing data. *Biometrika, 63*, 581–592.

Schwartz, J. E., & Stone, A. (2007). The analysis of real-time momentary data: A practical guide. In: A. A. Stone, S. S. Shiffman, A, Atienza, & L. Nebeling (Eds.), *The science of real-time data capture: Self-report in health research* (pp. 76–113). Oxford, UK: Oxford University Press.

Scollon, C. N., Kim-Prieto, C., & Diener, E. (2003). Experience sampling: promises and pitfalls, strengths and weeknesses. *Journal of Happiness Studies, 4,* 5–34.

Shenk, T. M., White, G. C., & Burnhamb, K. P. (1998). Sampling-variance effects on detecting density dependence from temporal trends in natural populations. *Ecological Monographs, 68,* 445–463.

Smyth, J. M., & Stone, A. A. (2003). Ecological momentary assessment research in behavioral medicine. *Journal of Happiness Studies, 4,* 35–52.

Stone, A., & Shiffman, S. (1994). Ecological momentary assessment (EMA) in behavioral medicine. *Annals of Behavioral Medicine, 16,* 199–202.

Tiffany, S. T., Conklin, C. A., Shiffman, S., & Clayton, R. R. (2004). What can dependence theories tell us about assessing the emergence of tobacco dependence? *Addiction, 99,* 78–86.

Vasseur, H. (1999). Prediction of tropospheric scintillation on satellite links from radiosound data. *IEEE Transactions on Antennas and Propagation, 47,* 293–301.

Walls, T. A., & Schafer, J. L. (2006). *Models for intensive longitudinal data.* New York: Oxford University Press.

Watson, D., & Tellegen, A. (1985). Toward a consensual structure of mood. *Psychological Bulletin, 98,* 219–235.

Watson, D., Wiese, D., Vaidya, J., & Tellegen, A. (1999). The two general activation systems of affect: Structural findings, evolutional considerations, and psychobiological evidence. *Journal of Personality and Social Psychology, 76,* 820–838.

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