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BAYESIAN ANALYSIS OF REPEATED EVENTS USING EVENT-DEPENDENT FRAILTY MODELS: AN APPLICATION TO BEHAVIORAL OBSERVATION DATA

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Abstract

In social interaction studies, one commonly encounters repeated displays of behaviors along with their duration data. Statistical methods for the analysis of such data use either parametric (e.g., Weibull) or semi-nonparametric (e.g., Cox) proportional hazard models, modified to include random effects (frailty) which account for the correlation of repeated occurrences of behaviors within a unit (dyad). However, dyad-specific random effects by themselves are not able to account for the *ordering* of event occurrences within dyads. The occurrence of an event (behavior) can make further occurrences of the same behavior to be more or less likely during an interaction. This paper develops event-dependent random effects models for analyzing repeated behaviors data using a Bayesian approach. The models are illustrated by a dataset relating to emotion regulation in families with children who have behavioral or emotional problems.

Keywords

Bayesian inference; emotion regulation; random effects; social interaction; survival model

1. INTRODUCTION

Repeated events are common in public health, medical and social behavioral studies. In recent years, the increasing availability of sequential behavioral data at the microlevel for social interactions has allowed researchers to model behavioral interaction processes between two or more individuals, such as couples, parents and children, and peers (Lindeboom and Kerkhofs, 2000; Dagne et al., 2002, 2003, 2007). An important feature of social interactions is the repetition of behaviors over time. For example, behavioral displays of anger by school-aged children often reoccur over time, and emotion regulation theory would hypothesize that children with behavioral or emotional problems may have difficulty regulating their anger (Morris, Silk, Steinberg, Myers, and Robinson, 2007). However, most existing models for sequential behavioral data analyses fail to account for correlations caused by *recurrent events* and *event dependence* (Gardener and Griffin, 1989). This failure can lead to misleading results. The purpose of this article is to present a new methodology that takes into account both recurrent (repeated) events and event dependence which are characteristics of social interactions, heart attacks or depression episodes.

There are two sources of correlation in such recurrent events. The first source of correlation is *heterogeneity*. Heterogeneity occurs when some subjects or dyads are more prone to experiencing repeated events than others. For example, parents and children may show substantial between-family differences in the rates at which anger and other emotions are displayed during ongoing family interaction (Snyder, Schrepferman, Brooker, and Stoolmiller, 2007). Heterogeneity across subjects produces within-subject correlation in the durations of recurrent events. Ignoring this source of correlation will result in understating the standard errors of estimates for the effects of covariates (Diggle, Heagerty, Liang, and Zeger, 2002, p. 19).

The second source of correlation in repeated events is *event dependence*. It is usually the case that second and subsequent events are likely to be influenced by the occurrence of the first event, producing event dependence. For example, a prior child display of anger or sadness during parent-child interaction may increase or decrease the probability of a subsequent display of anger or sadness. This dependence can be moderated by the characteristics of the child or the parent. Children characterized by high negative emotionality may display anger for increasingly longer durations during family problem solving with a parent who is hostile. In contrast, a child characterized by low negative emotionality may engage in increasingly shorter displays of anger during family problem solving with a parent who is empathic and supportive. Systematic between-child differences in the duration of emotion displays as those displays accumulate during social interaction directly reflect childrens capacity for emotion regulation at microsocial, real-time level. Such interdependence of recurrent events creates within-subject correlation which needs to be accounted for.

In this article, we present event-dependent frailty models that incorporate both sources of correlation of repeated events. We also investigate various functional forms for gauging the impact of event dependence on hazard rates. To our knowledge, no study has done so using a Bayesian approach which is a better method than the frequentist approach - especially in the case of small sample size (Gelman, Carlin, Stern, and Rubin, 1995). In the next section, the proposed models are developed. Section 3 discusses the estimation of the parameters of the models. In Section 4, the proposed models are applied to a family interaction dataset. Finally, Section 5 presents conclusions drawn from the results of analyses.

2. CONDITIONAL FRAILTY MODEL FOR DURATION DATA

In this section we present a conditional frailty model which separates and accounts for *event dependence* and *heterogeneity* in repeated events data. The model is a duration model based on a finite number of states (e.g., behaviors) according to a continuous-time semi-Markov chain. If the chain is in a certain state, it remains in that state for some duration before moving to another state. Hazard rates are used to characterize these termination or transition processes. The hazard rate measures the instantaneous rate at which a particular event that had lasted time t in an original state was terminated by another event. The hazard rate for a transition from an origin state to a destination after sojourn duration t_{ij} for the i th subject ($i = 1, 2, \dots, m$, where m denotes the total number of subjects or dyads in a sample) at the j th event ($j = 1, \dots, n_i$) is denoted by $h_{ij}(t_{ij})$, and it is given by

$$h_{ij}(t_{ij}) = \lim_{\Delta t_{ij} \rightarrow 0} \frac{P(t_{ij} < T_{ij} < t_{ij} + \Delta t_{ij} | T_{ij} \geq t_{ij})}{\Delta t_{ij}}. \quad (1)$$

The hazard rate function in (1) can be expanded to incorporate both heterogeneity and event dependence as follows.

Heterogeneity

Heterogeneity enters the hazard model in the form of random effects or frailty (Therneau and Grambsch, 2000). The underlying logic behind random effects is that some subjects or dyads have more propensity to experiencing recurrent events than others, inducing heterogeneity. The distribution of these random effects is assumed to be normal with mean zero and variance τ^2 , and we denote them by u_i for the i th subject. This specification gives a Gaussian frailty model. It is to be noted that even though the random effects models deal with heterogeneity, they ignore the ordering of event occurrences within dyads. Heterogeneity is to between subjects and event dependence is to within subjects variation.

Event dependence

Event dependence enters the hazard model by allowing the baseline hazard function to vary by event. The reason for making the baseline hazard function to vary by event is that the occurrence of one event may have a potential to make the occurrences of future events more or less likely. That is, the hazard function for an event is a function of the occurrence of previous bouts, implying event dependence. If the baseline hazard function is the same for all events then there is no event dependence, which is a case in a standard frailty model. To account event dependence, therefore, the hazard is allowed to vary as some function of the order of event j . For example, one way of expressing event dependence is $h_{oj}(t_{ij}) = jh_0(t_{ij})$. One can think of this as a baseline hazard that changes after each event, producing different and correlated event specific baseline hazards.

In this paper, we study various functional forms for the *event dependence* by setting the baseline hazard function as

$$h_{oj}(t_{ij}) = g(j)h_{0i}(t_{ij}) \quad (2)$$

where

$$g(j) = \begin{cases} \eta_1 j \\ \eta_2 \frac{1}{j} \\ \eta_3 \frac{1}{j^2} \\ \exp(\delta_1 j) \\ \exp(\delta_2 \frac{1}{j}) \\ \exp(\delta_3 \frac{1}{j^2}) \end{cases} \quad (3)$$

where j is the order of event occurrences; η 's and δ 's measure the effect of the order of event occurrences on the hazard function when the event dependence is expressed as a linear or exponential function, respectively. The second factor in (2) is $h_{0i}(t_{ij})$ which is the baseline hazard function. We assume that it has a Weibull distribution which is by far the most frequently used distribution in event history or survival analysis (Blossfeld and Gotz, 1995). That is,

$h_{0i}(t_{ij}) = \gamma t_{ij}^{\gamma-1}$ which is monotone in duration t_{ij} such that if $\gamma < 1$ it is decreasing, if $\gamma > 1$ increasing, and reduces to exponential hazard if $\gamma = 1$.

Incorporating event dependence in the hazard model may give a better fit, and we intend to study what functional forms can be used for such an event dependence by comparing the goodness of fits of those proposed functions given in (3) above.

Next, assuming random effects and event dependence operate multiplicatively on the baseline hazard and using (2), the Weibull conditional hazard frailty model is written as

$$\begin{aligned} h_{ij}(t_{ij}|u, \beta, X) &= h_{0ij}(t_{ij}) \exp(X_i \beta + u_i), \\ &= g(j) h_{0i}(t_{ij}) \exp(X_i \beta + u_i) \\ &= g(j) \gamma t_{ij}^{\gamma-1} \exp(X_i \beta + u_i) \\ &= \gamma t_{ij}^{\gamma-1} \exp(X_i \beta + u_i + \log(g(j))) \end{aligned} \quad (4)$$

and the associated Weibull probability distribution of t is

$$f_{ij}(t_{ij}) = \gamma t_{ij}^{\gamma-1} \exp\{X_i \beta + u_i + \log(g(j)) - \exp(X_i \beta + u_i + \log(g(j))) t_{ij}^\gamma\} \quad (5)$$

where u_i is a random effect or frailty for accounting heterogeneity among individuals and it is assumed to have a normal distribution with mean zero and variance τ^2 ; $h_{0ij}(t_{ij})$ is an event-specific baseline hazard function defined in (2); X is a p -dimensional vector of covariates, such that $\exp(X_i \beta)$ is the relative risk associated with the covariates, and t is a duration time or gap time.

The conditional frailty hazard model given in (4) incorporates both heterogeneity and event dependence and estimation of its parameters are described next.

3. ESTIMATION

A Bayesian estimation method is used to estimate the parameters in (4). The advantage of the Bayesian approach is that it gives not only point estimates of unknown parameters but also the entire posterior distributions for model parameters, making inference easier and more precise than frequentist asymptotic results. Recent advances in Bayesian computation via Markov chain Monte Carlo (MCMC) algorithm enable us to fit the proposed models in a straightforward manner. To implement the MCMC algorithm, a correct specification of the likelihood function for the data to be analyzed and the corresponding prior distributions for the unknown parameters in (4) should be provided. The likelihood function and prior distributions are given in Sections 3.1 and 3.2, respectively.

3.1. LIKELIHOOD FUNCTION

Based on the notations and assumptions given in Section 2 and conditional on the random effects (frailties), the likelihood function becomes

$$\begin{aligned} L(\beta, \delta, \gamma, u|data) &= \prod_{i=1}^m \prod_{j=1}^{n_i} f_{ij}(t_{ij}) \\ &= \prod_{i=1}^m \prod_{j=1}^{n_i} h_{0i}(t_{ij}) \exp\{X_i \beta + u_i + \log(g(j)) - \exp(X_i \beta + u_i + \log(g(j))) t_{ij}^\gamma\} \end{aligned} \quad (6)$$

where $h_{0i}(t_{ij})$ is a Weibull hazard function defined as

$$h_{0i}(t_{ij}) = \gamma t_{ij}^{\gamma-1} \quad (7)$$

3.2. SPECIFYING THE PRIOR DISTRIBUTIONS

We now specify prior distributions for the parameters in (4). We assume normal prior distributions for the fixed parameters, β , with mean β_0 and variance Σ_β which are chosen to make the distribution proper but diffuse with large variances. The parameter of *event dependence*, δ , is normally distributed with mean δ_0 and variance σ_δ^2 . The dyad-specific random effects, u_i , are also assumed to be normally distributed with mean zero and variance τ^2 , where τ^2 has an inverse gamma distribution with parameters a_1 and c_1 . Likewise, γ has a gamma distribution with parameters a_2 and c_2 . These hyper-parameters need to be assessed. For our example (see Section 4), we take $(a_1, c_1) = (.0001, .0001)$ for the prior distribution of τ^{-2} , and $(a_2, c_2) = (1, .0001)$ for γ . $\beta_0 = 0$, $\delta_0 = 0$, and $\Sigma_\beta^{-1} = 1.0E - 3I$, $\sigma_\delta^{-2} = 1.0E - 4$, where I is an identity matrix. We have also tried different values for the hyperparameters to check if the results were sensitive to different choices. All converged to the same results indicating that the data dominated the inference.

3.3. MODEL SELECTION

After fitting the proposed models, we will use a Bayesian model selection procedure to choose a model that fits our data well (see Section 4 for description of the dataset) and the type of functional forms for event dependence. A description of the selection procedure is discussed below.

Suppose a set of K^* models $M_1, \dots, M_{K^*}$ are under consideration with parameter sets $\theta_1, \dots, \theta_{K^*}$, respectively. For two models M_k and M_l in the candidate set, the evidence in favor of M_k as compared to M_l is given by the Bayes factor, denoted by B_{kl} . The Bayes factor is defined by

$$\begin{aligned} B_{kl} &= \frac{f(M_k|y)}{f(M_l|y)} \times \frac{f(M_l)}{f(M_k)} \\ &= \frac{f(y|M_k)}{f(y|M_l)} \\ &= \exp[\log f(y|M_k) - \log f(y|M_l)], \end{aligned} \quad (8)$$

where $f(M_k|y)$ and $f(M_k)$ are the posterior and prior probabilities for the k th model, respectively. $f(y|M_k)$ is the marginal likelihood for model M_k , which is obtained as (Kass and Raftery, 1995)

$$f(y|M_k) = \int L(\theta_k|y, M_k) \pi(\theta_k) d\theta_k, \quad (9)$$

where $L(\theta_k|y, M_k)$ is the likelihood function. Because the Bayes factor is often difficult, especially for models that involve many random effects or large number of parameters, a commonly used alternative is to find an approximation to the Bayes factor. Approximation may be obtained by using either the Laplace method or Bayesian information criterion (BIC) method (Schwarz, 1978).

Using the Laplace method we get,

$$f(y|M_k) \approx (2\pi)^{d_k/2} \left| \sum_k \right|^{1/2} L(\theta_k^*|y, M_k) \pi(\theta_k^*), \quad (10)$$

where θ_k^* is the posterior mode for θ_k , Σ_k is the posterior variance-covariance matrix evaluated at θ_k^* , and d_k is the dimension of θ_k . Whereas, using the Bayes information criterion (BIC), the integrated marginal likelihood for M_k is given by

$$f(y|M_k) \approx n^{-d_k/2} L(\hat{\theta}_k|y, M_k), \quad (11)$$

where $\hat{\theta}_k$ is the maximum likelihood estimate of θ_k , and n is the sample size.

Since WinBUGS does not produce maximum likelihood estimates directly, one can use the MCMC outcomes to approximate maximum log-likelihood (L) from the posterior log-likelihood score (LLS), $\text{Log}L(\hat{\theta}_k|y, M_k)$, which is calculated as the posterior expectation of the log-likelihood function, and $\hat{\theta}_k$ be the posterior mean of θ_k . Let $\tilde{\theta}_k$ be the maximum likelihood estimator of θ_k with variance $V = I^{-1}(\tilde{\theta}_k)$, which is the Fisher information matrix evaluated at $\tilde{\theta}_k$.

Based on the second-order Taylor series expansion, the log-likelihood of the mean of the posterior distribution is approximated by

$$\text{Log}L(\tilde{\theta}_k|y, M_k) \approx \text{Log}L(\hat{\theta}_k|y, M_k) - (\tilde{\theta}_k - \hat{\theta}_k)' V^{-1} (\tilde{\theta}_k - \hat{\theta}_k) / 2. \quad (12)$$

From (12) and assuming large samples, we get

$$-2[\text{Log}L(\tilde{\theta}_k|y, M_k) - \text{Log}L(\hat{\theta}_k|y, M_k)] \approx (\tilde{\theta}_k - \hat{\theta}_k)' V^{-1} (\tilde{\theta}_k - \hat{\theta}_k) \rightarrow \chi_{d_k}^2 \quad (13)$$

The result in (13) implies that the approximation of the maximum log-likelihood evaluated at $\hat{\theta}_k$ becomes

$$\text{Log}L(\hat{\theta}_k|y, M_k) \approx \text{Log}L(\tilde{\theta}_k|y, M_k) + \chi_{d_k}^2 / 2 \quad (14)$$

Thus, from the MCMC algorithm perspective the left hand side of (14) is easily approximated by taking the expectation of the terms in the right side of (14) as

$$\text{Log}L(\hat{\theta}_k|y, M_k) \approx E(\text{Log}L(\tilde{\theta}_k|y, M_k)) + d_k / 2, \quad (15)$$

where $E(\text{Log} L(\tilde{\theta}_k|y, M_k)) \approx \text{LLS}$, and d_k is the number of parameters in θ_k .

Based on simulation studies, Roeder and Wasserman (1997) compared the Laplace and BIC methods for approximating the marginal likelihood and suggested that BIC gives a better approximation. We therefore use BIC to approximate $f(y|M_k)$ in the example below.

We also use the deviance information criterion (DIC) developed by Spiegelhalter et al. (2002) for comparing competing models. The DIC is defined as $\text{DIC} = \bar{D} + p_D$ where $\bar{D}$, which measures model fit, is the posterior mean of the deviance and p_D , which measures model complexity, is the effective number of parameters in the model; $p_D = \bar{D} - D(\bar{\theta})$ where $D(\bar{\theta})$ is

the deviance evaluated at the posterior mean of the unknown parameters. It is a generalization of the Akaike information criterion (AIC), and works on similar principles. The models we compare are specified in (3) where we want to choose an optimal functional form for the impact of order on hazard rates.

4. EXAMPLE: MODELING CHILD EMOTION REGULATION

To illustrate the proposed method, we analyze data from a study of emotion regulation during parent-child interaction (Snyder et al., 2003). The participants for the study were 138 boys and 132 girls and their parents. The interaction of these children with their mothers was videotaped for one hour on each of two occasions during the kindergarten year (mean child age = 5.5 years). On each occasion, mother-child dyad engaged in a series of tasks including playing a novel game, problem solving, engaging in letter and number identification, and planning a fun activity. The videotaped interaction was coded by trained observers using the Specific Affect Coding System (SPAFF) (Gottman, McCoy, Coan, and Collier, 1996). Observers were trained to a criterion of 75% agreement prior to initiating coding of data used in the present report. Weekly recalibration sessions were used to reduce observer drift. The average observer agreement on specific codes in the SPAFF for the data presented in this report was 83% ($\kappa = .73$). The SPAFF codes the onset and offset of both the mother's and child's emotional displays and behavior into one of 19 mutually exclusive and collectively exhaustive categories along a real time line. Super-ordinate categories were created out of these 19 codes for this analysis. For a child, the emotion displays were: (1) anger (contempt, disgust), designated as "A", (2) sad/fearful, designated as "S", and (3) neutral (all other codes), designated as "N". For a parent, the superordinate coding categories used in these analyses were: (1) negative (anger, contempt, disgust, criticism), designated as "M", and (2) neutral or nonnegative (interest, enthusiasm, humor and all other codes), designated as "R". At a dyad-level, the conjoint child-parent emotion display state is indicated by the child emotion (i.e., A, S or N) first followed by the parent state designation (i.e., M or R). For example, "AR" is the dyadic state of child angry-parent neutral. T

Several child and parent characteristics hypothesized to be related to child emotion regulation during mother-child interaction were tested as covariates: maternal self-reported depression (MDEP) and hostility, (MHOS), parent rated child overt antisocial behavior (POA, covert antisocial behavior (PCA), and depression (CCDIM). All covariates were collected in the fall of the kindergarten year when the children were an average of 5.2 years of age. Parent hostility and depression were derived from the Brief Symptom Inventory (Derogatis and Melisaratos, 1983), and measures of child depression and antisocial behavior from the Child Behavior Checklist. The measures of antisocial behavior and depression were obtained from the 36-item parent Child Behavior Checklist (Achenbach and Edelbrock, 1991) which describes aggressive, oppositional, sneaky or stealthy child behavior in the home. The impact of these dyad-level covariates on duration of child emotion regulation during ongoing parent-child interaction is assessed below.

An example of the structure of the data and their layout for analysis is given in Table I. Table I shows a brief sample of real time microlevel observation data of one child's emotion display. The variables are (1) family id, (2) duration (of sad/fear emotion displays), (3) MDEP, (4) MHOS, (5) POA, (6) PCA, (7) ORDER and (8) SESSION coded as dummy variable (0 for Session 1 and 1 for Session 2). The accumulation of emotion display designated as "ORDER" was also calculated. In Table I all values of ORDER for FamilyID 1 are explicitly specified. For example, in SESSION 0 there are only two occurrences of child anger. Thus, ORDER takes values 1 and 2 to represent order of occurrences or equivalently accumulation of anger displays over time. Similarly, in SESSION 1 there are 12 occurrences of child anger. In this case, ORDER takes values from 1 to 12, representing accumulation of anger display over time.

Using the data, similar to those in Table I, the logarithm of the amount of time a dyad spent displaying child angry-parent neutral (AR) behavior until the transition to child neutral- parent neutral (NR) state was plotted against the order of occurrences of the AR dyadic states during interaction to assess order effect associated with affect state repetitions. Figure 1 displays these plots for typical families. The plots display the lowess curves of logarithms of duration against the accumulation of emotional displays (ORDER) for families (Figure 1) with the AR to NR transition at the top and the SR to NR transition in the bottom). The lowess curves suggest between-family variation in the relationships between durations of dyad emotion states and order with which they occur during parent-child interaction. For example, the plots in Figure 1 show that children variously display constant, increasing or decreasing durations of time spent in anger or sadness conditional on the successive occurrence of those states during parent-child interaction. That is, for some families, children display anger for progressively longer periods as anger displays accumulate over time while for others duration gets shorter and shorter as the displays accumulate.

These rich, time sensitive behavioral observations provide potentially powerful information on how children regulate and manage emotions during parent-child interaction. The two child emotion displays targeted in these analyses (given the parents are in a neutral state, (R) are the focus of our analyses: “anger” (A) and “sadness” (S). The duration of these child emotional states before the child transitions to neutral (or to dyadic state NR) is modeled using a hazard duration rate model given (4) above. Hazard rates measure the magnitude of the risk that the duration of a child emotion display will be terminated by moving to another behavioral state. These hazard rates would then reflect the facility with which children can down regulate their negative emotions given their mother is in a neutral emotional state.

This hazard model also has the capacity to describe and account for sequential and temporal dynamics of child emotion displays. Children’s display of emotion can be thought of as recurring events over time. A suitable statistical model for studying the durations and number of real-time occurrences of child emotion displays is a conditional event-dependent frailty model given in (4), which incorporates heterogeneity and event dependence. For event dependence, we let the hazard model be a function of accumulation of child emotion displays. The emotion display accumulation variable (labeled as “ORDER” in the Tables) is determined by the order of occurrence of the emotion display of interest. The duration of the first occurrence of an emotion display (event), the duration of the second occurrence of the same event, and so on may not be same over time during interaction. For example, the lowess curves in Figure 1 show durations of child emotion displays get shorter and shorter as those displays accumulate over time in some families while in other families the opposite occurs. The reasons for these systematic between-child variations in negative emotion regulation may be due to the differences in the characteristics of the children (depression, antisocial behavior) or the parents (hostility, depression). Thus, the hazard rate of terminating a current negative emotion display is likely to be influenced by (1) the sequencing or accumulation of the display (ordering of events), (2) parents’ characteristics, and (3) children’s characteristics.

Thus, model (4) is re-expressed to explicitly include these features of the data as follows.

$$h_{ij}(t)=h_{0i}(t)\exp(\alpha+X_i\beta+u_i+\log(g(j))), \quad (16)$$

where $h_{0i}(t)$ is the baseline hazard function which is defined as $h_{0i}(t) = \gamma t^{\gamma-1}$; and $g(j) = g$ (ORDER) measures the effect of sequential child-emotion displays during child-parent interaction. β is 4×1 vector of coefficients for fixed covariates $X_i = (POA, PCA, MDEP,$

MHOS, *CCDIM*), and u_i is dyad specific frailty. More detailed model specifications were provided in Section 2.

4.1. RESULTS

Estimation of the model parameters in (16) was carried out using the Markov chain Monte Carlo algorithm (Spiegelhalter, Thomas, and Best, 2002), which is a freely available program called WinBUGS 1.4. Convergence with a three-chain run was achieved after 25,000 iterations as it was judged by using trace plots and Gelman and Rubin's (1992) measure of scale reduction factor which was found to lie between .9 and 1.2. Posterior estimates of the parameters were computed based on subsequent 20,000 iterations after convergence and they are given in Tables II–IV that follow.

Several models were fitted to the data to find out the best model that describes the data very well (see Table II). A Weibull hazard model M_k , in the third row, contains main effects $X\beta$ comprising of five dyadic level covariates (POA, PCA, MDEP, MHOS, CCDIM) and random effects u , and a multiplicative function $g(ORDER)$. Different functional forms for $g(ORDER)$ are tested in Models $M_1 - M_6$. Models M_1 and M_4 use the impact of ORDER on the hazard rate as direct multiplicative linearly and exponentially. Models M_2 and M_5 present the impact of ORDER on the hazard rate as reciprocal multiplicatives which have decreasing effects on the hazard. Even more severe reduction of the hazard rates can be modeled by M_3 and M_6 , either linearly or exponentially.

In order to decide which one fits the data well, we use the Bayes factor. Assuming that the prior odds are all equal between these models, the estimated log-marginal likelihoods ($\text{Log } \hat{f}(y|M_k)$) along with the means of logarithm of posterior predictive distributions (LLS) are provided in columns 2–3 of Table II. The log-marginal likelihood for model M_1 is -1225.58 , and the corresponding value for model M_4 is -1226.22 . From these two values, the Bayes factor B_{41} in favor of M_4 is computed as $\exp(-1226.22 - (-1225.58)) = .527$. This suggests that there is a slight difference between M_1 and M_4 according to Raftery and Richardson's recommendation (1996). The Bayes factor for comparing M_4 to M_6 is 7.099, suggesting that there is a strong evidence in favor of M_4 . It seems, therefore, that Model M_4 is a relatively better choice for the fractional form of ORDER since its coefficient in the hazard model has a similar interpretation of hazard ratio as those of the coefficients of dyad-level covariates. Model M_7 expands M_4 by adding interaction terms. The value of the Bayes factor for comparing M_4 versus M_7 is $\exp(9.78)$ which is much greater than 1, indicating that M_7 is better than M_4 with only main effects. Model M_7 has also the lowest DIC value, confirming that the model is best in terms of goodness of fit with a penalty for complexity. Thus, we select model M_7 for making inferences about parameters of the proposed model in (16) and the results are provided in Tables III–IV.

Model M_7 is used to index children's regulation of negative emotions (anger and sadness/fear) and to examine the association of that regulation to characteristics of the parent (hostility, depression) and the child (overt and covert antisocial behavior, depression). The posterior means and posterior standard deviations of the effects of these covariates (or characteristics) are displayed in Tables III and IV. For ease of interpretation, note that a negative sign means the child is slower to terminate the current negative emotion display (either anger - Table III, or sadness/fear - Table IV) given the mother is neutral. A positive sign means the child is faster to terminate the current negative emotion display given the mother is neutral. The term "ORDER" in Tables III and IV refers to the accumulation of occurrences of emotions during the two hours of observed parent-child interaction, and the POA*ORDER, PCA*ORDER, CCDIM*ORDER, MDEP*ORDER, MHOS*ORDER terms refer to the interaction of the rate of accumulation of the child negative emotion over time with the designated parent or child characteristic on the hazard for the child to exit or down regulate the negative emotion.

4.1.1. CHILD CHARACTERISTICS AND ANGER REGULATION—The results for boys will be considered first. As can be seen on the left side of Table III, the coefficient for PCA or child covert antisocial behavior (1.066) has a 90% credible interval of (0.057, 2.048), indicating that boys with high relative scores on covert antisocial behavior are generally faster to terminate anger displays. There is no significant main effect for boys' overt antisocial behavior. The coefficient of the interaction term POA*ORDER is -0.154 with 95% credible interval of $(-0.252, -0.058)$. This interval does not contain zero. The interpretation is that boys who are rated by parents as high on overt antisocial behavior (POA) display increasingly longer durations of anger as anger accumulates (ORDER) or reoccurs during parent-child interaction. Boys high on overt antisocial behavior have increasing difficulty down-regulating anger displays as they repeatedly display anger over time. In contrast, the coefficient of the interaction PCA*ORDER is positive with a value 0.214 with 95% credible interval of (0.089, 0.345) which does not include zero. This indicates that boys who have reported by parents to be high on covert antisocial behavior (PCA) tend to experience increasingly shorter durations of anger as anger accumulates or repeats over time (ORDER) during parent-child interaction. That this means that boys high on covert antisocial behavior are increasingly able to turn off anger displays as they repeatedly display anger over time. Parents' ratings of boys' depression was unrelated to anger displays.

In contrast, as shown in the right hand side of Table III, the coefficient for the association of parent reported depression for girls (CCDIM) with girls' hazard for terminating displays of anger (-0.822) has a 95% credible interval of $(-1.409, -0.272)$. The negative coefficient indicates that girls who reported by parents to be more depressed were slower to down regulate their anger displays. As distinct from the findings for boys, girls' ability to down regulate anger was unrelated to parent-reported overt or covert antisocial behavior.

4.1.2. PARENT CHARACTERISTICS AND CHILD ANGER REGULATION—The associations of parent characteristics with boys' regulation of anger displays are shown in the bottom left portion of Table III. Parental self-reported depression was marginally predictive ($-.663$, $CI(-1.197, -0.109)$) of boys' slower down-regulation of anger, and parental hostility with faster (0.571 , $CI(0.012, 1.105)$) down regulation of anger. However, these patterns were reversed when considering the recurrence of anger over time. Parental depression was reliably associated with increasingly faster (0.084) down regulation of anger by boys as anger displays accumulated over time (i.e., MDEP*ORDER) and parental hostility was reliably associated with slower (-0.076) down regulation of anger as anger displays recurred over time during parent child interaction. Maternal depression may be an effective means of curtailing child anger displays but becomes less effective in doing so as the child repeatedly displays anger over time in parent-child interaction. Maternal hostility, in contrast, actually evokes longer displays of child anger, but this evocative effect wanes as child anger displays are repeated during parent-child interaction.

The associations of parental characteristics to the duration of girls' anger displays are shown in the bottom right portion of Table III. Unlike for boys, maternal self-report depression (MDEP) and hostility (MHOS) did not provide evidence of main effects on the duration of girls' anger. Similar to boys, however, the interaction between MDEP and ORDER was 0.173 with a 95% credible interval ($0.014, 0.327$), suggesting strong evidence. The effect of mother hostility (MHOS) on the duration of girls' anger display was modified by how often girls displayed anger during interaction with their parents. Parental hostility was reliably associated with increasingly slower (-0.086) down regulation of anger by girls as they repeatedly displayed anger during parent child interaction. The interpretation of the results is that girls as well as boys whose mothers show higher degree of hostility display increasingly longer duration anger as anger cumulates or recurs over time during interaction. The effect of mother depression (MDEP) on the duration of girls' anger display was modified by how often girls

displayed anger during interaction with their parents. Parental depression was reliably associated with increasingly faster (0.173) down regulation of anger by girls as they repeatedly displayed anger during parent child interaction. The interpretation of the results is that children whose mothers report higher levels of depression display increasingly longer duration anger as anger cumulates or recurs over time during interaction.

4.1.3. CHILD CHARACTERISTICS AND CHILD REGULATION OF FEAR/

SADNESS—The results of the analyses for boys' regulation of fear/sadness are shown in the left portion of Table IV. As a main effect, parent-reported overt antisocial behavior (POA) was unrelated to boys' down regulation of fear/sadness, but boys high on overt antisocial behavior were slower (-0.165) to down-regulate their fear/sadness displays as those displays accumulated over time during parent child interaction ($POA \times ORDER$). Parent-reported covert antisocial behavior (PCA) was associated with boys' quicker (1.156) down regulation of fear/sadness. Parent-reported depressive behavior (CCDIM) of boys was associated with faster (0.980) down regulation of fear/sadness, but this down-regulation became increasingly slower (-0.187) as boys' repeatedly displayed fear/sadness over time in parent-child interaction. No child characteristics were associated with the hazard rates for fear/sadness regulation for girls.

4.1.4. PARENT CHARACTERISTICS AND CHILD REGULATION OF FEAR/

SADNESS—The association of parent characteristics with children's regulation of fear/sadness is shown in the left bottom portion of Table IV for boys and the right bottom of that Table for girls. Mothers' self-reported depression was associated with faster transitions out of fear/sadness by boys. No other parent characteristics were associated with boys' regulation of fear/sadness.

Parental self-reported depression (MDEP) was reliably predictive (-1.712) of girls' slower down-regulation of fear/sadness, and parental hostility (MHOS) with faster (1.733) down regulation of fear/sadness. However, these patterns were reversed when considering the recurrence of displays of fear/sadness over time. Parental depression was marginally associated with increasingly faster (0.097) down regulation of fear/sadness by girls as those displays accumulated over time (i.e., $MDEP \times ORDER$). Parental hostility (MHOS) was reliably associated with slower (-0.224) down regulation of fear/sadness as child fear/sadness recurred over time during parent child interaction.

For girls, maternal depression is associated with more difficulty down-regulating fear and sadness on average in contrast to less regulation difficulty for boys. This may represent a gender-specific modeling effect or reflect girls' greater sensitivity to empathic reciprocity to their mothers' affect. Moreover, it appears that mothers' trait-like affective states, including both depression and hostility, have a more generalized and powerful effect on girls' than boys' own regulation of fear and sadness. Maternal hostility powerfully facilitated girls' but not boys' down regulation of fear/sadness although this effect diminishes as girls repeatedly display fear/sadness over time in parent-child interaction.

The other parameter of interest is the standard deviation of dyadic-specific random effects, which measures the degree of heterogeneity among dyads. The posterior estimate of the standard deviation of the random effects for girls under $AR \rightarrow NR$ transition is 0.401 with a 95% credible interval of (0.268, 0.566). The lower bound of the interval is not close to zero, suggesting that there is a moderate but significant variation among families (dyads) in the hazard rates from AR to NR transitions. If the lower bound were zero in the credible interval, there would have been no heterogeneity among families.

5. CONCLUSION

In this paper we have demonstrated that a Bayesian event-dependent frailty model provides a powerful tool by which to assess emotion expression and regulation from a time-dynamic perspective. Time-dynamic measures of children's capacity to down-regulate negative emotions during mother-child interaction was estimated along two dimensions: how fast on average a child ceased the expression of anger or sadness/fear once it was initiated, and how the speed of this down regulation changed over time as those emotions were repeatedly displayed during ongoing social interaction. Individual differences in the speed of children's down-regulation of emotions on both of these dimensions were associated in gender-specific ways with the chronic maternal emotional climate to which children were exposed. The size of these associations ranged from relatively small (increases or decreases by 15 to 40%) to quite large (increases by a factor of 4 or more). In general sense, these findings support the formulation by Morris et al. (2007) that children's emotion regulation is affected by the general affective climate of the family. Boys' and girls' regulation of emotional responses appear to be affected by chronic maternal distress in fundamentally different ways. Chronic maternal distress predominantly affected anger regulation by boys and fear/sadness regulation by girls. More specifically, exposure to increasing maternal hostility was related to boys' faster down-regulation of anger and to girls' faster down-regulation of fear/sadness. Conversely, maternal depression was associated with boys' slower down-regulation of anger and girl's slower down-regulation of fear/sadness. From a functional perspective, these patterns of emotion regulation may reflect boys' general tendency to utilize a more instrumental approach and girls to utilize a more empathic, relationship-enhancing approach to social transactions (Zahn-Waxler et al., 2005), perhaps amplified by chronic negative maternal mood states. Once expressed, boys may down-regulate anger more quickly given chronic exposure to generalized maternal hostility because sustained expression of anger is simply not instrumentally productive in attaining maternal attention or access to desired materials and activities. Given exposure to chronic maternal depression, boys may be slower to down-regulate anger because sustained anger is functional in escaping maternal demands or in taking advantage of maternal emotional vulnerability to obtain desired materials and activities. Girls' may more rapidly down-regulate fear/sadness when exposed to generalized maternal hostility because sustained fear/sadness and associated help-seeking behaviors are not likely to be met with supportive maternal reactions. Given chronic exposure to generalized maternal depression, girls may be slower to down-regulate expressions of fear/sadness in order to communicate empathy and support, or as a result of modeling and emotion contagion.

However, the observed propensities for faster or slower down-regulation of anger and fear/sadness in response to chronic and generalized maternal distress are not tonic responses. Boys' faster down-regulation of anger in the context of generalized maternal hostility and slower down-regulation of fear/sadness in the context of generalized maternal depression, and girls' faster down regulation of fear/sadness in the context of generalized maternal hostility and slower down regulation of fear/sadness in the context of generalized maternal depression appeared to be increasingly difficult to sustain as children expressed those emotions repeatedly during mother-child interaction. In absolute size, these temporally-accumulating and countervailing shifts in the increased or decreased average speed of down-regulation of negative emotions were relatively small, but experientially dynamic. Boys, for example, may try to minimize the duration of anger expressions in the context of general maternal hostility, but it appears that suppression of anger becomes increasingly difficult to sustain during ongoing interaction with their mothers. Similarly, girls may slow down-regulation of expressions of sadness and fear in the context of general maternal depression, but the duration of empathic expressions of fear and sadness wanes during ongoing interaction with their mothers. Diminution in the speed of down-regulation of negative emotions with their repeated expression may reflect a cumulative sensitization in which the effortful regulation of an

emotion becomes increasingly difficult as those emotions are repeatedly experienced over a short period of time (Snyder et al., 2003).

The findings reported in this paper need to be contextualized by a number of limitations. The data were derived from a cross-sectional, correlational design. As such, causality cannot be inferred and the direction of the relationships among the variables is not clear. Child conduct problems and depression were derived solely from parent report, which is known to have inherent biases. The data were derived from an at-risk community sample from one geographic location and school, limiting the degree to which generalizations to other, more broadly representative samples can be inferred. The hypotheses linking general parental distress to child emotion regulation, and child emotion regulation in turn to child adjustment infers a mediational model, but a mediational model could not be adequately tested in this cross-sectional study. In addition, there are no time-varying covariates in the dataset that may potentially be used to explain the nature of effects of accumulating events (event dependence) on the hazard function of emotions, which could be a weakening or a strengthening effect.

The analyses and data in this report have several general implications. First, empirical progress in the study of child emotions and emotion regulation may be facilitated by taking an increasingly differentiated view of their phenomenology; anger and fear/sadness seem to have potentially different functions, are responsive to different environmental contexts, and their relative dysregulation may pose different risks. Second, the observation of the time dynamic expression of emotions provides a useful methodological and theoretical complement to more trait-like, temperamental approaches to emotions and emotion regulation. Third, sophisticated repeated-events duration models are now available to successfully address a number of very challenging analytic issues that have limited interpretation of the real-time expression and function of emotions in previous research (Dagne et al., 2003, 2007).

6. BIBLIOGRAPHY

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Figure 1.
Lowess Plots for Duration of Behavior of Selected Children: $AR- > NR$ (Top Row) and $SR- > NR$ (Bottom Row)

Table 1
Layout of Data Structure for Emotion Display of Female Children and Parents During Family Interaction

FamilyID	Duration	MDEP	MHOS	POA	PCA	ORDER	SESSION
1	0.70	0.461	0.091	0.283	0.115	1	0
1	4.93	0.461	0.091	0.283	0.115	2	0
1	2.00	0.461	0.091	0.283	0.115	1	1
1	1.80	0.461	0.091	0.283	0.115	2	1
1	1.4	0.461	0.091	0.283	0.115	3	1
1	0.93	0.461	0.091	0.283	0.115	4	1
1	2.2	0.461	0.091	0.283	0.115	5	1
1	2.0	0.461	0.091	0.283	0.115	6	1
1	0.8	0.461	0.091	0.283	0.115	7	1
1	2.47	0.461	0.091	0.283	0.115	8	1
1	2.23	0.461	0.091	0.283	0.115	9	1
1	0.84	0.461	0.091	0.283	0.115	10	1
1	1.97	0.461	0.091	0.283	0.115	11	1
1	0.8	0.461	0.091	0.283	0.115	12	1
2	1.26	0.538	0.636	0.674	0.096	1	0
2	1.50	0.538	0.636	0.674	0.096	2	0
2	2.2	0.538	0.636	0.674	0.096	3	0
:	:	:	:	:	:	:	:

POA = parent overt antisocial report of child, PCA = parent covert antisocial report of child, MDEP = mother depression, and MHOS = mother hostility. CCDIM = child depression, ORDER = order of behavior occurrence

Table II**Model Comparison**

Model	LLS	$\text{Log } \hat{f}(y M_k)$	DIC
Main Effects			
$M_k : h(t) = g(\text{order})h_0(t)\exp(X\beta + u)$			
where			
$M_1: g(\text{order}) = \text{order}$	-1200.32	-1225.58	2439.39
$M_2: g(\text{order}) = 1/\text{order}$	-1200.24	-1225.50	2439.34
$M_3: g(\text{order}) = 1/\text{order}^2$	-1201.36	-1226.62	2439.49
$M_4: g(\text{order}) = \exp(\text{order})$	-1200.96	-1226.22	2440.83
$M_5: g(\text{order}) = \exp(1/\text{order})$	-1201.65	-1226.91	2440.38
$M_6: g(\text{order}) = \exp(1/\text{order}^2)$	-1202.92	-1228.18	2440.56
$M_7: \text{main Effects + Interactions}$	-1196.49	-1216.44	2436.02

LLS = posterior log-likelihood score; *DIC* = deviance information criterion; $g(\text{order})$ is a functional form for event dependence in terms of the order of an event.

Table III

Posterior Estimates of Parameters of an Event-dependent Hazard Model for $AR \rightarrow NR$ Transition by Gender

Variable	$AR \rightarrow NR$					
	Boys			Girls		
	Mean	SD	95% Interval	Mean	SD	95% Interval
Intercept	-1.576	.237	(-2.036, -1.123)	-.983	0.193	(-1.367, -0.617)
Child Covariates						
POA	-0.086	0.408	(-0.893, 0.727)	0.590	0.527	(-0.438, 1.613)
PCA	1.066	0.607	(0.057, 2.048)*	-2.042	1.284	(-4.541, 0.514)
CCDIM	0.252	0.341	(-0.405, 0.93)	-0.822	0.289	(-1.409, -0.272)
ORDER	0.037	0.021	(0.002, 0.071)*	-0.013	0.023	(-0.057, 0.032)
POA* ORDER	-0.154	0.049	(-0.252, -0.058)	0.008	0.059	(-0.104, 0.124)
PCA* ORDER	0.214	0.065	(0.089, 0.345)	-0.222	0.160	(-0.530, 0.101)
CCDIM* ORDER	-0.028	0.034	(-0.095, 0.039)	0.050	0.037	(-0.025, 0.121)
Parent Covariates						
MDEP	-0.663	0.334	(-1.197, -0.109)*	-0.302	0.398	(-1.107, 0.454)
MHOS	0.571	0.335	(0.012, 1.105)*	0.416	0.334	(-0.242, 1.067)
MDEP* ORDER	0.084	0.039	(0.008, 0.163)	0.173	0.080	(0.014, 0.327)
MHOS* ORDER	-0.076	0.038	(-0.149, -0.005)	-0.086	0.049	(-0.164, -0.005)*
SESSION	0012	0096	(-0.175, 0.200)	-0.137	0.093	(-0.319, 0.046)
γ (Shape)	1.564	0.043	(1.479, 1.647)	1.699	0.053	(1.597, 1.803)
σ	0.455	0076	(0.322, 0.617)	0.401	0.076	(0.268, 0.566)
Deviance	2394.0	11.25	(2374.0, 2418.0)	1865.0	10.88	(1845.0, 1888.0)

* 90% Credible Interval

AR = child angry and parent neutral, NR = child neutral and parent neutral, POA = parent overt antisocial report of child, PCA = parent covert antisocial report of child, $MDEP$ = mother depression, and $MHOS$ = mother hostility. $CCDIM$ = child depression, $ORDER$ = order of behavior occurrence

Table IV
Posterior Estimates of Parameters of an Event-dependent Hazard Model for $SR > NR$ Transition by Gender

Variable	SR->NR					
	Boys			Girls		
	Mean	SD	95% Interval	Mean	SD	95% Interval
Intercept	-2.356	0.294	(-2.967, -1.812)	-1.662	0.2403	(-2.134, -1.2)
Child Covariates						
POA	-0.136	0.514	(-1.11, 0.899)	0.315	0.642	(-0.914, 1.562)
PCA	1.156	0.677	(0.035, 2.276)*	-1.015	1.426	(-3.79, 1.737)
CCDIM	0.980	0.462	(0.057, 1.886)	-0.081	0.448	(-0.033, 0.036)
ORDER	0.101	0.044	(0.019, 0.191)	0.001	0.018	(-0.948, 0.809)
POA* ORDER	-0.165	0.085	(-0.339, -0.007)	0.079	0.079	(-0.076, 0.232)
PCA* ORDER	0.142	0.121	(-0.099, 0.377)	-0.112	0.172	(-0.452, 0.221)
CCDIM* ORDER	-0.187	0.059	(-0.300, -0.070)	-0.030	0.052	(-0.137, 0.068)
Parent Covariates						
MDEP	0.767	0.368	(0.053, 1.497)	-1.712	0.43	(-2.569, -0.881)
MHOS	-0.489	0.371	(-1.253, 0.213)	1.733	0.344	(1.065, 2.407)
MDEP* ORDER	0.035	0.049	(-0.066, 0.125)	0.097	0.057	(0.003, 0.191)*
MHOS* ORDER	-0.031	0.057	(-0.135, 0.088)	-0.224	0.039	(-0.300, -0.146)
SESSION	0.045	0.105	(-0.160, 0.254)	0.215	0.096	(0.026, 0.403)
γ (Shape)	1.106	0.037	(1.034, 1.18)	1.08	0.033	(1.017, 1.144)
σ	0.579	0.099	(0.408, 0.794)	0.694	0.102	(0.573, 0.915)
deviance	2735.0	11.46	(2715.0, 2759.0)	3381.0	11.33	(3361.0, 3405.0)

* 90% Credible Interval

SR = child sad and parent neutral, NR = child neutral and parent neutral, POA = parent overt antisocial report of child, PCA = parent covert antisocial report of child, $MDEP$ = mother depression, and $MHOS$ = mother hostility. $CCDIM$ = child depression, $ORDER$ = order of behavior occurrence